2013

The Devil Undone: the science and politics of Tasmanian Devil facial tumour disease

Josephine Veronica Warren
University of Wollongong, jowarren@uow.edu.au

The Devil Undone:

The Science and Politics of Tasmanian Devil Facial Tumour Disease

Josephine Veronica Warren BA (Hons)

University of Wollongong

School of Social Sciences, Media and Communication

20 December 2013

This thesis is presented for the degree of Doctorate of Philosophy

Abstract

The Tasmanian devil is a carnivorous marsupial endemic to the island state of Tasmania, part of the larger continent of Australia, threatened with extinction from a deadly cancer. The research into the cancer, termed Devil Facial Tumour Disease (DFTD), followed a pathway that supported the hypothesis that the cancer was transmissible, passed from devil to devil by biting, called an allograft. By adopting a political sociological approach, I analyse the scientific research into the devil cancer through the concept of undone science, which I expand by developing a typology of reasons, both practical and political, for deficits of knowledge.

My analysis initially finds that scientific evidence has not been established to confirm the transmission of the cancer by biting. The devil cancer research has also failed to produce convincing support for the precedent of a dog transmissible cancer. Whilst allograft research was pursued, the competing hypothesis that chemicals used in plantation forestry might have contributed to the disease was neglected. There were many calls for further toxicology studies but to date these have not been undertaken.

Due to the devils' listing as endangered under the *Environment Protection and Biodiversity Conservation Act 1999* and the scientific uncertainty surrounding the cause of the cancer, the precautionary principle is relevant. Applying it would enable decision makers to seek further scientific studies into the cause of the cancer and to mitigate the harm by further restricting or banning the use of the chemical atrazine used in plantation forestry. I analyse four wildlife cancers, including the Tasmanian devil, to demonstrate that in all cases toxicology studies have been neglected.

Close relations between the Tasmanian government and the forestry industry, when operations should be at arms length, have resulted in a conflict of interest in the regulation of chemical use in plantations and the overseeing of the Tasmanian devil scientific research. I recommend that public participation and lay knowledge be incorporated into the future governance of environmental issues.

Statement of Candidate

I certify that this thesis entitled 'The Devil Undone: The Role of Scientific Ignorance and Politics in the Struggle to Save the Tasmanian Devil', is entirely my own work except where I have given full documented reference to the work of others, and that the material contained in this thesis has not been submitted for formal assessment in any formal course.

Josephine Veronica Warren
20 December 2013

Acknowledgements

I would like to express the sincerest appreciation to my principal supervisor, Professor Brian Martin and my co-supervisor Professor Sharon Beder for their constant encouragement, guidance and support throughout the entire process of completing this research project. I would also like to thank Richard Gosden, Alison Bleaney and the many others in Tasmania who contributed their knowledge and whose enthusiasm and dedication helped to maintain my commitment to the project. To my fellow post-graduates and Gayl Vidgen at the Batemans Bay Campus a big thanks for reading and providing helpful comments on my thesis and for sharing the journey. Finally, I would like to thank my family Brick, Rosalie, Troy and Zac for their understanding and patience.

Note on Referencing

In place of a bibliography at the end of this thesis, full citations are given in the footnotes in each chapter. *Ibid* is used when citing the previous reference.

Table of Contents

Introduction

Chapter 1 – The Tasmanian devil (Sarcophilus harrissi

Chapter 2 – The political sociology of science and undone research

Chapter 3 – The allograft theory

Chapter 4 – The science selected for study

Chapter 5 – DFTD toxicology studies – the undone science

Chapter 6 – The precautionary principle and its role in the mitigation of harm to wildlife species

List of Appendices

List of Abbreviations

1080	Sodium Fluoroacetate (1080) poison
2,4-D	2,4-dichlorophenoxyacetic acid
ABC	Australian Broadcasting Commission
ACSH	American Council on Science and Health
AEI	American Enterprise Institute
AFRS	Alan Fletcher Research Station
AGAL	Australian Government Analytical Laboratories
AIDS	Acquired immune deficiency syndrome
AMA	Australian Medical Association/American Medical Association
APVMA	Australian Pesticides and Veterinary Medicines Authority
ARAZPA	Australasian Regional Association of Zoological Parks and Aquaria
ASCHEM	Agricultural, Silvicultural and Veterinary Chemicals Council
AST	Analytical Services Tasmania
BaP	Benzo[a]pyrene
CAR	Comprehensive, Adequate and Representative Reserve System for Forests
CBD	Convention on Biological Diversity
CCC	Community Consultative Committee
CCIA	Children's Cancer Institute Australia
CCRI	Children's Cancer Research Institute

CEO	Chief Executive Officer
CMG	Common mechanism group
CNN	Cable News Network
COMEST	World Commission on the Ethics of Scientific Knowledge and Technology
CRC	Cooperative Research Centres
CSC	Clonal stem cells
CSHL	Cold Spring Harbor Laboratory
CSL	California Sea Lions
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CTL	cytotoxic T lymphocytes
CTVT	Canine Transmissible Venereal Tumour
CYP	Cytochrome
DACT	diaminochlorotriazine
DDT	Dichloro-Diphenyl-Trichloroethane
DEA	desethyl-s-atrazine
DFTD	Devil Facial Tumour Disease
DIA	desisopropyl-s-atrazine
DIER	Department of Infrastructure and Energy Resources
DNA	Deoxyribonucleic acid
DPIPWE	Department of Primary Industries, Parks, Water and Environment

EDCs	Endocrine disrupting contaminants (or chemicals)
EDO	Environmental Defenders Office
EMPCA	Environmental Management and Pollution Control Act
EPA	Environmental Protection Agency
EPBC	Environment Protection and Biodiversity Conservation Act
ESA	Ecotox Services Australasia
EU	European Union
FHRMG	Forest Herbicide Research Management Group
FIAT	Forestry Industry Association of Tasmania
FOI	Freedom of Information
FPA	Forest Practices Authority
FPB	Forest Practices Board
FPC	Forest Practices Code
FPP	Forestry Practices Plan
FT	Forestry Tasmania
GIS	Geographical Information Systems
GM	Genetically modified
GMOs	Genetically modified organisms
HSIS	Hazardous Substances Information System
IARC	International Agency for Research on Cancer
ICSU	International Council for Science

IIS	Integrated Impact Statement
IUCN	International Union for Conservation of Nature
IUPAC	International Union of Pure and Applied Chemistry
mg/L	milligrams per litre
MHC	Major histocompatibility
MIS	Managed investment scheme
MRL	Maximum residue limit
NAFI	National Association of Forest Industries
NATA	National Association of Technical Authorities
NCI	National Cancer Institute
NFA	National Farmers Association
NFPS	National Forestry Policy Statement
NHL	Non-Hodgkins Lymphoma
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NIEHS	National Institute of Environmental Health Services
NIWA	National Institute of Water and Atmospheric Research
NMI	National Measurement Institute
NRA	National Registration Authority
NRDC	Natural Resources Defense Council
NPSS	New Political Sociology of Science
PAHs	Polycyclic aromatic hydrocarbon

PFT	Private Forests Tasmania
PBBs	Polybrominated biphenyls
PBDEs	Polybrominated diphenyl ethers
PCBs	Polychlorinated biphenyls
PCDD	Polychlorinated dibenzodioxins
PCDF	Polychlorinated dibenzofurans
PFT	Private Forests Tasmania
ppb	parts per billion
PPT	Plantation Platform of Tasmania
PR	Public Relations
PTR	Private Timber Reserve
RCWQI	River Catchment Water Quality Initiative
REACH	Registration, Evaluation and Authorisation of Chemicals
RFA	Regional Forest Agreement
RPDC	Resource and Planning Development Commission
SAPs	Scientific Advisory Panels
SARS	Severe acute respiratory syndrome
SBR	Scammell & Bleaney Report
SCOPE	Scientific Committee on Problems of the Environment
SETAC	Society of Environmental Toxicology and Chemistry
SLAPP	Strategic Litigation Against Public Participation
SLE	St Lawrence Estuary

STBA	Southern Tree Breeding Association
STDP	Save the Tasmanian Devil Program
STS	Science and Technology Studies
TAPP	Tasmanians Against the Pulp Mill
TCA	Timber Communities Australia
TCDD	Tetrachlorodibenzodioxin
TCDF	Tetrachlorodibenzo-furan
TFCA	Tasmanian Forest Community Agreement
TFPS	Tasmanian Forest Practices Scheme
THING	Tasmanian Haematology, Immunology and Neoplasia Group
TIE	Toxicity Identification Evaluation
TT	Triazine Tolerant
TSP	Threatened Species Protection Act
TWQI	Tasmanian Water Quality Initiative
US	United States
USDA	United States Department of Agriculture
UTAS	University of Tasmania
WCS	Wildlife Conservation Society

List of Figures

List of Tables

Introduction

The Tasmanian natural environment, like many environments worldwide, is under threat from human activities and in particular from industrial agricultural practices through the use of pesticides that contaminate soil, water and the atmosphere. A consequence of this contamination is an increase in wildlife diseases and chronic human health problems. It is the wildlife diseases, and in particular the cancer threatening the extinction of the Tasmanian devil, that is the focus of this thesis. It has been termed devil facial tumour disease (DFTD). Whilst the human health problems are not inconsiderable, I concentrate on the Tasmanian devil cancer as a bio-indicator of these problems. I will argue that the use of pesticides, chemicals designed to kill living organisms, the consequence of contamination of water bodies in Tasmania and a lack of action by the Tasmanian government to restrict pesticide use, is a credible hypothesis for the cause of the devil cancer. This is in direct contrast to the working hypothesis adopted by the Tasmanian government and a body of scientists working on the devil disease. Their hypothesis is that the cancer originated in one cell in one devil and through a series of unfortunate events became a transmissible cancer passed from devil to devil through their propensity to bite.

The environmental problems arising in Tasmania are not isolated events. Through the integration of science and industry, human activities have greatly improved living conditions for a large proportion of the world's population and continue to do so. The benefits have been considerable but unforeseen harmful consequences are also ongoing and cumulative. These include acid rain, depletion of the ozone layer, contamination of surface and ground water, air pollution, depletion of resources such as wild fish stocks,

climate change and the loss of biodiversity; the last leading to possibly the earth's sixth mass extinction.[1] Often the consequences have been the result of ignorance but there have also been early warnings. Rachel Carson was one of many voices giving early warnings of the dangers of pesticides in her book *Silent Spring*.[2] Theo Colburn and colleagues in *Our Stolen Future* also warned of the dangers of endocrine disrupters, chemicals that mimic an organism's natural hormones thus interfering with normal developmental and reproductive functioning.[3]

These are complicated problems often occurring across diverse organisms in highly complex ecosystems. In order to address these problems there is a requirement for action by decision makers who need to rely on informed scientific opinions. But scientific knowledge is often uncertain which can lead to action being thwarted by dominant interest groups keen to protect the status quo. To interrogate this supposition my thesis focuses on the struggle to save the Tasmanian devil from a deadly facial tumour threatening its extinction. My approach, using a framework of practical and political reasons for undone science, takes a political sociological perspective in investigating the scientific research into the disease. I have found the research is shaped by vested interests that support the hypothesis that DFTD is a transmissible tumour, called an allograft. The competing theory, that chemicals used in plantation forests play a role in the etiology of the disease, has been abandoned, and those proposing it have been marginalized and ignored.

---

[1] Barnosky AD, Matzke N, Tomiya S, Wogan GOW, Swartz B, Quental TB, Marshall C, McGuire JL, Lindsey EL, Maguire KC, Mersey B & Ferrer EA, 2011, Has the Earth's sixth mass extinction already arrived? *Nature* Vol 471, pp 51-57

[2] Carson R, with an introduction by Al Gore, 1994, *Silent Spring*, Houghton Mifflin, Boston

[3] Colborn T, Dumanoski D & Myers JP, 1996, *Our Stolen Future, Are We Threatening Our Fertility, Intelligence, and Survival? – A Scientific Detective Story*, Little, Brown and Company, London

Tasmania - an island under threat?

Tasmania is a small island state, separated from the island continent of Australia by Bass Strait. It is endowed with superb natural beauty of majestic ancient forests and abundant water. This idyllic landscape has undergone a major transformation since the arrival of European settlers in the early 1800s. Their arrival precipitated many changes, not all of which have benefited the island's natural environment. The island has sustained numerous wildlife extinctions, most notably the Thylacine or Tasmanian tiger. Although island species generally experience more extinctions than larger landmasses through natural events,[4] the extinctions in Tasmania have been mainly the result of human activities.

Early European settlers arriving in Tasmania were confronted with an unfamiliar natural environment, the Australian bush, but this did not prevent them from exploiting the island's natural resources. They soon engaged in the clearing of native vegetation for pasture, the logging the forests for timber and mining the ground for minerals. These activities created great economic benefits but were achieved at unforeseen human and environmental costs. The clearing of vegetation for pasture brought the pastoralists and farmers into direct conflict with the native wildlife. This generated an eradication program to shoot, trap and poison competing native species. The logging of native forests destroyed natural habitat and the mining of minerals devastated vast areas of land. These activities began in the early 1800s and continue today with an increasingly detrimental impact on native flora and fauna.

[4] Frankham R, 1998, Inbreeding and Extinction: Island Populations, *Conservation Biology*, Vol 12(3), pp 665-675

In Tasmania a combination of habitat destruction and an increase in plantation forestry has correlated with the recent rapid increase in wildlife diseases. They include a platypus skin ulcer,[5] chytrid fungus in frogs,[6] sarcoptic mange in wombats,[7] wobbly possum disease possibly caused by a virus in Brush tailed possums,[8] and cancer in devils. The latest victims are the devils' closest relatives and members of the Dasyurid family, the Spotted Tail Quoll. The Tasmanian Spotted Tail Quoll population is rapidly decreasing but the cause is unknown. The decline in quoll population is puzzling, because they compete with devils for food and it was expected that a decline in devil population would see an increase in their number.[9]

Although these diseases may not be related they are indicative of an ecosystem at risk. Coincidental with the wildlife diseases are continuing abnormalities in commercial oysters and occasional mass deaths in oysters and inter-tidal organisms.[10] Human health problems are also on the increase notably chronic diseases, including cancer. This situation prompted the Tasmanian branch of the Australian Medical Association (AMA) to call for an inquiry into the apparent rise in cancers and neurological diseases in patients in the north east of Tasmania.[11]

---

[5] Connolly JH, Obendorf DL, Whittington RJ & Muir DB, 1997, Causes of Morbidity and Mortality in Platypus (Ornithorhynchus Anatinus) from Tasmania, with particular reference to Mucor Amphibiorum infection, *Australian Mammology*, Vol 20, pp 177-187

[6] Obendorf D, 2005, *Draft Report for the Australian Government Department of the Environment and Heritage, Application of field & diagnostic methods to survey for chytridiomycosis in Tasmanian frogs*, Central North Field Naturalists Inc., Tasmania

[7] Cox A, 2007, *Interim Report on Eradicating Mange in Wild Wombats*, Wombat Protection Society of Australia

[8] Philips A & Driessen M, 2008, *Strategy for managing wildlife disease in the Tasmanian Wilderness World Heritage Area*, Department of Primary Industry and Water, Hobart, Tasmania

[9] Waterhouse C, 2010, Survival fears for quolls, *The Mercury*, 16 July 2010. Available at: http://www.themercury.com.au/article/2010/07/16/159131_print.html accessed 14 August 2010

[10] Percival S, 2004, *Oyster Health in Georges Bay, Collation and analysis of data*, Tasmanian Department of Primary Industry, Water and Environment, Hobart, Tasmania

[11] *PM with Mark Colvin*, 2004, radio program, Australian Broadcasting Corporation, Sydney

The problems in human and wildlife health are correlated in time and space across the state not only with habitat destruction but also with the development of eucalypt plantation forests and their management practices. Therefore, the focus of my research is not only an analysis of the scientific research into Tasmanian devil DFTD but also an investigation of possible links to plantation forest management practices, and in particular the widespread use of pesticides and their regulation. These issues are covered later in the thesis in Chapter 9.

Theoretical framework

The framework used to analyse the scientific research into DFTD is based on David Hess's concept of undone science.[12] Hess developed the concept when analysing the scientific research into conventional methods of medicine and agriculture and their alternatives. He found that the vast majority of funding followed the pathways of the dominant theories, whilst competing alternative approaches, such as alternative medicine or organic farming, received little funding and 'withered on the vine'.[13]

I have expanded Hess's concept further by developing a typology of practical and political reasons for undone science. Practical reasons include a lack of technical or theoretical knowledge whilst political reasons include the possibility of producing findings that would be perceived as damaging to vested interests. Hess found that elites in society, for example those in government and industry, fund the majority of research. In the case of the Tasmanian devil research the majority of the funding has come from the Australian federal government and the Tasmanian state government. Both have a vested interest in protecting jobs, infrastructure and investment in forestry plantations.

---

[12] Hess D.J., 2007, *Alternative pathways in science and industry*, MIT Press, Cambridge, Massachusetts
[13] ibid, p 42

Drawing on the concept of the social construction of scientific knowledge, Hess also focuses on the rarely acknowledged "selection" of research areas for study. This approach has also been incorporated into my analysis of the DFTD research to show that studies supporting the allograft theory have been preferentially chosen for funding. My analysis therefore covers both the research selected for study, which will be shown to have produced findings that are contradictory, and the research that has been marginalized or abandoned. This includes alternative hypotheses, such as the role of viruses or carcinogens in the initiation or promotion of the cancer, which form a body of undone science. A detailed description of my theoretical analysis and methodology is given in Chapter 2, 'The political sociology of science and undone research'.

Devil Facial Tumour Disease (DFTD) science selected for research – the allograft hypothesis

Currently the Tasmanian devil is listed as endangered under the national *Environment Protection and Biodiversity Conservation Act 1999, (EPBC Act 1999)* under the *Tasmanian Threatened Species Protection Act 1995 (TTSP Act 1995)* and on the *2008 IUCN Red List of Threatened Species.*[14] The major threat to the devils' survival is confirmed as DFTD.

Initially DFTD researchers acknowledged the possibility of a chemical causality of the cancer but following a pilot study this was abandoned.[15] The spread of the devil cancer has been accounted for by the devils' habit of biting. It is a cancer hypothesized to be

[14] Australian Government, Department of the Environment, Water, Heritage and the Arts, *Sarcophilus harrisii - Tasmanian Devil, Legal Status*. Available at: http://www.environment.gov.au/cgi-bin/sprat/public/publicspecies.pl?taxon_id=299 last accessed 8 August 2010

[15] Pyecroft SB, 2007, Chemicals: Their role in DFTD, *Devil Facial Tumour Disease Senior Scientist's Scientific Forum*, 20-22 February 2007, University of Tasmania, Hobart

transmissible (spread from devil to devil) by biting and described as an allograft. Anne Maree Pearse, a scientist working at the Department of Primary Industries, Parks, Water and Environment (DPIPWE) Mt Pleasant laboratory in Launceston, northern Tasmania and her laboratory assistant Kate Swift published a brief account of the allograft hypothesis in *Nature* in 2006.[16] The hypothesis is based on a study of eleven devils in which identical chromosomal abnormalities were observed in all the tumour cells. Further support for the hypothesis was the observation that one devil had an anomaly in chromosome 5 that was not found in the tumour chromosome 5, where it would have been expected had the tumour arisen in the host devil.

Pearse and Swift also proposed that the only other known transmissible cancer, which occurs in dogs and is termed canine transmissible venereal tumour (CTVT), was a precedent for the allograft hypothesis. Confirmation of the transmission of the dog tumour was published in 2006 when the *c-myc* oncogene was found to be present in all the dog tumour samples.[17] The original successful transmission studies had been undertaken by Novinski in 1876.[18] Similar studies have not confirmed the transmission of the devil cancer. On the contrary entirely novel research has been selected to support the allograft hypothesis.

Potentially inconsistent with the allograft hypothesis is the recent disclosure that the chromosomes in the devil cancer cells are unstable, like all cancers. Pearse and Swift had claimed in the *Nature* article that the devil chromosomes, like the dog

[16] Pearse AM & Swift K, 2006, 'Transmission of devil facial-tumour disease' *Nature*, Vol 439(2), p 549
[17] Murgia D., Pritchard J.K., Kim S.Y., Fassati A. & Weiss R.A., 2006, *Cell*, Vol 126, pp 477-487
[18] ibid, Novinski MA, 1876, Zur Frage uber die Impfung der Krebsigen Geschwulste, Zentralbl. Med. Wissensch. Vol 14, pp 790-791

chromosomes, were identical and stable. What this might mean for the allograft hypothesis and its precedent the dog transmissible cancer is discussed in Chapter 3.

An anomaly in the allograft theory: the devils' immune system

A scientific riddle in the allograft hypothesis that has concerned the devil scientific researchers has been the ability of the tumour cells to establish and proliferate in the new host. This research has been confounded by studies into the structure of the devil immune system, which to date have shown the immune system to be competent.[19] (It is common in cases of similar malignant cancers for an immune system to be compromised allowing the cancer to proliferate and spread.[20]) This unusual finding led researchers to propose a further hypothesis, that the devils' lack of genetic diversity is the reason for the transmission of the cancer. Confirmation appeared to come from a study showing a lack of diversity in a group of genes, the major histocompatibility (MHC) genes that enable the immune system to recognize foreign invaders (bacteria, viruses or cancer cells).[21] However, this hypothesis was abandoned when a skin graft study showed the immune system did in fact recognize foreign tissue.[22]

Questions have arisen due to these anomalies in the original hypothesis. Why does the devil immune system not reject the cancer? If the immune system is competent, which it is proposed, how do the cancer cells establish in the host? Answering these questions should have directed the research. These questions and the role of the devils' immune system are also discussed in Chapter 4, 'The science selected for study'.

---

[19] Woods GM, Kreiss A, Belov K, Siddle HV, Obendorf DL & Muller HK, 2007, The Immune Response of the Tasmanian Devil (Sarcophilus harrisii) and Devil Facial Tumour Disease, *EcoHealth* 4, pp 338-345
[20] Brower V, 2011, Portents of malignancy, *Nature*, Vol 471, pp S19-S21
[21] Siddle HV, Sanderson C & Belov K, 2007, Characterization of major histocompatibility complex class I and class II genes from the Tasmanian devil (Sarcophilus harrisii), *Immunogenetics*, Vol 59, pp 753-760
[22] Kreiss A, Cheng Y, Kimble F, Wells B, Donovan S, Belov K & Woods GM, 2011, Allorecognition in the Tasmanian Devil (Sarcophilus harrisii), an Endangered Marsupial Species with Limited Genetic Diversity, *PloS One*, Vol 6(7), pp 1-8

DFTD and undone science

Unlike the dog transmissible cancer, which has been confirmed by two independent studies, the devil cancer transmission studies are yet to be completed. Stephen Pyecroft, Principal Veterinary Pathologist at the DPIPWE Mt Pleasant laboratory, undertook transmission trials and published an abstract of the results in 2007, which were said to be variable.[23] No further transmission trials have been undertaken. Similar genetic studies to those undertaken for the dog transmissible cancer have not been undertaken to confirm DFTD is transmissible. Scientific research into the devil cancer began officially in 2003 which means ten years has elapsed and still these and other studies await completion.

Two competing hypotheses, that a virus is involved and that the widespread use of pesticides and/or poisons used in plantation forestry and agriculture may be involved in initiating or promoting the cancer, have also received scant attention. Pearse was the first to propose a virus when she suggested a flea (*Uropsylla tasmanica)*, unique to Dasyurids – devils and their cousins the native cats (quolls) - could have been the vector.[24] This line of inquiry has never been examined. Toxicology studies first proposed by the DPIPWE as part of the investigation into the cancer were abandoned following a pilot study on a limited number of chemicals. These results were only released following a Freedom of Information request but were not made public. However, two summaries of the findings, one by Professor Michael Moore and the other by Dr Tony Ross, both suggested that further studies were needed. Further research into the role of environmental toxins or poisons used in plantation forests or

[23] Pyecroft SB, 2007, Transmission trials: Devil Facial Tumour Disease, *Devil Facial Tumour Disease Senior Scientist's Scientific Forum,* 20-22 February 2007, University of Tasmania, Hobart
[24] Personal communication, June 2011

agriculture has not been undertaken. The findings of my analysis using the concept of undone science are discussed in Chapter 5, 'DFTD toxicology studies and undone science'.

I argue that the scientific studies have not been undertaken because of the possible negative impact on the forestry industry in Tasmania. The greatest threat to species extinction worldwide is habitat destruction through both loss and contamination from human activities; this is also the situation in Tasmania. The shaping of the scientific research and the neglected and abandoned research into the devil cancer is but one aspect of the problem for this species. The other force driving its extinction is plantation forestry industry practices with inadequate regulation of chemicals.

In chapter 6 I propose that the precautionary principle be implemented due to the growing scientific uncertainty as a result of the undone research. The precautionary principle is a decision making tool for action, first called for in Tasmania in the *Scammell Report* in 2003, to halt aerial spraying of chemicals in plantation forests until further research could be undertaken. The need for the adoption of the precautionary principle is discussed in Chapter 6 'The precautionary principle – its role in the Tasmanian devil cancer'. In the following chapter, in support of my proposal for its adoption, four wildlife cancer case studies, including the Tasmanian devil cancer, are analysed.

The plantation forestry industry in Tasmania

The Tasmanian economy is heavily dependent on the forestry industry for export trade and jobs. It has therefore become normal to think of forestry interests as the major interests in Tasmania. This perception or worldview has shaped the economic, cultural,

social and scientific thinking of Tasmania. Historically native and old growth forests have been logged for timber and wood chips but this has engendered intense opposition from environmentalists. In order to alleviate the political situation and continue to rely on forest products the Australian federal, territory and state governments introduced a plan in the mid-1990s, which was revised in 2002, called the *Plantations for Australia: The 2020 Vision*.[25] In Tasmania this initiative has seen a massive growth in plantations facilitated by the *Tasmanian Regional Forest Agreement 1997 (RFA)* and by the state's introduction of the *Forestry Growth Plan* developed by Forestry Tasmania.[26] Commencing in the north-east of the state, plantations have now spread to occupy 44 of the 48 water catchments. Plantation forests are championed as the solution to saving old growth and native forest from destruction but as will be demonstrated they are beset with a whole new set of environmental problems.

A history of contamination of waterways in Tasmania

The intensification of plantation forestry has correlated with an increase in surface and drinking water contamination and an increase in wildlife diseases and human health problems.[27] The introduction of the *Vision 2020* provided the incentive for an increase in plantation forests of both soft and hard wood forests but especially eucalypts. Eucalypt plantations are the focus of my research because they suffer heavy predation from native species, which leads to a greater need for pesticide use. Pesticides are aerially sprayed on plantation forests increasing the potential for off-target dispersion of chemicals. In an early study by Peter Davies and colleagues on the potential for

[25] Australian Government, 2002, *Plantations for Australia: The 2020 Vision*, Department of Agriculture, Fisheries and Forestry Available at http://www.plantations2020.com.au/Index.html last accessed 30 September 2007

[26] Australian Senate Rural and Regional Affairs and Transport Legislation Committee, 2004, *Australian forest plantations, A review of Plantations for Australia: The 2020 Vision,* Department of the Senate, Canberra

[27] *Environmental Problems Georges Bay, Tasmania: Collated by Dr Marcus Scammell from information Gathered, in Particular, Between February 2004 to June 2004 [The Scammell Report]*, 2004, Hobart, Tasmania

chemicals such as the triazines, atrazine and simazine to contaminate waterways, they found that between 1989 and 1992, 20 of the sampled 29 streams draining plantation forests contained detectable residues of these chemicals.[28] It was therefore known early in the establishment of plantations that the potential for water contamination from the use of pesticides existed.

However, it was not until 2004 when a flood event in the George River coincided with a helicopter crash that the full extent of the risk of pesticide use in plantations became apparent. These events resulted in a huge loss of commercial oysters and other aquatic and terrestrial organisms in the inter-tidal zones of the Georges Bay at St Helens on the east coast of Tasmania. This was not an isolated incident. There had been numerous reports of water contamination of both municipal drinking water and private property tanks over the years. The most serious was when the domestic drinking water supply to the town of Lorinna was contaminated and people were poisoned.[29]

Tasmanian government reports continue to be produced and the government monitoring of water for contamination continues to detect pesticides used in plantations, but no decisive action to restrict the use of these chemicals or ban aerial spraying has been taken.

The need for improved chemical regulations

Chemicals in Australia are controlled at both national and state levels of government. Nationally the Australian government's Australian Pesticides and Veterinary Medicines

[28] Davies PE, Cook LSJ & Barton JL, 1994, Triazine Herbicide Contamination of Tasmanian Streams: Sources, Concentrations and Effects on Biota, *Australian Journal of Marine and Freshwater Research*, Vol 45(2), pp 209-226

[29] *Sunday,* 2004, television program, Ninemsn, 26 September, accessed 14/5/2007, http://sunday.ninemsn.com.au/sunday/cover_stories/article_1649.asp

Authority (APVMA) is the body authorized to register chemicals and set safe use labeling. At the state level regulators control the use, according to the label, of chemicals in either agricultural or forestry practices. There are currently approximately 38,000 chemicals in use in Australia. In Tasmania chemicals used as active ingredients in plantation forests are included in an 18-page list of products registered by the APVMA.[30] Even this extensive list omitted terbuthylazine, fluazifop and 1080, all known to be used in Tasmanian plantation forests. Terbuthylazine being used under a Research Permit and fluazifop being used under an off-label permit are not registered chemicals whilst it is unclear why 1080 was omitted. Many of these chemicals continue to be detected by government water monitoring.

Chemical regulators both in Australia and overseas are being increasingly challenged to update their current risk assessment criteria.[31] This includes assessing chemical mixtures, cumulative risk and broadening the criteria from chemical toxic effects (where the dose determines the level of harm) to include effects, where chemicals are harmful below current toxicity testing levels. At present regulators assess chemicals individually and according to their toxic effects but it has recently been established that chemicals which act as endocrine disrupters (which mimic hormones and interfere with developmental and reproductive functions at critical times) cause harm at extremely low levels at parts per billion and lower.[32] Chemical regulators both in Australia and overseas have been slow to implement these new risk assessment regimes.

---

[30] Australian Government Senate Rural and Regional Affairs and Transport Legislation Committee, Answers to Questions on Notice, Budget Estimates May 2009, Agriculture, Fisheries and Forestry, Australian Pesticides and Veterinary Medicines Authority, Response to Question on Notice, Question: APVMA06 Attachment 1, *Hansard*, 26 May 2009, p 96

[31] Assessing Risk Posed by Chemicals in Mixtures, *Health & Environment*. Available at: http://healthandenvironmentonline.com/2011/07/29/assessing-risk-posed-by-chemicals-in-mixtures/ last accessed 23 August 2011

[32] deFur, PL, 2004, Use and Role of Invertebrate Models in Endocrine Disruptor Research and Testing, *ILAR Journal*, 45(4), pp 484-493

Consequently regulators are operating with huge gaps in their knowledge whilst trying to protect the environment and human health from the effects of harmful chemicals. One reason for the delay in change appears to be an undue influence on regulators by vested interests.

I explore this situation through the framework of regulatory capture, revealing that both the United States (US) and Australian regulatory bodies are persuaded by the chemical industry to continue the registration of harmful chemicals. In the US the Environmental Protection Agency (EPA) oversees the registration of pesticides. This process examines 'the ingredients of a pesticide; the site or crop on which it is to be used; the amount, frequency and timing of its use; and storage and disposal practices'.[33] Similarly, in Australia the national body the Australian Pesticides and Veterinary Chemicals Authority (APVMA) registers agricultural chemical products before they can be legally supplied, sold or used.[34] A key aspect of this registration process is the assessment and approval of labels for use. One chemical in particular, atrazine, has been the site of controversy in both the US and Australia over its reregistration. It has been banned in Europe under the new regulatory program Registration, Evaluation, Authorisation and Restriction of Chemical substances (REACH) using the precautionary principle. In the US the manufacturer of atrazine, Syngenta, has used industry science, suppression of knowledge and political influence to prolong the registration of this chemical. In Australia the APVMA follows the lead set by the US EPA in the regulation of atrazine.

[33] United States Environmental Protection Agency, Registering Pesticides. Available at: http://www.epa.gov/pesticides/regulating/registering/ last accessed 3 April 2014
[34] Australian Government, Australian Pesticides and Veterinary Medicines Authority, Product Registration. Available at: http://www.apvma.gov.au/registration/index.php last accessed 3 April 2014

In the regulation of chemicals it appears that at both the national and state levels the regulatory processes have become what Murray Edelman describes as 'symbolic'.[35] The APVMA and state government regulators' role is essentially to protect the public from the excesses of industry. When they fail they inadvertently protect particular interest groups, either the chemical industry or the forestry industry. The impediments to the regulation of atrazine are the topic of chapter 8.

Conflict of interest in Tasmanian forestry practices

The role of regulatory bodies is to protect the environment and human health from the excesses of industrial activities, not to protect industries from profit loss. However, in Tasmania it appears the government is primarily committed to aiding the forestry industry. This outcome is partly achieved through a forest practices system based on a co-regulatory approach. This involves self-regulation by the industry with the government Forest Practices Authority monitoring and enforcing compliance. Under this system the responsibility for protection of people and water resources from use of chemicals, according to the *Forest Practices Code 2000*, is devolved to the forest owners.[36] This means it is the responsibility of the forest owner or manager to ensure that the appropriate chemicals are used and that they are applied correctly. Non-compliance or breaches of the Code are enforced but only infrequently with only 10 cases in the period 2009-2010.[37] There were no fines related to chemical use during that time although monitoring of streams from plantations continues to detect pesticides.

When the same government department, in this case DPIPWE, is responsible for the use of agricultural chemicals used in plantation forests and the Save the Tasmanian Devil

[35] Edelman M, 1985, *The Symbolic Uses of Politics*, University of Illinois Press, Urbana and Chicago
[36] Forest Practices Board, 2000, *Forest Practices Code*, Forest Practices Board, Hobart, Tasmania
[37] Forest Practices Authority, *Annual Report on Forest Practices 2009-10,* Forest Practices Authority, Hobart, Tasmania

Program (STDP) then a conflict or interest is evident. The STDP also works closely with the University of Tasmania (UTAS) in particular the Zoology Department. A close relationship also exists between the forestry industry and UTAS through the Cooperative Research Centre for Forestry, which works to enhance and develop all areas of the industry including eucalypt plant development.[38] This close association means that both the industry and university researchers and students have developed a mutual reliance, the industry for knowledge and the university for funding. This symbiotic approach works well in many universities where industry funding enables important research but when there is a conflict of interest, when the research findings may have negative implications for the funding industry, then the relationship can have a chilling effect on research decisions.

Scientific uncertainty in DFTD and the precautionary principle

The scientific research into the devil disease has been shaped so as to promote the allograft theory of a transmissible cancer. But all avenues of scientific research must be fully explored if the Tasmanian devil is to avoid the threat of extinction from this deadly cancer. In an environment contaminated by chemicals used in plantation forestry the relevant toxicology studies should be undertaken to assess the possibility that they are involved in the initiation or progression of the disease. The undone research has exacerbated the uncertainty surrounding the devil cancer and in this situation the precautionary principle, as a foundational principle of the *EPBC Act,* needs to be implemented. It would then be possible for decision makers to mitigate the likely irreversible harm and direct the appropriate research if the Tasmanian devil is to be saved from extinction.

---

[38] CRC for Forestry, nd. *CRC for Forestry technical reports series.* Available at: http://www.crcforestry.com.au/publications/technical-reports/index.html last accessed 30 September 2013

Meanwhile, the Tasmanian government must stand at arms length from the research, which needs to be assessed by scientists independent of industry and government influence. The forestry industry must accept the limitations to its expansion and profit margin to ensure that both the environmental and human health in Tasmania is not threatened by its activities. This case study is a view of what happens in a social, cultural and economic environment where contrary voices are stifled, science is hindered and industry interests dominate. This thesis argues that all avenues of scientific research must be fully explored if the Tasmanian devil is to be saved; that improved regulations of forestry activities and use of chemicals must be implemented to mitigate harm; and suppression of dissent must end to allow all voices to be heard to restore the overriding principles of the scientific process.

Chapter 1 – The Tasmanian devil (*Sarcophilus harrissi*)

1.1 Introduction

A healthy environment is important for maintaining the wellbeing of human and wildlife populations. Increasing destruction and degradation of the environment is, however, leading to species extinction and the emergence of infectious diseases, which are two of the more serious global concerns facing humanity.[1] These processes are tightly intertwined, with parasitic and microbial infections acting as a cause for, and possibly attributing to, biodiversity loss. Hence there is a need for a better understanding of the environmental co-factors that facilitate the spread of disease or the susceptibility of hosts. In this worldwide phenomenon, the small island state of Tasmania, which forms part of the larger nation of Australia, is not exempt.

In Tasmania there are significant threats to a broad range of native fauna species including the critically endangered orange-bellied parrot and the endangered spotted-tailed quoll, giant freshwater crayfish as well as flora species including the critically endangered windswept spider orchid.[2] The threatened extinction of the Tasmanian devil is the focus of

---

[1] Kiesecker JM, Belden LK Shea K & Rubbo MJ, 2004, Amphibian Decline and Emerging Disease, What can sick frogs teach us about new and resurgent diseases in human populations and other species of wildlife? *American Scientist*, Vol 92, pp138-147

[2] Australian Government Department of the Environment, EPBC Act List of Threatened Fauna. Available at: http://www.environment.gov.au/cgibin/sprat/public/publicthreatenedlist.pl?wanted=fauna#birds_critically_endangered last accessed 3 April 2014

this research. In this chapter I provide an historical review of the published research on the Tasmanian devil population unrelated to the cancer and will give an account of the developments in the research into the Tasmanian devil facial tumour disease.

1.2 Tasmanian devil – what is known?

The devil disease DFTD has received wide media coverage and public attention but this tends to obfuscate the fact that little scientific knowledge exists on the species in the wild. Much of the research is based on observations of devils in captivity. In order to gain a better understanding of the devil cancer it is useful to review what is known about the devil physiology, habits and evolution.

In 1969 Eric Guiler then a zoologist at the University of Tasmania was the first to write an account of devils in the wild.[3] David Pemberton, Vertebrate Curator at the Tasmanian Museum and Art Gallery and Deane Renouf, a Canadian harbor seal expert, wrote the only other account of the devils in the wild in 1993.[4] David Owen and David Pemberton in 2005 wrote the most comprehensive book about the devils, when the threat of devil extinction was becoming a reality. Their book was prefaced with the statement 'it is the world's largest living marsupial carnivore, about which we have limited understanding'.[5] A brief summary of these publications and other papers on the Tasmanian devil is given in the following paragraphs.

---

[3] Guiler ER, 1970, Observations on the Tasmanian Devil, Sarcophilus harrisii, *Australian Journal of Zoology*, Vol 18, pp 49-62

[4] Pemberton D & Renouf D, 1993, A Field Study of Communication and Social Behaviour of the Tasmanian Devil at Feeding Sites, *Australian Journal of Zoology*, Vol 41(5), pp 507-526

[5] Owen D & Pemberton D, 2005, *Tasmanian Devil, a unique and threatened animal,* Allen & Unwin, Sydney, p 1

The Tasmanian devil, shown in Figure 1:1 below, is now the largest surviving carnivorous marsupial, as the former largest, Thylacine (Tasmanian tiger), became extinct in the 1930s. The devil was first described in 1808 and given the zoological name *Sarcophilus harrisii* by Pierre Boitard in 1841.[6] They are a short-lived species with an average life span in the wild of up to 5 years, are approximately 51 to 70 cm in length and weight between 4 and 12 kilograms.[7] The earliest white settlers in the then named Van Diemen's Land gave it the name Devil or Native Devil because of its 'forbidding expression and black colour'.[8] Devils inhabited Tasmania long before Europeans arrived as indicated by the recording of the Bruny Island and Southern tribes of Aborigines in Tasmania who gave it the name '*tarrabah*'.[9]

Figure 1:1 The Tasmanian Devil[10]

[6] Morton SR, Dickman CR & Fletcher TP, 1989, 'Dasyuridae' in DW Walton & BJ Richardson (eds), *Fauna of Australia,* Volume 1B, Australian Government Publishing Service, Canberra,
[7] National Geographic, Tasmanian Devil (Sarcophilus harrisii). Available at: http://animals.nationalgeographic.com.au/animals/mammals/tasmanian-devil/ last accessed 30 November 2013
[8] Troughton E, (ed.), 1967, *Furred Animals of Australia*, Angus and Robertson, Cremorne p 43
[9] Guiler ER, 1992, *The Tasmanian Devil*, St. David's Park Publishing, Hobart, Tasmania
[10] Source: Dave Walsh. Available at: http://davewalshphoto.com/2011/06/19/tasmanian-devils-sierra-magazine/ last accessed 3 June 2013

Tasmanian devils belong to the mammal group but unlike placental mammals its young are born at a very early developmental stage and migrate to a pouch. Pouched mammals are known as marsupials. Marsupial fossils have been found in Australia from the Oligocene epoch, which lasted from about 33 to 23 million years ago, before Antarctica and Australia split into two continents. Today they only inhabit the continents of South America and Australia but are known to have once existed in North America where only one species survives. The Tasmanian devil is a member of the Family Dasyuridae and Sub-family dasyurinae and is regarded as the Australian group most like the original marsupials because many of their morphological characters appear to have retained the primitive state.[11] Tasmanian devils once roamed the mainland of Australia becoming extinct there approximately 400 years ago. Their extinction is speculatively attributed to the introduction of the dingo (wild dog) but research by Johnson and Wroe proposes that an increase in population and resulting human activity may have also had a significant impact.[12] There are remains of dasyurids from the Pleistocene era, 1.8 million to the Holocene 10,000 years ago, in every Australian state.[13] The dasyurid fauna inhabiting Australia today is the end-product of alteration and adaptation over the past half a million years.[14] Tasmanian devil fossils have been found in caves on the mainland and probably migrated to Tasmania over the land bridges that existed at various times.

---

[11] Morton SR, Dickman CR & Fletcher TP, 1989, 'Dasyuridae' in DW Walton & BJ Richardson (eds), *Fauna of Australia,* Volume 1B, Australian Government Publishing Service, Canberra
[12] Johnson CN & Wroe S, 2003, Causes of extinction of vertebrates during the Holocene of mainland Australia: arrival of the dingo, or human impact? *The Holocene*, Vol 13, pp 941-948
[13] Cited Dawson, 1982a, ibid
[14] Morton SR, Dickman CR & Fletcher TP, 1989, 'Dasyuridae' in DW Walton & BJ Richardson (eds), *Fauna of Australia,* Volume 1B, Australian Government Publishing Service, Canberra

In his 1967 edited book *Furred Animals of Australia*, Ellis Troughton grouped Tasmanian devils with Native and Tiger Cats (quolls) under the sub-family Dasyurinae. Although both the devils and the quolls differ in appearance they are 'linked by a common origin, shown in a progressive adaptation of the teeth for a flesh diet, and the similarity of structure of the ear, muzzle, and palm- and sole-pads. They are also similar in having but two premolars each side, above and below'.[15] According to Tasmanian zoologist Menna Jones and others scientists, including Charles Darwin, the dentition of the devils is similar to that of the canine (dog species). However the devils' canine teeth are almost circular or rounded in cross-section due to their bone-eating habits.[16] Devils have extremely powerful jaws but are incapable of chewing through the largest bones of their prey. For example, they are known to eat wombat but leave the backbone and adjoining skin, being too tough to chew.[17] This is also 'consistent with their strong, crushing, but generally non-penetrating killing bite, to the chest, head or nose of the prey'.[18] Devils swallow their food in chunks and their digestive system finishes the job of breaking it down. They also exhibit cat like qualities using both paws for washing their faces, 'placing them together to form a cup-like depression which is thoroughly licked and rubbed over the head' as can be seen in Figure 1:2 below.[19]

---

[15] Troughton E, (ed.), 1967, *Furred Animals of Australia*, Angus and Robertson, Cremorne, Sydney
[16] Jones ME, 2003, Convergence in Ecomorphology and Guild Structure among marsupial and placental carnivores in M Jones, CR Dickman & M Archer, 2003, *Predators with Pouches: The Biology of Carnivorous Marsupials,* Publishing, Collingwood, Victoria, p 292
[17] Owen D & Pemberton D, 2005, *Tasmanian Devil, a unique and threatened animal,* Allen & Unwin, Crows Nest, Sydney, p 20
[18] ibid,
[19] ibid, p 43

Figure 1:2 Tasmanian devil washing its face[20]

Devils are mainly scavengers but are also known as ambush predators.[21] They are known to scavenge and eat almost anything from grasses to grubs but are particularly fond of native species such as the pademelon (a small wallaby), they will also eat carrion and will even eat their own kind. Devils store fat in their tails. They like to drink water.

Devils have been found across Tasmania in their more favoured habitats. In Guiler's study he found the density of devils in one location to be approximately thirty animals per two and a half square kilometres - but this was unusually high.[22] In a study by Menna Jones and colleagues they estimated the potential core distribution of devil population densities to

---

[20] Source: http://media.popularmechanics.com/images/PMX0705TW007_smasll.jpg last accessed 29 May 2007

[21] Owen D & Pemberton D, 2005, *Tasmanian Devil, a unique and threatened animal,* Allen & Unwin, Crows Nest, Sydney. p 22

[22] Guiler ER, 1970, Observations on the Tasmanian Devil, Sarcophilus harrisii, *Australian Journal of Zoology*, Vol 18, pp 49-62

be approximately one devil per two square kilometres.[23] Population densities are low in dense wet forests, low heathlands, alpine areas, open grasslands and extensively cleared farmland.[24] They are more abundant in open eucalypt forests, woodlands and coastal scrub where dense populations of their prey - wallaby, wombats and possums - are found.

1.2.1 Tasmanian devil social habits

Devils are solitary not social animals; they do not live in organised groups.[25] Guiler found that devils are also not territorial, moving from one area to another in search of food, staying under cover but using open tracks and roads to transit. David Croft from the University of New South Wales noted, although most carnivorous marsupials are solitary species, they still need to mate.[26] It is during mating and parent-offspring encounters that most devil tactile communication occurs. Tactile communication or touching is important for dasyurids particularly for the mother and offspring. The mother devil licks and cleans both her pouch and her young in it and the licking also stimulates the young to urinate and defecate. It is also important in mating when the male 'paw-on-partner' contact is used to test for receptivity of the female.[27] During mating the male holds the female around the abdomen and in a neck-grip used across the dasyurid species.

---

[23] Jones ME, Paetkau D, Geffen G & Moritz C, 2004, Genetic diversity and population structure of Tasmanian devils, the largest marsupial carnivore, *Molecular Ecology*, Vol 13, pp 2197-2209
[24] ibid.
[25] Pyers G, 2005, *Life Cycles of Australian Animals*, *Tasmanian Devils*, Echidna Books, Melbourne
[26] Croft DB, 2003, 'Behaviour of Carnivorous Marsupials' in M Jones, CR Dickman & M Archer, 2003, *Predators with Pouches: The Biology of Carnivorous Marsupials,* CSIRO Publishing, Collingwood, Victoria, p 337
[27] ibid, p 339

1.2.2 Tasmanian devil mating behaviour

The Tasmanian devil is a monoestrous species, mating only once a year in March/April with the young being born in May.[28] Ronald Strahan in his book *The Mammals of Australia* also noted that the devil breeding is highly synchronized, with the young starting to leave the den in November and fully independent by February.[29] However, mortality is high in the first year of life. Greg Pyers in his book *Life cycles of Australian Animals, Tasmanian Devil* noted up to 60% of young devils die in this first year.[30] Young devils eat mainly insects and occasionally a mouse.[31] Most female devils breed at 2 years and both sexes grow to adult size by 2-3 years. Male devils rather than female devils leave the area in which they were born. This, according to biologist Peter Slater, University of St Andrews, discourages inbreeding.[32]

1.2.3 Tasmanian devil feeding habits

Devils forage for food singly although several individuals may feed simultaneously on a large carcass, giving rise to much squabbling and although this does not result in physical contact most of the time, the occasional bite can be substantial.[33] Although devils are solitary if they find a large carcass they will feed together. This communal feeding practice is well structured and is properly described as ritualised behaviour. The apparent fighting is an elaborate combination of eleven vocalisations (sounds) and 20 postures (visual) that

---

[28] Jones ME, Dickman CR & Archer M, 2003, *Predators with Pouches: The Biology of Carnivorous Marsupials,* CSIRO Publishing, Collingwood, Victoria
[29] Strahan R, (ed), 1995, *The Mammals of Australia* (2nd Ed), Australian Museum/Reed New Holland, Sydney
[30] Pyers G, 2005, *Life Cycles of Australian Animals*, *Tasmanian Devils*, Echidna Books, Melbourne
[31] Markle S, 2005, *Animal Scavengers, Tasmanian Devils*, Lerner Publications Company, Minneapolis
[32] Slater, PJB, 1999, *Essentials of Animal Behaviour*, Cambridge University Press, Cambridge, UK
[33] Strahan R, (ed), 1995, *The Mammals of Australia* (2nd Ed), Australian Museum/Reed New Holland, Sydney, p 83

maintain order.[34] The sounds emitted by the devils alert other devils to join in feeding - the greater the noise the bigger the carcass.[35] This noise often alerts their cousins the quolls to join in the feeding also. However, in a study at Cradle Mountain in 1993 Jones noted, when it came to competing for food, devils were dominant over quolls at large food sources.[36] The study noted that devils feed primarily on large mammals such as wallabies and wombats and secondarily on medium-sized mammals. No mention is made in this study, over two and half years, of devils causing physical injury to each other or the quolls at these encounters.

1.2.4 Tasmanian devil aggressive behaviour

Recent media images and stories of devils have focused on their apparent savage, biting and snarling habits, when in fact the record shows they are generally timid, sensitive and easily subdued animals. Guiler who handled more than 7,000 devils 'found them docile to the point of being lethargic and could be handled with ease'.[37] However, Guiler's observations published in 1970 did note '[i]ntraspecific fighting results in severe facial injuries and may lead to death'.[38] Contrary to this, in his book *The Tasmanian Devil* published in 1992, Guiler states '[f]eeding is accompanied by much squabbling, loud

---

[34] Pemberton D & Renouf D, 1993, A Field Study of Communication and Social Behaviour of the Tasmanian Devil at Feeding Sites, *Australian Journal of Zoology*, Vol 41(5), pp 507-526 p 507
[35] Owen D & Pemberton D, 2005, *Tasmanian Devil, a unique and threatened animal,* Allen & Unwin, Crows Nest, Sydney, p 13
[36] Jones ME and Barmuta LA, 1998, Diet overlap and relative abundance of sympatric dasyurid carnivores: a hypothesis of competition, *Journal of Animal Ecology*, Vol 67, pp 410-421
[37] Nowak RM, 1999, *Walker's Mammals of the World*, Baltimore, Johns Hopkins University Press, p 64 cited in D Owen & D Pemberton, 2005, *Tasmanian Devil, a unique and threatened animal,* Allen & Unwin, Crows Nest, Sydney
[38] Guiler ER, 1970, Observations on the Tasmanian Devil, Sarcophilus harrisii, *Australian Journal of Zoology*, Vol 18, pp 49-62, p 60

screams, growls, jaw chomping, jostling and general aggression'[39] but '[n]ot much serious damage is inflicted except for nips and bites, much of the aggression being a ritualistic display'.[40] Guiler concluded that intense competition for limited food resources might have been the cause. Lack also attributed the possible causes of fighting to food shortages.[41]

Pemberton and Renouf in their three year study of devils in the wild, the first description of wild devils' social interactions, found little physical damage resulting from communal feeding and little evidence of injury in animals they trapped.[42] The study was carried out at Mt William National Park where it was estimated over 200 devils were present.[43] The trapping occurred every four months over the study period. Examination of trapped animals showed that 29.5% had scars or open wounds with all but one appearing on males. The records of physical damage are shown in the Table 1:1 below. Of the wounds only 6% were recorded as open and bleeding. Overall the damage sustained to the muzzle (48.4%) was the equivalent to that sustained to other parts of the body. Moreover, in a study of 119 interactions at a feeding site, set up by the researchers, only one encounter resulted in physical damage and that was to the rump of a fleeing animal.

---

[39] Guiler ER, 1992, *The Tasmanian Devil*, St. David's Park Publishing, Hobart, Tasmania, p 8
[40] ibid.
[41] Lack D, 1954, *The Natural Regulation of Animal Numbers*, Clarendon Press, Oxford cited in ER Guiler, 1970, Observations on the Tasmanian Devil, Sarcophilus harrisii, *Australian Journal of Zoology*, Vol 18, pp 49-62,
[42] Pemberton D & Renouf D, 1993, A Field Study of Communication and Social Behaviour of the Tasmanian Devil at Feeding Sites, *Australian Journal of Zoology*, Vol 41(5), pp 507-526, p 519
[43] Cited Pemberton, D, 1990 in ibid.

Table 1:1 Frequency of occurrence and location of scars and wounds on male and female Tasmanian devils[44]

Wound and scar location	No of males with scars	No of females with scars
Muzzle	43	16
Ears	4	4
Shoulders	2	0
Claws missing	3	2
Legs	1	0
Back	7	5
Rump	15	4
Tail	12	4

Aggressive behavior in animals both in attack and defence is found in two areas, sexual competition and resource competition. Males compete for the chance to mate and for food whilst females compete for food. This behaviour is typically accompanied by visual signals and devils display an 'open-mouth threat' that reveals their teeth, especially canines, as shown in Figure 1:3 below, and is usually accompanied by a harsh vocalisation and a raised forepaw.[45] They also neck-threat, nip in the direction of another's neck, and walk stiff-legged.[46] These displays constitute a typical high intensity threat with maximum exposure of weaponry. Devils can open their mouths 120 degrees whereas a dog can only open its mouth 70 degrees.[47]

---

[44] Pemberton D & Renouf D, 1993, A Field Study of Communication and Social Behaviour of the Tasmanian Devil at Feeding Sites, *Australian Journal of Zoology*, Vol 41(5), pp 507-526, p 521
[45] ibid, p 515
[46] ibid, p 512
[47] Pyers G, 2005, *Life Cycles of Australian Animals*, Tasmanian Devils, Echidna Books, Melbourne

Figure 1:3 Tasmanian devil open-mouth threat[48]

In his book *Essentials of Animal Behaviour* Peter Slater notes natural selection matches behaviour extremely well to an animal's particular environment and way of life.[49] If biting proved detrimental to the devil population it would have ceased being an inherited display. However, he points out that there are factors, which can affect aggression including hormones, shortage of food, presence of rivals and contested resources.

Devils are not the only animals that display aggressive behaviour. Other animals display an armoury of antlers, horns or teeth that a rival risks encountering, if it engages in a fight. As

[48] Tasmanian Devil at Taronga Zoo, Photo: Rick Stevens
[49] Slater, PJB, 1999, *Essential of Animal Behaviour*, Cambridge University Press, Cambridge, UK

Slater states,

> [a]ggression becomes easier to understand if individuals act only for their own good, indeed one might expect them to fight a tremendous amount the whole time, each being out for its own ends and careless about possible damage to others. This certainly does not occur, but the reason is probably simply just that fighting is dangerous.[50]

Aggression tends to be limited where it could have a dangerous outcome for either of the participants. It is therefore more usual for animals to display and threaten until the other retreats.[51] As noted previously devils do not defend territories, eliminating the need to fight over territory.

Devils that did incur injuries, Guiler observed, had incredible recuperative powers from both tissue and bone damage, which meant that any damage was not sustained long term. In observing severe wounds in poisoned devils, Guiler observed one devil with 'both frontal bones shattered over the brain leaving a hole' in the skull and a second devil with a wound from a .22 bullet, both had recovered from their injuries before succumbing to deliberate chemical poisoning.[52] In 1992 he concluded that the main cause of premature death for devils was through human activities such as poisoning and trapping.

The recent media images, both photographs and films of devils, are taken in captivity. In this artificial environment devil behaviour is not in response to its natural environment.

---

[50] ibid, p 150
[51] ibid, p 151
[52] Guiler ER, 1992, *The Tasmanian Devil*, St. David's Park Publishing, Hobart, Tasmania, p 12

Animals in captivity are generally more socially intolerant.[53] Hence the devil's reputation as an aggressive and fierce animal, ready to bite at the least provocation, may be due to the fact that most observations of devils have been in captivity in close proximity to other devils. In their natural wild state the evidence suggests they are predominately nocturnal and solitary creatures. The Tasmania devil species has survived thousands of years of natural environmental change and adaptation to now face extinction from a deadly cancer. Other changes in the environment, as has been suggested – increased pressure for food, higher density of population and more aggressive behavior - might have accounted for an increase in biting and contributed to the transmission of the cancer but as will be shown there is no evidence that this is the case.

1.3 The Tasmanian devil cancer

The malignant and deadly cancer termed DFTD is decimating the Tasmanian devil population. The only unaffected populations are isolated on the west coast. Devil numbers have been reduced in some areas by over ninety percent particularly in the northeast, where the disease was first identified in 1996. Christo Baars, a wildlife photographer, then working for the Australian Antarctic Division,[54] captured the first images of a devil with the cancer in the Mt William National Park in the far north east of the state.

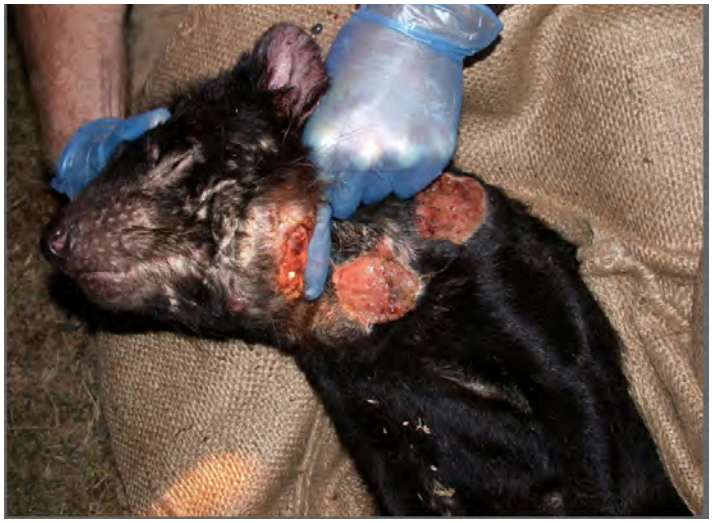
---

[53] Croft DB, 2003, 'Behaviour of Carnivorous Marsupials' in M Jones, CR Dickman & M Archer, 2003, *Predators with Pouches: The Biology of Carnivorous Marsupials,* CSIRO Publishing, Collingwood, Victoria, p 337

[54] Australian Antarctic Data Centre, Taxon Documents and Images. Available at: https://data.aad.gov.au/aadc/biodiversity/taxon_documents.cfm?taxon_id=1060 last accessed 25 November 2012

In 1999 Menna Jones observed another devil with the cancer 250 kilometres south of the first location. Devils with the facial cancer continued to be identified across the eastern part of Tasmania but it would be seven years before the Tasmanian government was convinced of the need to investigate the disease. A devil with the facial cancer is shown in Figure 1:4 below.

Figure 1:4 Tasmanian devil with facial cancer[55]



In October 2003 the Tasmanian government, through the DPIPWE and following the noticeable decline in devil numbers, convened an urgent meeting of wildlife specialists to develop a strategy to address the problem.[56] The meeting was conducted with the exclusion of television, radio or newspaper journalists who were told they could not attend, talk to scientists or report on the meeting.[57] However, following the meeting a brief communique

---

[55] Source: Richmond Loh Cern-Wan, Loh, R, 2006, *The Pathology of Devil Facial Tumour Disease in Tasmanian Devils (Sarcophilus harrisii)* Master of Philosophy, Murdoch University Perth, Western Australia
[56] Darby A, 2003, Search for what in the Tasmanian devil is killing them, *Sydney Morning Herald*. Available at http://www.smh.com.au/articles/2003/10/13/1065917349653.html?from=storyrhs last accessed 9 December 2009
[57] Personal communication.

from DPIPWE was provided to Rohan Wade, a journalist with the *The Mercury*, the daily paper in Hobart, Tasmania.

In February 2005 the DPIPWE released the *Tasmanian Devil Facial Tumour Disease (DFTD) Disease Management Strategy.* It reported a scientific consensus amongst the researchers that the cancer was a neuro-endocrine tumour of unknown origin.[58] In the same year DPIPWE published a *Progress Report* identifying key areas for investigation; the relevant fields included haematology, blood biochemistry, immunology and endocrinology and the identification of the aetiology (cause) of the disease.[59] A viral aetiology was discounted because a test for virus particles had proved negative but a trial to test for a range of chemical toxins was proposed. This research was to investigate if toxins or poisons were the cause of the chromosome instability in DFTD.[60] This need for chemical testing was reported in a local newspaper by Simon Bevilaqua: '[i]t has been speculated that a chemical in the environment, maybe a farm or forestry pesticide or herbicide, has triggered development of the cancerous cell line in one, or a handful, of devils'.[61] Also recommended for future investigations were transmission trials for the passage of tumour cells to determine whether the cancer was transmissible.

---

[58] Loh, R, 2006, '*The Pathology of Devll Facial Tumour Disease in Tasmanian Devils (Sarcophilus harrisii)'*, MPh, Murdoch University, Perth, p. 90. Available at http://wwwlib.murdoch.edu.au/adt/pubfiles/adt-MU20061019.131524/01Front.pdf last accessed 18 September 2007

[59] Research into the Tasmanian Devil Facial Tumour Disease (DFTD), *Progress Report*, 2005, Tasmania Department of Primary Industries, Water and Environment, Hobart

[60] See Appendix A

[61] Bevilaqua S, 2006, Difficult devil science, *The Sunday Tasmanian*. Available at:http://www.news.com.au/mercury/story/0,22884,18824659-3462,00.html last accessed 28 October 2007

In 2006 a novel hypothesis was proposed: that the devil cancer was a transmissible tumour – an allograft – spread from devil to devil via biting when they mate or feed. Anne Maree Pearse conducting cytogenic research at the Tasmanian Government DPIPWE Mt Pleasant laboratory in Launceston had arrived at this hypothesis from an observation in one devil. Pearse had observed a chromosomal anomaly (a peri-centric inversion of chromosome 5) in all the cells of one devil that was not observable in any of its tumour cells where it would have been expected if the cancer had been initiated within its own body. Pearse and her laboratory assistant Kate Swift published these findings, the basis for the allograft theory, in the Brief Communications section of the prestigious scientific journal *Nature* in February 2006.[62] In proposing that it was a transmissible tumour, they still acknowledged in their conclusion that a carcinogen may have been the initial cause of the disease.

1.4 What is Devil Facial Tumour Disease (DFTD)?

Devil facial tumour disease (DFTD) is the official term given to the devil cancer. The disease has been clinically described as 'lesions occurred subcutaneously and form circumscribed masses with a flat ulcerative surface'.[63] In other words the lesions occur just under the skin[64] and often as shown in Figure 1:5 below under the tongue. Death occurs within five months, resulting from a breakdown in bodily functions or starvation.[65] Also

---

[62] Pearse AM & Swift K, 2006, 'Transmission of devil facial-tumour disease' *Nature* Vol 439(2), p 549
[63] Loh, R, 2006, '*The Pathology of Devll Facial Tumour Disease in Tasmanian Devils (Sarcophilus harrisii)*', MPh, Murdoch University, Perth, p. 90. Available at http://wwwlib.murdoch.edu.au/adt/pubfiles/adt-MU20061019.131524/01Front.pdf last accessed 18 September 2007, p 33
[64] According to the Medicine.net. the definition of subcutaneous is under the skin. Available at http://www.medterms.com/script/main/art.asp?articlekey=8265 last accessed 9 June 2010
[65] Pemberton D & Renouf D, 1993, 'A Field Study of Communication and Social Behaviour of the Tasmanian Devil at Feeding Sites' *Australian Journal of Zoology*, Vol 41(5), pp 507-526

associated with the disease is altered reproductive behaviour resulting in devils breeding at a younger age and with births scattered across the seasons.[66]

Figure 1:5 Cancer in the mouth seen here as a lesion under the tongue[67]

The pathology of the disease has yet to be confirmed but the original consensus strongly supported a tumour of neuroendocrine origin. This raised the possibility of chemical involvement particularly the type of chemicals used as herbicides in Tasmanian forestry.[68] Richmond Loh, who at the time was a Tasmanian government DPIPWE pathologist, confirmed that there was 'strong evidence for classifying DFTD as an undifferentiated neuroendocrine tumour which is unlike any other seen in humans or animals'.[69] Loh also suggested that in devils the neuroendocrine tissues are derived from the embryonic neural crest, they are widely dispersed throughout the body and they are in especially high

---

[66] ibid
[67] Source: Dr Richmond Loh (DPIPWE)
[68] The IARC has accepted that 'atrazine appears to disrupt neuroendocrine pathways in the hypothalamus by as yet undetermined mechanisms' International Agency for Research on Cancer, 1999, Atrazine. Available at http://www.inchem.org/documents/iarc/vol173/73-03.html last accessed 23 August 2007
[69] Loh R, 2006, '*The Pathology of Devll Facial Tumour Disease in Tasmanian Devils (Sarcophilus harrisii)'*, Master of Philosophy, Murdoch University, Perth, p. 90. Available at http://wwwlib.murdoch.edu.au/adt/pubfiles/adt-MU20061019.131524/01Front.pdf last accessed 18 September 2007, p 90

concentrations in tactile tissues such as the finger tips and lips (Meuten, 2002) and in the whisker-bed (Halata et al., 2003). Loh observed it was these sites where DFTD neoplasms most commonly originate.[70] [71]

Whilst the majority of scientists concluded DFTD was of neuroendocrine origin, Stephen Pyecroft had suggested that the initial classification of a lyphosarcoma, the malignant and abnormal growth of cells in the infection-fighting lympathic system, may have indicated a type of cancer that he suspected was caused by a virus.[72] But further investigations have not confirmed a viral cause.[73] Loh confirmed '[i]t's a confusing picture' because '[t]he cells look like lymphosarcoma but aren't strictly behaving that way'.[74] This opinion is supported by Clare Hawkins, wildlife biologist, also of DPIPWE and colleagues who concluded that the 'disease is an undifferentiated sub-epithelial sarcoma of possible neuroectodermal origin'.[75] A devil with lymphosarcoma tumours is shown in Figure 1:6 below.

---

[70] ibid, p 85

[71] Richmond Loh, a fish veterinarian, had been employed as a veterinarian for DPIPWE when Margaret Williams, his manager, provided him with the opportunity to enrol in a Masters degree at Murdoch University in Western Australia. He was to study the pathology of the Tasmanian devil cancer. His thesis was submitted in 2006. The project was funded by the DPIPWE, the Commonwealth Research Training Scheme and supported by the Australian Wildlife Health Network. His academic supervisors were Shane Raidal and Amanda O'Hara and his workplace supervisor was Stephen Pyecroft, veterinary pathologist with DPIPWE. Loh has since left Tasmania and the DPIPWE. Available at: http://www.thefishvet.com.au/index.html last accessed 25 November 2012

[72] *Australian Broadcasting Corporation Science Online Features*, n.d., Tassie Devil Terror. Available at http://www.abc.net.au/science/features/tassiedevil/default.htm last accessed 12 August 2007

[73] DFTD under the microscope, *Devil Facial Tumour Disease Newsletter,* March 2006, Tasmanian Government, Department of Primary Industries and Water. Available at http://tassiedevil.com.au/docs/devilNews_March2006.pdf last accessed 9 June 2010

[74] *Australian Broadcasting Corporation Science Online Features*, n.d, Tassie Devil Terror. Available at http://www.abc.net.au/science/features/tassiedevil/default.htm last accessed 12 August 2007

[75] Hawkins CE, Baars C, Hesterman H, Hocking GJ, Jones ME, Lazenby A, Mann D, Mooney N, Pemberton D, Pyecroft S, Restani M & Wiersma J, 2006, Emerging disease and population decline of an island endemic, the Tasmanian devil *Sarcophilus harrisii, Biological Conservation,* Vol. 131(2), pp 325-337, p 309

Figure 1:6 Devil with lymphosarcoma tumours[76]

Whilst the classification of the Tasmanian devil cancer remains confusing, devils are not only succumbing to DFTD. There are two other cancers afflicting devils, which to date have not been fully documented, a mammary cancer in female devils and a skin lymphoma shown in Figure 1:7 below.

[76] Source: Dr Richmond Loh, (DPIPWE)

Figure 1:7 Tasmanian devil with skin lymphoma[77]

Cancer is an extremely complicated disease involving a web of multiple causes but it is known that preventing exposure to known carcinogens prevents the disease.[78] Among the possible causes are environmental factors, including heavy metals, certain chemicals, viral agents, the effects of radiation, and the regulation of hormones on cell growth and differentiation. DFTD as a cancer is necessarily the subject of much speculation and uncertainty but this has been exacerbated by its framing as a new and emerging disease. DFTD manifests on the face and neck of affected devils, it is malignant, known to cause secondary cancers and is fatal in every case.

---

[77] Source: Pearse, AM *The trouble with devils* unpublished
[78] Clapp RW, Jacobs MM & Loechler EL, 2007, *Environmental and Occupational Causes of Cancer,* Lowell Center for Sustainable Production, Lowell, MA

1.5 The biting theory

Nick Mooney, a wildlife biologist with the Nature Conservation Branch of the DPIPWE, was the first to suggest that the devil cancer could be spread via biting. In an interview on the *ABC Science* radio program on 1 August 2003 he proposed it was spread when 'animals quarrel or mate sexually'.[79] At this early stage it was still a possibility that a virus could be the vector. However, based on this assumption he believed devils would not become extinct because isolated populations would survive. Menna Jones supported this assumption in an interview with Julia Limb on the *ABC The World Today* on 14 October 2003, when she claimed it was an infectious disease.[80] This interview coincided with the first workshop of scientists and disease experts held in Launceston, Tasmania. A flowchart from the meeting suggested that the cancer was a 'transmissible disease'.[81] A further report of the disease being infectious came again from Mooney when in April 2004, in an ethics application to the Tasmanian University (UTAS), he suggested that the disease had spread over much of the eastern half of Tasmania and that the 'infectious mechanism is not yet clear although infection rates suggest it is possibly highly infectious between devils'.[82]

However, the plausibility of an infectious cancer, spread via biting, was from the beginning confounded by anomalies. AusVet Animal Health Services Pty Ltd, a private company that provided epidemiological advice to DPIPWE, noted that '[t]he modes of transmission of

---

[79] *ABC News in Science*, Mystery cancer wiping out Tasmanian devils, 1 August 2003, http://www.abc.net.au/science/articles/2003/08/01/915506.htm last accessed 30 November 2008
[80] *ABC The World Today*, Wildlife specialists concerned about Tassie Devil disease, 14 October 2003, http://www.abc.net.au/worldtoday/content/2003/s966941.htm last accessed 30 November 2008
[81] Owen D & Pemberton D, 2005, *Tasmanian devil, a unique and threatened animal*, Allen & Unwin, Sydney, p 188
[82] Mooney N, 2004, *Minimising the unnatural spread of Tasmanian Devil facial tumour disease*, University of Tasmania, Hobart

the tumour are not certain, but are likely to include contact associated with damage to the skin around the head and neck, as occurs with fighting, scratching and biting'.[83] But the company queried the finding 'that lesions are rarely observed on other parts of the body that are also subject to trauma (such as the legs)'.[84] Hamish McCallum and Menna Jones were also confounded to discover that '[d]espite individual devils being capable of moving up to 50 kilometres in one night, the disease appears to have taken three years to travel the 30 kilometres of the Freycinet Peninsula in eastern Tasmania'.[85] Meanwhile, Steve Marvanek, a Commonwealth Scientific and Industrial Research Organisation (CSIRO) expert in applying geographical information systems (GIS) to resources and environmental problems, reported a further discrepancy. He noted that DFTD appears to 'have broken out spontaneously' in three separate locations 'rather than moved in from nearby', as might have been expected if the disease was contagious and spread via biting.[86] Further inconsistencies and anomalies have arisen in relation to the transmission of the disease and these will be explored in Chapter 4. The possibility that the spread of the disease was an artifact of reporting rather than a real event has not been resolved.[87]

---

[83] AusVet Animal Health Services Pty Ltd, 2005, *Tasmanian Devil Facial Tumour Disease Technical Workshop Final Report* to the Department of Primary Industries, Water and Environment, Hobart, Tasmania, p 9

[84] ibid.

[85] McCallum H & Jones M, 2006, To Lose Both Would Look Like Carelessness: Tasmanian Devil Facial Tumour Disease, *PLoS Biology*, Vol 4(10), pp 1671-1674, p 1671

[86] Marvanek S, 2007, Application of GIS to visualizing DFTD distribution, *Devil Facial Tumour Disease Senior Scientist's Scientific Forum Handbook,* Department of Primary Industries and Water, Hobart, Tasmania

[87] Hamede RK, McCallum H & Jones, M, 2012, Biting injuries and transmission of Tasmanian devil facial tumour disease, *Journal of Animal Ecology*, published online 3 September 2012 before publication in an issue.

1.6 Outlook for the Tasmanian devils

In the mid-1900s the survival of the devils seemed secure. Earlier Troughton had noted that the devil 'is not considered to be in as much danger of extermination as the …thylacine'.[88] This is reflected again in 1993 when Guiler predicted the future for the devils was good with its greatest protection being its lack of economic value. However by 2003 Jones and colleagues in *Predators with Pouches* noted that human-induced declines had been reported in all of Australasia's eight larger marsupial carnivores with the most significant being the extinction of the Thylacine.[89] The Tasmanian devil by this time was classified as Lower Risk – Least Concern. In the same year the Tasmanian government implemented its first strategy to address the devil cancer. The devils' demise has since accelerated and in 2008 it was listed under the *Environment Protection and Biodiversity Conservation Act 1999* as endangered and facing imminent extinction. Its future now rests with conservation efforts including introduction to one of Tasmania's offshore island.[90]

1.7 Conclusion

The Tasmanian natural environment is proving increasingly hazardous to native wildlife species. Australia's marsupials, including the Tasmanian devil, are real survivors demonstrating 'the considerable evolutionary fine-tuning that has allowed them to cope with the drastically altered climates and escalating environmental stress of the last five

---

[88] Troughton E, (ed.), 1967, *Furred Animals of Australia*, Angus and Robertson, Cremorne, Sydney, p 44
[89] Jones ME, Oakwood M, Belcher CA, Morris K, Murray AJ, Woolley PA, Firestone KB, Johnson B & Burnett S, 2003, 'Carnivore Concerns: Problems, Issues and Solutions for Conserving Australia's Marsupial Carnivores' in M Jones, D Dickman & M Archer, (eds), 2003, *Predators with Pouches, The Biology of Carnivorous Marsupials*, CSIRO Publishing, Collingwood, Victoria.
[90] Larkins D, 2012, Tasmanian devils shipping off to Maria Island, *ABC Local Radio*, Australian Broadcasting Commission, Hobart. Available at: http://www.abc.net.au/local/audio/2012/11/14/3632739.htm last accessed 25 November 2012

million years'.[91] However, more recently the major causes of species' declines worldwide are human-induced habitat loss through farming and forestry practices, excessive use of resources, the impact of introduced species and pollution.

The devils' habit of biting is hypothesised as being the means by which the cancer is transmitted from devil to devil. It can also be asked, is it possible that an evolutionary adaptation of ritual behaviour in defending a food source or competing for the chance to reproduce its genes has contributed to the devils' possible extinction? Or could human activities, such as habitat destruction and water contamination from forestry activities, offer a more plausible hypothesis for the cause of the devil cancer?

In order to get closer to the answer this investigation will analyse the scientific research that has been undertaken into the devil disease and question why certain research pathways have been pursued and others avoided, abandoned or neglected. The next chapter lays out a framework for a typology of undone science and describes the methodology underpinning the case study, the Tasmanian devil cancer.

---

[91] Morrison R & Morrison M, 1988, *The Voyage of the Great Southern Ark*, Sydney Lansdowne Press, p 292 cited in D Owen & D Pemberton, 2005, *Tasmanian devil, a unique and threatened animal*, Allen & Unwin, Sydney, p 38

Chapter 2 – The political sociology of science and undone research

2.1 Introduction

To analyse devil cancer research I have two different conceptual frameworks and hence divided the thesis into two parts. The first part, chapters 2 to 5, includes this chapter, which describes the main analytical framework, grounded in the political sociology of science and focusing on the concept of knowledge and its opposite, lack of knowledge, as ignorance and undone science. In the following three chapters, using this framework, I analyse the scientific research undertaken into the Tasmanian devil cancer, its anomalies and the undone science. In the second part, chapters 6 to 10, I explain why the precautionary principle, as a tool for decision makers to adopt in the face of scientific uncertainty surrounding the Tasmanian devil cancer and other wildlife cancers as a result of the undone research, might be a way to proceed. I also analyse impediments to action such as regulatory capture and conflict of interest in the regulation and control of use of hazardous chemicals, in particular atrazine.

To support my use of the concepts of political sociology of science and how undone research fits within these concepts, I begin by giving a brief account of the history of what is conventionally accepted as scientific research. Then I briefly define how credible scientific knowledge is established through a long process involving skepticism and critique. This outline traces the orthodox and conventional methods of the scientific process, but as the literature is vast and contested, for the purposes of this thesis, I have limited my review to the more accepted methods of scientific research. This research takes a sociological approach, thus I will outline the concept of the social

construction of scientific knowledge and propose how it can be expanded through a political sociology of science. This includes an outline of the approach of Stuart Blume in his book *Toward a Political Sociology of Science,*[1] the ideas of Imre Lakatos's progress in research programs[2] and David Hess's concept of undone science.[3]

This chapter also includes a section on methodology. In this section I detail how I endeavoured to collect the information necessary to undertake my analyses of the scientific research into the Tasmanian devil disease.

2.2 What is science?

Science, according to Imre Lakatos, evolved from the Latin word for knowledge *scientia* to become the most highly respected type of knowledge.[4] There exists an enormous range of debates on the philosophical and historical meaning of science and its practices. Although these debates are important for establishing the nature of truths about the natural world it is not my intention here to enter into them, but rather to give an overview. For this purpose I have relied on the writings of Lakatos, one of the major philosophers of science.

Within the debate about how we as humans cognitively know if scientific knowledge attains the truth about the natural world, positions range from believing observations of the world are made by a mind that is a '*tabula rasa'* to believing they are made by a mind that is already shaped to conceive the world from a particular viewpoint. A

[1] Blume SS, 1974, *Toward a Political Sociology of Science*, The Free Press, A Division of Macmillan Publishing Co, Inc. New York
[2] Lakatos I, 1978, *The methodology of scientific research programmes*, Cambridge University Press, Cambridge
[3] Hess DJ, 1997, *Science Studies, An Advanced Introduction*, New York University Press, New York and London
[4] Lakatos I, 1978, *The methodology of scientific research programmes*, Cambridge University Press, Cambridge, p 1

further debate is whether the human mind remains within this shaped cognitive or ideological framework or if it can actively re-shape its worldview. Although these contentious issues exist and may have implications for this research, they are not pursued in this thesis. I accept, however, that scientists approach their work, not from a mind that is devoid of knowledge but one that is socially shaped to perceive the world from a particular perspective, in other words, socially constructed. In adopting this approach, I further acknowledge that scientific research is conducted according to rigorous and long established procedures.

2.3 Orthodox methods and conventions of scientific research

Historically scientific knowledge evolved from the human ability to observe and make sense of the world. A commonly held view is that it is 'a formal activity that accumulates knowledge by directly confronting the natural world'.[5] Thus, through a rigid set of scientific practices and procedures, the truth about the natural world is revealed. Initially knowledge was judged by verification. If it could be proved then it was deemed knowledge; if it could not be proved, then it was false. However, by the time of the Enlightenment there was a realization of fallibility, that the human capacity for knowing nature was limited, and that there would be unknowns and uncertainty in scientific knowledge. According to Lakatos this revelation showed that humans were both fallible and ignorant.[6]

All scientific research is conducted according to orthodox methods - a set of procedures or conventions that allow for a certain amount of consistency. This enables scientists, if

---

[5] Sismondo S, 2004, *An Introduction to science and technology studies*, Blackwell Publishing, Malden USA, p 1

[6] Lakatos I, 1978, *The methodology of scientific research programmes*, Cambridge University Press, Cambridge

not to provide definitive and equivocal evidence about the truths of the natural world, to establish a scientific consensus. Scientific claims are designed to be subject to skepticism, experiment and the challenges of rival theories. According to Edward O Wilson '[w]ithout this vulnerability, they will not be accorded the status of scientific theories'.[7] Science, according to Wilson, 'is the organized, systematic enterprise that gathers knowledge about the world and condenses the knowledge into testable laws and principles'.[8] The principles that distinguish science from pseudoscience are replication, simplicity, prediction, accuracy and consistency.

Replication in science means repeating the same experiment, preferably by independent investigation, where the findings are interpreted and confirmed or disproved, this also constitutes scientific verification. Simplicity is the view that the fewer supporting theories to account for a phenomenon the better, as was first expressed in the 1320s by William of Occam 'What can be done with fewer assumptions is done in vain with more'.[9]

Prediction and accuracy can be understood together. The best theories are accurate in the predictions they make across many phenomena and those predictions are easiest to test by observation and experiment. An example of the ability to predict is clearly demonstrated by the astronomer Edmund Halley. Following the observation of a comet's trajectory and applying Sir Isaac Newton's laws of gravity and motion, Halley

---

[7] Edward O Wilson, 1998, *Consilience, the unity of knowledge*, Alfred A Knopf, New York, p 52
[8] ibid, p 53
[9] Williams R, nd. *Ockham's Razor*, Australian Broadcasting Corporation, Radio National. Available at: http://www.abc.net.au/radionational/programs/ockhamsrazor/podcasts/ last accessed 25 June 2013

predicted that it would return in 75 years. His prediction proved to be correct, when 75 years later the comet, subsequently named in his honour, returned.[10]

Consistency, on the other hand, was demonstrated by early observations of the natural world exposing the rhythms and repetitions that formed the universal laws of nature. According to Carnap, these universal laws of nature are fundamentally unchangeable.[11] However, complexities and variations in the natural world do produce exceptions to the laws. In circumstances where universal laws are not appropriate statistical laws or the laws of probability are used. These laws enable humans to make decisions based on known observations but where there is uncertainty in relation to all possible observations. For example, if all swans observed are black then it is probable that all swans are black, because it may be impossible to observe every swan. Hence, natural science operates with a degree of uncertainty. Future observations may nevertheless provide answers to the unknowns but there may be others that may never be known. Anomalies on the other hand, according to Lakatos, may need further explanation, but nature does not allow exceptions.[12] Anomalies raise a problem, according to Hess, when theories are adjusted to accommodate new data. Lakatos also supports this view, suggesting scientists do not discard a useful theory in the light of apparently contradictory evidence but attempt to harmonize the findings.[13]

---

[10] Lakatos I, 1978, *The methodology of scientific research programmes*, Cambridge University Press, Cambridge p 5
[11] Carnap R, 1995, *An Introduction to the Philosophy of Science*, Dover Publications Inc, New York
[12] Lakatos I, 1978, *The methodology of scientific research programmes*, Cambridge University Press, Cambridge, p 47
[13] Lakatos, I, 1980, *The methodology of scientific research programmes, Philosophical Papers Volume 1*, (Ed Worrall J and Currie G) Cambridge University Press, Cambridge

As well as the orthodox epistemology of science briefly described above, Robert K Merton's assumptions about the norms of doing science included openness, transparency, critical analysis, organized skepticism, objectivity and publication through peer-review.[14] Merton also emphasised the importance of originality and the significance of establishing the individual's priority in making a discovery. These orthodox methods and conventions of scientific research do not take place in a vacuum but are embedded in and informed by broader social, cultural and economic influences.

Science, according to JD Bernal, was once 'the occupation of curious gentlemen or of ingenious minds supported by wealthy patrons' but today it has become 'an industry supported by large industrial monopolies and by the State'.[15] This situation has contributed to science attaining a paradoxical condition. It is at once acclaimed as the preeminent source of knowledge, invested as Hess states, with the authority to proclaim 'what is and can be the case',[16] and at the same time challenged by those threatened by its findings; this is particularly evident in environmental science.

2.4 The social construction of science

Sociologists through analyses of different dimensions of science and its progress have found that scientific research, far from being conducted as an autonomous pursuit by individuals seeking objective truth, is undertaken by scientists who have preconceptions, commitments, agendas and biases.

---

[14] Merton RK, 1968 (Enlarged Edition), *Social Theory and Social Structure*, Collier Macmillan Publishers, London

[15] Bernal JD, 1939, *The Social Function of Science*, Routledge & Kegan Paul, London, p xiii cited in Stuart S Blume, 1974, *Toward a Political Sociology of Science*, The Free Press, A Division of Macmillan Publishing Co. Inc. New York

[16] Hess DJ, 1997, *Science Studies, An Advanced Introduction*, New York University Press, New York and London, p 21

Scientists generally research within a community where they bring social, cultural and economic values to their observations. Scientific research is also no longer self-funded but relies heavily on finances from both government and industry. These social values and pressures influence scientists' choices in the type of science they undertake, where they study and what (either academic or industry) employment they seek. Consequently scientific knowledge production is driven by technological innovation and by economic imperatives, substantially determining the direction of scientific progress.

In order to understand how scientific research is selected for study an analysis of the scientific community, including its institutional commitment and external influences, is necessary. In this study it is not the behavior of individual scientists within the laboratory but their role as participants in a research program, controlled by a government entity, the Tasmanian government Department of Primary Industries, Parks, Environment and Water (DPIPWE), that is investigated. Public scientific controversy is usually researched through the interrogation of both sides of the controversy. There is usually a challenge to, or disagreement over, knowledge production. In the case of Tasmanian devil cancer there is, however, no challenge to the authority or expertise of those proposing the allograft hypothesis, consequently there is no public controversy.

I have subsequently broadened my research position to situate the devil cancer within the scientific controversy surrounding the causes of cancer documented by Robert Proctor in his book *Cancer Wars: How Politics Shapes What We Know and Don't Know About Cancer.*[17] Proctor investigates the influences that shape the research pathways or as he describes it, 'why scientific tools are sharp for certain kinds of

[17] Proctor RN, 1995*, Cancer Wars: How Politics Shapes What We Know and Don't Know About Cancer,* BasicBooks, New York

problems but are dull for others'.[18] This view supports Hess's concept of undone science and his analogy that some lines of inquiry flourish, whilst others 'wither on the vine'.[19]

Within a research community, studies are conducted within boundaries of scientific thinking and these boundaries are generally maintained by a commitment to a particular theory. Lakatos proposes two pathways for new research programs to follow.[20] The first is an initial naïve model or a first version, based on a discovery that is often seen as an anomaly to an already accepted theory. This model is, however, eventually replaced as the program develops. This new research program may finally gain autonomy and establish a 'hard core' or dominant theory, surrounded by auxiliary hypotheses. In the case of the Tasmanian devil disease I have positioned the cancer within the hard core of the orthodox or mainstream cancer theory. Consequently, because it is proposed to be a transmissible cancer, the research program can be understood as an auxiliary hypothesis to the dominant theory.

The second pathway of scientific research program development is through a consistent increase in content, developed from 'a series of conjectures and refutations'.[21] This consistent increase in the research program results in a progressive shift in both the theoretical and empirical knowledge. Lakatos stresses consistency must remain the most important guiding principle and any deviations must be seen as problems.[22] This

---

[18] ibid, p 9
[19] Hess DJ, 2007, *Alternative Pathways in Science and Industry, Activism, Innovation, and the Environment in an Era of Globalization*, The MIT Press, Cambridge, Massachusetts
[20] Lakatos I, 1978, *The methodology of scientific research programmes*, Cambridge University Press, Cambridge
[21] ibid, p 4
[22] ibid, p 57

methodology, according to Lakatos, is important in order to avoid commitment to absurd beliefs. He further states that '[b]lind commitment to a theory is not an intellectual virtue; it is an intellectual crime'.[23] The implications of these views for the development of the Tasmanian devil research program are fully explored in the following chapter.

Meanwhile, Stuart Blume makes the observation that scientists have social and cultural values, as well as personal goals and allegiances that intrude into the scientific process.[24] The scientific research community over time develops a culture that can be traced back to its original discovery, which is informed by the historical conditions in which it was embedded. The community will also over time develop a resilience and logic of its own, such that it responds to outside interests from the perspective of its own values and logic.[25] For Blume, this community might also exhibit traits such as 'secrecy, selective citation and resistance to new discoveries'.[26]

Notwithstanding the shaping of the research by social and cultural views, more important for this study is the funding of the research by the elites in society, or vested interests. Hess proposes that this particularly informs this new field of analysis, the political sociology of scientific research. It is the ability to fund the choice of research agenda and the selection of what is to be studied that ultimately leads to certain fields of research being neglected.

---

[23] ibid, p 1
[24] Blume SS, 1974, *Toward a Political Sociology of Science*, The Free Press, A Division of Macmillan Publishing Co. Inc. New York, p 78
[25] Hess DJ, 1997, *Science Studies, An Advanced Introduction*, New York University Press, New York and London, p 75
[26] Blume SS, 1974, *Toward a Political Sociology of Science*, The Free Press, A Division of Macmillan Publishing Co. Inc. New York p 78

2.5 The new political sociology of science (NPSS)

In modern politics science is often used to inform those who govern society and set public policy. By building on the ideas of social construction of science I now shift the focus more specifically to the study of a politicalisation of science, which began in the early 1970s and was refined more recently by the new political sociology of science (NPSS) laid out by Frickel and Moore in 2006.[27] It identifies a conjunction between science and politics. Science theorists in the mid-1970s realized that the relationship between governments and scientists had created close ties, which had politicized the scientific endeavour.

This is best demonstrated by scientists moving outside the laboratory and beginning to actively participate in the political process, through their involvement in public activities, such as voicing objections to the development of nuclear weapons. There was also a shift in residence, from being mainly academic advisors within universities into positions within government, particularly in the US. This shift, according to Blume, meant scientists working for governments, as funders of the research, tend to comply with the dominant ideology.[28] Scientists might also be selected because they share the same economic, cultural and social values as the incumbent government, potentially resulting in scientists choosing research topics they know will be funded, thus inadvertently avoiding areas that might prove detrimental to governments. Also influencing scientists' ability to deviate from the research agenda are the organizational rules within governments.[29] Another aspect of the scientific endeavour that could

---

[27] Frickel S & Moore K, (Eds), 2006, *The New Political Sociology of Science: Institutions, Networks, and Power,* The University of Wisconsin Press, Madison, Wisconsin
[28] Blume SS, 1974, *Toward a Political Sociology of Science*, The Free Press, A Division of Macmillan Publishing Co. Inc. New York p 78
[29] ibid, p 88

influence decisions is the reward system, which could also play into the politicizing of scientists and their research. All of these influences have the potential to create what Hess calls 'undone science'.

Rewards allocated under conditions of autonomy deliver prestige to scientists based on their ability to solve problems in their field, but when governments bestow scientists, not only with financial rewards, but also with prestige, via appointments to advisory bodies, membership in commissions of inquiry and prizes, it can constitute political inducement. As Blume claims, recognition and rewards are the commodities of the scientific exchange system.[30] The process may mean that the scientific community as a whole complies with the objectives of their funders, whilst individuals may operate on separate research projects within a project, unaware of this compliance. It is also probable that one or two scientists liaise with the government agent or body appointed to oversee the research. It is these scientists who then steers the research on behalf of the government. These individual scientists who comply with the dominant overview and produce the results or findings conducive to the government are then rewarded appropriately. As Blume states:

> Employed scientists may be offered inducement to accept status and money in return for their work. By this means, and its control of research facilities and its influence over choice of research problems, the organization seeks to "usurp" control of the process of science.[31]

Governments have adopted industrial research management practices in seeking a substantial measure of influence over the topics upon which employed scientists work.[32] As will be shown in the following chapter, scientists employed by the Tasmanian

---

[30] ibid, p 28
[31] ibid, p 87
[32] ibid, p 92

government in the Tasmanian devil research have received rewards that represent substantial benefits: on completion of research, individuals are moved into higher academic appointments; younger post-doctoral students are granted appointments at prestigious academic institutions; others are retained and rewarded through prizes and public acknowledgements.

Consequently, it is proposed that through selecting what research is to be funded, supported by rewards and publications, that elites shape the research pathway. The result is that certain fields of research are neglected. These fields of research are what Hess has termed 'undone science'. It is recognized that for various reasons, not all areas of research are explored. To further expand the concept of undone science, or as I have referred to it 'undone research', I have developed a typology, by classifying the different types into practical or political reasons as to why studies might be left undone.

2.6 Hess's alternative pathways in science

Hess developed his framework from observations that certain research was selected for study, whilst other research was left undone.[33] Using case studies of conventional research versus alternative research he found a disparity in the numbers of research studies. These case studies included an alternative hypothesis that bacteria might be the cause of cancer, comparisons of research programs on orthodox and alternative medicine, and of conventional and organic agriculture. The bacteria causation hypothesis of cancer was not well received by the biomedical establishment whose research priorities include genetic inheritance and lifestyle factors as the major causes of cancer. Likewise, alternative medicine and organic agricultural practices have been

[33] Hess DJ, 2007, *Alternative Pathways in Science and Industry, Activism, Innovation, and the Environment in an Era of Globalization*, The MIT Press, Cambridge, Massachusetts

little studied. Hess found that the majority of funding for research undertaken in the major institutions focused on orthodox research with very little directed towards alternative theories. These areas for Hess constitute 'undone science' or neglected research areas and the motivation for the gap is political.

In his book *Can bacteria cause cancer?* Hess analyses how the dominant biomedical model of medicine shapes the research agenda.[34] Like Lakatos, he found that a field of research increases its autonomy as it becomes more defined, routinized and guided by the generally accepted research program. According to Hess, the cancer research community confirms this pattern, adding that there is a formative period when the basic direction of the research program is set in place (such as the refusal to see cancer as a metabolic, nutritional, or infectious disease and one that could be treated by vaccines, sera, and nutritional therapies).[35] Furthermore, as a field of research becomes increasingly technical and specialized, the choices that were so evident at the beginning become largely forgotten. In the field of cancer research, Hess documents the cancer controversy of which James Ewing and William B. Coley were initially a part. It subsequently lost ground as the noninfectious nature of cancer came to dominate.[36] Support for the dominant theory did not emerge entirely from internal, intellectual processes such as the consideration of evidence but rather, the consensus was compliant with the dominant political and economic forces that provided incentives for therapies

---

[34] Hess DJ, 1997, *Can Bacteria Cause Cancer? Alternative Medicine Confronts Big Science,* New York University Press, New York and London
[35] ibid, p 73-74
[36] Hess DJ, 1997, *Can Bacteria Cause Cancer? Alternative Medicine Confronts Big Science,* New York University Press, New York and London

oriented toward X-ray machines, radium, pharmaceuticals, and other industrial products.[37]

The scientific research agenda has been largely directed by the pursuit of profit under the guise of progress, but this progress and the benefits it bestows have now been the subject of more critical analysis. Science has long been associated with industry in both the private and public sectors. In the private sector, funding of research for the development of technologies has provided modern society with benefits in the workplace, in the home, for leisure and in the pursuit of knowledge. Science has also benefited from public funding in more altruistic pursuits such as, space exploration and the development of computer technology. Both have provided humanity with huge benefits but are there unintended consequences and costs?

Wherever large corporations substantially influence government policies, legislation and laws, this promotes priorities in culture, the economy and in scientific research that benefit corporations at the expense of the public. It is these influences that lead to research being shaped by vested interests, through the choice of studies to be undertaken, leaving other research undone.

Funding priorities are not the only areas where undone research is evident; it is also evident in the knowledge-making process. The ability to create a body of scientific knowledge increasingly relies on the latest technology and methods. The cost involved in accessing sophisticated equipment is often exorbitant, hence, the dominant network, with the most funding, tends to have the most access. In the case of DFTD, the

---

[37] ibid, pp 73-74

Government has its own laboratory for carrying out tests but it also has access to expensive and highly sophisticated genetic testing laboratories, such as Cold Spring Harbor Laboratory in the United States and the Wellcome Trust Sanger Institute in the United Kingdom. This can have the effect of silencing the alternative views by giving the impression that the dominant research field, with access to world class technology, is pursuing better science.[38]

In the knowledge making process Hess moves beyond the debate that scientific knowledge is socially constructed, to be more concerned with which research is selected as deserving attention and which is not considered worth pursuing.[39] He terms this problem the "selection" of knowledge, in contrast to the construction of knowledge.[40] His use of the word "selection" is understood as "choosing" from an already limited range of choices imposed on the less powerful. For Hess the question is no longer how knowledge is socially shaped, but is instead a structural question of what research is selected.[41]

Various social, economic and political factors impact on scientific research programs in this knowledge making process. They constitute both internal and external pressures and strongly influence the type of knowledge that is built upon a program. Whilst the studies that are selected for research contribute to a body of knowledge, those abandoned or left undone create a body of non-knowledge or ignorance. The next section describes the rationale for categorizing undone research or what Hess terms

---

[38] Hess, DJ, 2007, *Alternative Pathways in Science and Industry, Activism, Innovation, and the Environment in an Era of Globalization*, The MIT Press, Cambridge, Massachusetts, p 24
[39] ibid, p 28
[40] ibid, p 29
[41] ibid, p 29

'undone science'. It begins with an outline of the internal and external pressures and continues, under the more general framework of ignorance, to define undone research. I conclude with a typology of the different reasons, either practical or political, for undone research.

2.7 Internal and external pressure

Factors that influence scientific research vary, therefore it is necessary to analyse them in detail. To begin, there are two broader elements: external and internal pressure. External pressure, according to Hess, is the result of political influence exerted by elites on scientific research, and is a principal reason for undone science.[42] In these circumstances elites have the power and financial capacity to direct scientific research along certain pathways and either avoid or neglect others, resulting in relationships with the potential to obscure the boundaries between science and politics.

Internal pressures, on the other hand, include factors such as commitment to a particular theory or paradigm within a research community, which can equally influence the direction scientific research takes.

A close investigation of the broad external pressures influencing DFTD research indicates that elites, as described by Hess, might indeed play a role in directing the research. In Tasmania the Tasmanian Government through the DPIPWE, the University of Tasmania (UTAS) and the forestry industry comprise the elites. These three entities have close ties and engage in both the development of plantation forestry in Tasmania and the scientific research into the Tasmanian devil cancer. The DPIPWE managers are

[42] ibid, p 22

charged with both the monitoring of pesticide contamination of drinking water sourced from catchments heavily forested with plantation and with the protection of endangered species including the Tasmanian devil. When one government department is responsible for both the monitoring of chemicals in water and the possible role of those chemicals in the devil cancer, it would also appear to constitute a conflict of interest. This issue is covered in depth in chapter 9. Further, through the *Save the Tasmanian Devil* program, DPIPWE, in collaboration with UTAS, also co-ordinates the funding of the scientific research into the Tasmanian devil disease. This further close association has led to the control by the DPIPWE of the devil research and the direction of research along a selected pathway. Further analysis is provided in the following chapter.

Internal pressure can also play a role in directing research pathways, particularly when scientific communities are committed to a theory. According to sociologist Harriet Zuckerman it can be so strong it can lead scientists to "preempt" some possible problem areas as not worth researching, potentially leading to pockets of undone science.[43] In Tasmania, although the toxicology studies were abandoned, it was not because they were deemed unworthy of research. An investigation of a possible competing theory of DFTD causation, that agrichemicals are somehow involved, far from being deemed unworthy of study and therefore not researched, was identified as warranting study on three separate occasions: firstly, by the DPIPWE in its *Progress Report*[44] on the Devil disease, secondly by Pearse and Swift[45] in their article in *Nature* and thirdly in the

---

[43] Zuckerman H, 1978, Theory Choice and Problem Choice in Science, *Sociological Inquiry* Vol 48(3-4), pp 65-95, p 75

[44] Tasmanian Government Department of Primary Industries, Water and the Environment. 2005. *Research into the Tasmanian Devil Facial Tumour Disease (DFTD) Progress Report.* Department of Primary Industry, Water and Environment, Hobart, Tasmania

[45] Pearse AM & Swift K, 2006, Transmission of devil facial-tumour disease, *Nature* 439(2), p 549

Scammell Report[46] based on information collected by Dr Marcus Scammell (marine ecologist), Dr Alison Bleaney (Area Medical Officer) and marine farmers. The toxicology studies, as undone science, are the focus of chapter 5. Further analysis of internal effects relates to what constitutes knowledge in a research program. In the DFTD research program, knowledge about the devil cancer is accumulated from studies informed by the hypothesis that the tumour is contagious.

From these broader perspectives I now give details of how I have determined whether the research studies are in fact undone, and why. The methodology for my investigation is discussed in the relevant sections below. In order to establish the validity of the concept of undone science I begin by reviewing the literature on ignorance. A gap in scientific knowledge constitutes a deficit or lack of information, which may or may not alter the course of the research. However, if the lack of information has the potential to provide protection to vested interests, then a closer examination of the reason for the gap is warranted. Hence, I proceed to outline a typology of practical and political reasons for undone science, which further inform my analysis of the Tasmanian devil case study.

2.8 Ignorance - a deficit of knowledge

Ignorance is particularly relevant when scientific research is conducted into new and emerging diseases, such as AIDS or SARS, because then it is operating within narrow boundaries of knowledge. In science, ignorance is the umbrella term for the general field that includes nescience and non-knowledge. There are only two main branches of

---

[46] Scammell M, (collator), 2004, *Environmental Problems Georges Bay, Tasmania.* Available at http://www.tfic.com.au/domino/tfic/tficweb.nsf/vwTitle/07.04%20Scammell%20Report last accessed 13 May 2007

ignorance: the deep ignorance of nescience, in which we are not even aware of what we do not know, and the knowable forms of ignorance, represented by the concept of "non-knowledge". Nescience and non-knowledge are more fully described in the following sections. It is also the production of knowledge, which brings about a paradox – the more we know the more we realize how much we don't know. Wolfgang Krohn describes it as 'every state of knowledge opens up even more notions of what is not known'.[47] This dilemma of knowledge has existed since Socrates who insisted that his 'wisdom' lay in knowing that he did not know.

For Matthias Gross ignorance is '[k]nowledge about the limits of knowledge in a certain area…'.[48] Ignorance therefore necessarily constitutes a known gap in existing knowledge. From a different perspective, Robert Merton saw that unanticipated consequences of ignorance can have desirable effects, which he termed 'serendipity', an anomalous finding that gives rise to a new theory.[49] Merton made ignorance a central theme in his deliberations and defined two types - unrecognised and specified ignorance. In a comparison between knowledge and ignorance he stated 'yesterday's uncommon knowledge becomes today's common knowledge and yesterday's unrecognized ignorance becomes today's specified ignorance'.[50] Merton further recognised that new knowledge brought an awareness of more specified as well as unspecified ignorance. An example of current scientific ignorance is in the area of environmental pathways and modes of action of endocrine disrupters, synthetic chemicals that mimic natural

---

[47] Krohn W, 2001, 'Knowledge Societies', pp 8139-43 in NJ Smelser & P Baltes, eds, *International Encyclopedia of the Social and Behavioral Sciences*. Blackwell, Oxford, p 8141

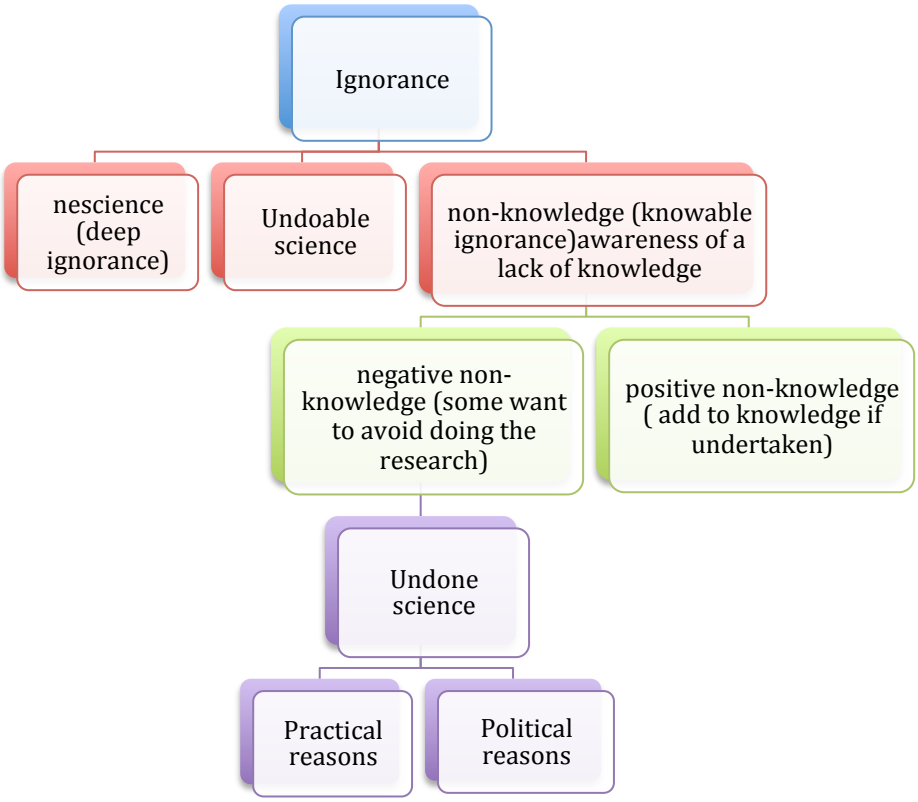
[48] Gross M, 2007, The Unknown in Process: dynamic Connections of Ignorance, Non-Knowledge and Related Concepts, *Current Sociology* Vol 55(5), pp 742-759, p 751

[49] Merton R, 1968 (Enlarged Edition), *Social Theory and Social Structure*, Collier Macmillan Publishers, London

[50] Merton R, 1987, Three Fragments from a Sociologist's Notebooks: Establishing the Phenomenon, Specified Ignorance, and Strategic Research Materials, *Annual Review of Sociology* Vol 13, pp 1-28, p 10

hormones in living organisms.[51] The various forms of ignorance are shown in Figure 2:1 below. The figure expands non-knowledge to include undone science. These categories are further described in the following sections.

Figure 2:1 Categories of Ignorance



2.8.1 Nescience

Gross categorises nescience as 'lack of any knowledge: prerequisite for a total surprise beyond any type of anticipation…'.[52] It is the complete lack of knowable ignorance of the existence of potential knowledge. It is what Ann Kerwin has termed 'unknown

[51] Myers JP, Krimsky S & Zoeller RT, 2001, Endocrine Disruptors – A Controversy in Science and Policy: Session III Summary and Research Needs, *NeuroToxiciology*, Vol 22, pp 557-558
[52] Gross M, 2007, The Unknown in Process: dynamic Connections of Ignorance, Non-Knowledge and Related Concepts, *Current Sociology* Vol 55(5), pp 742-759, p 751

unknowns'.[53] It is similar to Brian Wynne's definition of indeterminacy when applied to environmental policy.[54] Wynne views indeterminacy as 'the open-endedness in the processes of environmental damage due to human interventions'.[55] Peter Wehling describes nescience as a complete unawareness of non-knowledge, which can only be made visible in sociological analysis, when, like knowledge, its utterances, constructions or negotiations can be registered.[56] According to Gross it 'belongs to a fundamentally different epistemic class from non-knowledge or ignorance' since it can only be detected in retrospect.[57] He elaborates further '[n]o one can refer to their own current nescience because it is not part of their consciousness… At most, people can refer to someone else's or their own earlier nescience'.[58] The unanticipated and surprisingly detrimental outcome of the use of DDT is an example of nescience. It was only in retrospect that scientists identified a lack of knowledge of the unforeseen harmful effects of the widespread use of the chemical.

2.8.2 Non-knowledge (knowable ignorance)

Non-knowledge, according to Gross who groups ignorance and non-knowledge as connected, is defined as knowledge about what is not known.[59] Gross further categorises it as 'knowledge about what is not known but taking it into account for

---

[53] Kerwin A, 1993, None too Solid: Medical Ignorance, *Knowledge: Creation, Diffusion, Utilization* Vol 15(2), pp 166-185
[54] Wynne B, 1992, Uncertainty and Environmental Learning: Reconceiving Science and Policy in the Preventive Paradigm, *Global Environmental Change* Vol 2(2), pp 111-127
[55] Wynne B, 1992, Uncertainty and Environmental Learning: Reconceiving Science and Policy in the Preventive Paradigm, *Global Environmental Change* Vol 2(2), pp 111-127, p 119
[56] Wehling P, 2001, Jenseits des Wissens? Wissenschaftliches Nichtwissen aus soziologischer Perspektive, *Zeitschrift fur Soziologie*, Vol 30(6), pp 465-484 cited in M Gross, 2007, The Unknown in Process: dynamic Connections of Ignorance, Non-Knowledge and Related Concepts, *Current Sociology* Vol 55(5), pp 742-759
[57] Gross M, 2007, The Unknown in Process: dynamic Connections of Ignorance, Non-Knowledge and Related Concepts, *Current Sociology* Vol 55(5), pp 742-759, p 746
[58] ibid.
[59] Gross M, 2007, The Unknown in Process: dynamic Connections of Ignorance, Non-Knowledge and Related Concepts, *Current Sociology* Vol 55(5), pp 742-759

future planning'.[60] In a general crisis of knowledge there has been an increased acceptance that ignorance and uncertainty in science exist, subsequently there is a necessity to know about what is unknown. As an example of non-knowledge Gross describes the state of knowledge in relation to the flooding of an abandoned brown coal strip mine in Germany. The engineers decided to flood the mine aware of their lack of knowledge as to the rate of ground water and runoff it would take to fill the mine. They decided to go ahead with the flooding with totally unexpected results.[61]

2.8.3 Undoable science

Science can be 'undoable' due to constraints from existing methods or technology. However, according to Frickel et al science that appears to be 'undoable' can in fact be thwarted by insufficient resources and technical ability.[62] This is particularly evident when scientists are faced with chemicals that act as endocrine disrupters. These chemicals are dispersed from non-point sources throughout the environment. They are broken down into metabolites that add to the parent chemicals and mix with other chemicals used in the environment. These chemicals then often work in synergy to enter organisms in ways often unknown and to finally interact with hormonal and other systems at the molecular level. Endocrine disrupting chemicals challenge the boundaries of scientific knowledge and it is often only the harm they cause that is truly evident.

---

[60] ibid, p 749
[61] Gross M, 2007, The Unknown in Process: dynamic Connections of Ignorance, Non-Knowledge and Related Concepts, *Current Sociology* Vol 55(5), pp 742-759, pp 751-752
[62] Frickel S, Gibbon S, Howard J, Kempner J, Ottinger G & Hess D, 2010, Undone Science: Charting Social Movement and Civil Society Challenges to Research Agenda Setting, *Science Technology, & Human Values* Vol 35(4), pp 444-473

2.8.4 Undone Science as negative or positive non-knowledge

In all scientific endeavours there will exist scientific questions and problems, which are, according to Kuhn, not followed because they are simply not seen.[63] It is also beyond the scope of most research projects to pursue all avenues of enquiry. Consequently, a quantity of potential scientific research is left undone. This undone science is classified as non-knowledge, known ignorance. It can also be further categorised into either, negative or positive non-knowledge when viewed from different perspectives. Negative non-knowledge is that which is stifled or avoided when viewed from the perspective of those who would think or feel intuitively that the findings of studies might produce results damaging to their interests. On the other hand, those interested in addressing environmental problems would perceive the undone science as positive non-knowledge, because these findings could add empirical data to support their contention that industry or human activities are responsible for a perceived harm.

2.9 Reasons for undone science

Hess asserts that a special sort of undone science frequently occurs when research pathways are selected and funded by 'elites' in society, not for scientific reasons but for political expediency. Thus research agendas can be politicized, which requires a new framework of political sociology of science to analyse how knowledge is shaped, not only by the scientific communities, but also by industry and government influence.[64]

---

[63] Kuhn TS, 1970, *The Structure of Scientific Revolutions*, Chicago University Press, Chicago, p 24
[64] Hess DJ, 2009, Potentials and Limitations of Civil Society Research: Getting Undone Science Done *Sociological Inquiry* 79(3), pp 306-327, p 309

In order to distinguish the political aspects of knowledge production I divide the types of undone science according to whether practical or political reasons exist for not undertaking research: See Table 2:1 below, the terms of which will be explained below.

Table 2:1 Reasons for undone science

Practical Reasons	Political Reasons
Non-knowledge – knowable forms of ignorance	Knowledge considered 'not worth exploring'
Nescience – deep ignorance or unawareness of limits of knowledge	Uncertainty in science and in interpretation of existing research
'undoable science' – limited resources or practical constraints	'forbidden knowledge' – not funded on ethical grounds – stem cells, cloning
	Scientist targeted research abandoned due to ethics - weapons, nuclear
	'negative non-knowledge' or 'harmful knowledge' to mainstream – problematic, irrelevant or dangerous, incomplete, non-selected
	Self-imposed censorship: the 'chilling effect'
	Suppressed knowledge – suppression of intellectual dissent
	Formal and informal manifestations of power – control or capture of research

2.9.1 Practical reasons for undone science

Practical reasons for gaps in research, which form undone science according to Gross in his cateorization of knowledge, include non-knowledge, ignorance and nescience.[65] Non-knowledge, ignorance and nescience describe gaps in the research or as expressed in Frickel et al 'a deficit of research'.[66] These gaps in scientific knowledge or lack of

[65] Gross M, 2007, The Unknown in Process: dynamic Connections of Ignorance, Non-Knowledge and Related Concepts, *Current Sociology* Vol 55(5), pp 742-759
[66] Frickel S, Gibbon S, Howard J, Kempner J, Ottinger G & Hess D, 2010, Undone Science: Charting Social Movement and Civil Society Challenges to Research Agenda Setting, *Science Technology, & Human Values* Vol 35(4), pp 444-473

research are primarily due to constraints in either technical knowledge or equipment. A deficit of theoretical framework would also inhibit progress in scientific research resulting in knowledge gaps. Hence the science is not necessarily avoided for political reasons or because it is deemed not worth researching but because there are practical constraints on the research. Nescience as an unknown unknown falls easily into a practical reason for undone science. Non-knowledge as a practical reason for undone science relies on an awareness that the knowledge is not known but there is no immediate pressure or desire to carry out the research. Negative non-knowledge, as opposed to positive non-knowledge, is more likely to occur for as political reasons. Undoable science, when there are constraints arising from existing methods or technology, fits into the category of practical restraints on scientific research. There are practical reasons for undoable science, but the reasons are political if science is labeled undoable as an excuse, for example if used by regulators or toxicologists to extend the registration of endocrine disrupting chemicals.

2.9.2 Political reasons for undone science

The following sections describe the types of research that fall into the categories of political reasons for undone science.

2.9.2.1 Negative non-knowledge or forbidden knowledge

Political reasons for undone science, include what Gross types as 'negative non-knowledge'. Undone science as a form of ignorance or non-knowledge can be perceived as dangerous knowledge by those who fund research, similar to Hess's

science left undone by elites.[67] Undone science from the perspective of vested interests or those who do not want the research done is viewed as negative non-knowledge and consequently abandoned. In other words, the research is left undone for political reasons. In the case of the devil disease, toxicology results that may have identified dangerous levels of chemical residues in devil tissues constitute 'negative non-knowledge' as is described in Chapter 5.

However, there are circumstances where scientific research can be classified as 'negative non-knowledge' for ethical reasons. It becomes 'forbidden knowledge' and is not funded on ethical grounds. Science left undone or abandoned because it is considered unethical has included the testing of new designs for nuclear weapons and the cloning of human embryos. The science is considered either by some scientists or the public as too dangerous to pursue and hence pressure is put on governments and industry to leave it undone. These are political reasons for undone science. In Tasmania the scientific research into the devil disease has not been abandoned or left undone due to ethical concerns.

2.9.2.2 Uncertainty in science

When science is undoable due to either limitations in technology or non-knowledge, as is the case with the mode of action for endocrine disrupting chemicals, it can lead to uncertainty in science for practical reasons. Val Gunter and Steve Kroll-Smith point out that knowledge limits can also ensue from uncertainty in the interpretation of the results

[67] Hess DJ, 2009, Potentials and Limitations of Civil Society Research: Getting Undone Science Done, *Sociological Inquiry* Vol 79(3), pp 306-327

of research that does exist.[68] This uncertainty can genuinely stem from disagreements amongst researchers 'because both the production and interpretation of "facts" rest on models and background assumptions that are open to dispute'.[69] Uncertainty in science is often found in environmental problems where the complexities are extreme. This uncertainty can also provide reasons for delays in decision-making by policy makers and regulators resulting in benefits to vested interests. When science is conducted in a limited and secretive manner then uncertainty can be manufactured and used to the advantage of vested interests.[70] The uncertainty created by the undone research in the Tasmanian devil cancer and three other wildlife cancers is the prompt for me to suggest it would be prudent to invoke the precautionary principle. The need for the precautionary principle is discussed in full in Chapter 7.

Meanwhile, openness and transparency in research and publication through peer review allow scientific uncertainty over research results and different interpretations of research to be openly debated, negotiated, mediated and resolved.

2.9.2.3 Censorship and the 'chilling effect'

Scientific research that is compromised by a lack of openness and transparency can produce a further two types of undone science due to censorship: first, suppressed knowledge, when the science is done but not made public and second, censorship either by powerful elites or by self-censorship. Suppression according to Brian Martin is 'restraint or inhibition without physical force' such as blocking of publications which is

---

[68] Gunter V & Kroll-Smith S, 2007, *Volatile Places: A Sociology of Communities and Environmental Controversies*, Pine Forge Press, Thousand Oaks, California
[69] ibid, p 113
[70] Michaels D, 2006, Manufactured uncertainty: Protecting public health in the age of contested science and product defense, *Annals of the New York Academy of Sciences* Vol 1076, pp 149-162

an exercise in power.[71] Martin found that scientists avoid doing research if they expect an attack if they do so. Martin terms this self-intimidation. Joanna Kempner agrees with Martin that intellectual suppression has been the focus of most censorship along with distortion or manipulation of knowledge in the intimidation and silencing of researchers.[72] Kempner also agrees with Martin's notion of self-intimidation, that scientists frequently practice self-censorship, which she called the "chilling effect". In her study she found that scientists themselves employed a variety of methods in order to self-censor. These included:

- disguise the most controversial aspects of their research
- remove potential "red flag" words from titles or abstracts
- delete sensitive keywords
- complete silence i.e. not publish
- minor modifications
- omissions
- the reframing of studies in ways thought less politically sensitive
- dropped studies or non-renewal of studies thought to be politically non-viable.
- changing careers

Hess also notes suppression can occur through employment, where dismissal is threatened, or actions such as funding cuts, media campaigns and litigation are implemented to discredit and exhaust challengers. Hess further points out that the worst suppression is reserved for high-status challengers, the results of which not only have a 'chilling effect' on the targeted scientists but also on other 'would-be sympathizers and challengers'.[73]

---

[71] Martin B, 1999, Suppression of Dissent in Science, *Research in Social Problems and Public Policy,* Vol 7, pp 105-135
[72] Kempner J, 2008, The Chilling Effect, *PloS Medicine* Vol 5(11), pp 1517-1578
[73] Hess DJ, 2009, Potentials and Limitations of Civil Society Research: Getting Undone Science Done, *Sociological Inquiry,* Vol 79(3), pp 306-327

In her study Kempner did not find a causal relationship between political controversy and self-censorship but she did find that the political environment might serve as a powerful force in shaping scientific research practices. Her research also concluded that political controversy might also encourage scientists to avoid some areas of scientific inquiry, but no studies have formally investigated this possibility. Both Hess and Kempner call for an investigation into why certain science is left undone and what role political influence or controversy might play.

2.9.3 Summary of practical versus political reasons for undone science

A typology of undone science enables gaps in scientific knowledge to be attributed to either practical or political reasons. As described above, there are often practical reasons that inhibit the development of knowledge: the existence of ignorance, nescience and non-knowledge about a subject area, and undoable science sometimes, due to a lack of technical capabilities and/or a lack of funding. Practical reasons exist therefore, because the research genuinely cannot be carried out. There are no obstacles to further studies based on political decisions. The studies have not been avoided, abandoned or ignored by those who have the power to make decisions, as Hess describes the 'elites'.

By comparison, political reasons for undone science, described by Hess as 'absences of knowledge', involve the shaping of the research through the selection of particular pathways by those who fund the research.[74] Political reasons for undone science include uncertainty in science, negative non-knowledge, research abandoned for ethical reasons

---

[74] Hess DJ, 2009, Potentials and Limitations of Civil Society Research: Getting Undone Science Done, *Sociological Inquiry* Vol 79(3), pp 306-327, p 307

and censorship or suppression of knowledge that has been produced. Uncertainty in science is often the catalyst for an increase in studies but when it is used by decision makers to delay rulings or is manufactured to similarly delay actions, it is deemed political. Negative non-knowledge, the possible production of scientific knowledge considered dangerous to vested interests, is the most frequently avoided. Political pressure brought to bear on government or industry for ethical reasons is limited; pressure through censorship, either from elites or 'self' and suppression of knowledge is more widespread.

The results of my analysis of the Tasmanian devil cancer research program using the concept of undone research form the content of chapters 3 to 5. The research methods I used to gather my information are described in the next section.

2.10 Methodology

I have undertaken this investigation as a social scientist in the field of STS, not as a member of the Tasmanian devil scientific community. I have applied the concept of undone science to critically analyze the scientific research into the novel hypothesis that the devil cancer is contagious. This cancer epidemic is particularly significant because it threatens the survival of the Tasmanian devil. An alternative to the now dominant hypothesis was proposed in 2004 with the release of the *Scammell Report*.[75]

It noted a correlation in time and space between the increase in plantation forests and their reliance on pesticides, abnormalities in commercial oysters and the devil disease in

---

[75] *Environmental Problems Georges Bay, Tasmania: Collated by Dr Marcus Scammell from information Gathered, in Particular, Between February 2004 to June 2004 [The Scammell Report]*, 2004, Hobart, Tasmania

the north east of Tasmania. The immediate response from the Tasmanian government and the chemical industry association, CropLife Australia, was a vehement attack on the report. It was the publication in the media of this event that prompted my interest in the devil cancer. In the course of the research therefore, not only do I analyse the scientific research using Hess's concept of undone science, I also interrogate why the *Scammell Report* provoked such a response.

Plantation forests are important to both the forestry industry and the Tasmanian government and were linked to a concerted effort by both to establish Australia's largest pulp mill in the north of the state. The plantation forests, especially the hardwood eucalypts, were seen as a solution to the long controversy over the logging of old-growth and native forests, as it has been proposed that plantation forests will replace these resources. The attempt by Gunns Limited to establish a pulp mill has failed but the growth in plantation forestry continues.

In Tasmania, there are three distinct struggles taking place and in order to gain an insight into the role of the participants, I conducted unstructured interviews. All three struggles can be linked to the rapid development of plantation forests in that state. The first struggle is to save the Tasmanian devil from a transmissible cancer, Devil Facial Tumour Disease (DFTD). This struggle is not a controversy. There is no group of activists or scientists contesting the allograft theory. However, I will argue that an alternative competing hypothesis exists - chemicals used in plantation forests, and known to be harmful, may have contributed to the devil cancer.

The second struggle concerns the chemical contamination of waterways by pesticides used in plantation forests with many activists seeking action from the government and the forestry industry. This struggle has since included an attempt to confirm or deny that eucalypt trees in plantation forests are genetically modified. The third struggle, connected to the second, was to stop the proposed building of a pulp mill. With the failure to establish the mill, this struggle has abated. The struggles are linked by the forestry industry's need to maintain the plantation forests. The two latter struggles, unlike the first, continue to be the subjects of considerable public controversy in Tasmania.

I began by contacting scientists involved in the devil cancer research, in an attempt to gain a better insight into their roles. As a non-scientist I needed to become familiar with the different roles and research being undertaken. I conducted unstructured interviews allowing the participant to lead the conversation but guided by the use of relevant themes. These conversations, although initiated on a theme, gave me an understanding of the issues.

2.11 Intervention

According to Brian Martin, in the science studies field intentional and planned intervention is rare. In his article, "Sticking a Needle into Science: The Case of Polio Vaccines and the Origin of AIDS", Martin describes his experiences of partisan intervention.[76] Martin reports that one of the benefits of intervention is the large volume of correspondence received from the activists and scientists. Similarly, my intervention in the controversial issues also generated new ideas and strategies along

[76] Martin B, 1996, Sticking a Needle into Science: The Case of Polio Vaccines and the Origin of AIDS, *Social Studies of Science*, Vol. 26(2), pp 245-276

with confidential material, drafts of letters, articles, submissions and emails. Like Martin, the benefit to me was that had I not been perceived as a participant, I would not have been privy to this information. I was also able to make enquiries, raise issues and questions, and provoke responses from which I was able to evaluate the veracity of my own assessments of the various situations as they arose. Sharon Beder also undertook an interventionist role in her investigation of the Sydney Water Board's system of disposing of sewerage from ocean outfalls and the subsequent pollution of Sydney beaches.[77] In her investigation it was the actions of participants that prompted her to 'delve deeper' into the issue.[78] I have also found that actions of participants have guided my research, leading to new discoveries.

2.11.1 Intervention in the chemical contamination controversy

I approached the activists involved in the chemical contamination controversy as a researcher willing to assist in their aim to control the use of hazardous chemicals. As the controversy is centred in Tasmania and I conducted the majority of my research from mainland Australia, most of the contact was via email or telephone, although I did travel to Tasmania and elsewhere to speak to activists in person. Initially, I spoke to the oyster farmers in St Helens on the east coast of Tasmania who had instigated an independent investigation into the cause of the mass mortality of their oysters in the Georges Bay at St Helens. They related local anecdotal knowledge about the practices of chemical use in plantation forests. It was from these initial conversations that I gained a sense of the seriousness of the problem of water contamination in Tasmania.

---

[77] Beder S, 1996, Sewerage treatment and the engineering establishment in Brian Martin (ed), *Confronting the Experts,* State University of New York Press, Albany, NY
[78] ibid, p 12

On 29th April 2005 I contacted Craig Lockwood, an oyster farmer and activist, via email and asked if he could put me in contact with Alison Bleaney. Alison was the local Area Medical Practitioner and an activist who had published with Marcus Scammell, marine ecologist, the *Scammell Report* in 2004. This report had made a correlation in time and space between the increase in plantation forests, the ongoing oyster health problems and mass mortality and the Tasmanian devil disease. It concluded that further research was needed, including toxicity assessments of water following aerial spraying and subsequent rainfall events, and the biological monitoring of non-target organisms. It also recognized that this research would take several years. As an alternative, it called for the implementation of the precautionary principle to immediately halt the aerial spraying of chemicals in the plantations in the Georges River catchment until such practices could be shown to be safe. Although this action has not been implemented, some concessions on the part of the Tasmanian government and the forestry industry have been made, such as monitoring of surface water. However, the continued detection of chemicals used in plantation forestry indicates the issue is far from resolved.[79]

In July 2005 Alison sent an email to say she would like to have a chat. I sent my telephone number and we soon developed a mutually beneficial relationship. We assisted each other by communicating, via email, telephone and in person, our specific knowledge on each aspect of the controversy as it arose. Alison also kept me informed of relevant conferences, talks, meetings and discussions, which I subsequently attended, when practical. For the research, I travelled to Hobart, Launceston, Melbourne, Sydney

---

[79] Tasmanian Government, Department of Primary Industries, Parks, Water and the Environment, 2014, Latest Pesticide Water Monitoring Results. Available at: http://www.dpipwe.tas.gov.au/inter.nsf/WebPages/LBUN-96T943?open last accessed 3 April 2014

and Brisbane. On these trips I was able to share knowledge with other activists involved in similar controversies thus expanding my knowledge.

In June 2007 I was invited by Alison to attend a meeting in Canberra organized by the Australian Pesticides and Veterinary Medicines Authority (APVMA), the government regulators, to discuss a review of registration for the chemical atrazine. At this meeting, I was introduced to Jo Imming, a member of the APVMA Community Consultative Committee and the National Toxics Network. I also met and spoke with Professor Tyrone Hayes, head of Integrative Biology at the University of California and an outspoken activist against the use of atrazine. He has undertaken many studies on the effects of atrazine on frogs and it was the concerns he raised that focused my attention on this particular chemical. In August 2008 I attended a Society of Environmental Toxicology and Chemistry (SETAC) Conference in Sydney where I again met and talked with Dr Scammell and Professor Hayes. It was at this conference, following Hayes's presentation, that a pro-industry scientist, who was also a former colleague, challenged the veracity of his data. In 2009 I attended the Combined Scientific Meeting of the Tasmanian Haematology, Immunology and Neoplasia Group (THING) organised by members of the Menzies Research Institute and DPIPWE in Launceston.

Through my association with Alison I also gained access to the media. This included, Matthew Denholm, the Tasmanian correspondent for *The Australian* and John Watts of *The Guardian* newspaper in the United Kingdom. However, following discussions based on my research, the correspondents informed me that because the issue was politically sensitive, and some feared legal action if they reported my findings, nothing ever came of the conversations.

On a more personal level Alison and I exchanged ideas and drafts of newspaper articles, letters, submissions and strategies. This correspondence would be conducted via email, each seeking comments, suggestions, appraisal or assessment depending on the type of material being produced. Likewise we would send ideas, chapters and articles for comments and verification. This process engendered a deep respect for each other's opinions, which sometimes differed, and was extremely useful as a sounding board for ideas. This approach gave me access to information via personal emails and facilitated my introduction to DFTD research scientists. This also led to my being made privy to confidential information.

2.11.2 Intervention in the proposed pulp mill activism

I was invited by activists to attend meetings of the Tasmanians Against the Pulp Mill (TAPP) group where I was viewed and introduced as a supporter and as such gained access to other activists and information. These meetings provided a forum for members actively seeking to address what they saw as corrupt or unjust practices of both the Tasmanian government and the forestry industry. My experiences and conversations encouraged me, as Beder found in her research, to 'delve deeper'. These activists gave freely of their information at all times. I met and spoke to Frank Strie, a former forester whose expertise allows him to expose flaws in the forestry industry's claims of best practice.

I have interviewed the Deputy Mayor of the Meander Valley Council, Bob Loone, in relation to chemical use practices in the plantation forests. I was also given the opportunity to observe devils feeding on a carcass in the wild on the west coast of Tasmania.

I sometimes took an interventionist role at the meetings I attended. At one particular meeting in Launceston in July 2010 I advised an activist group, Tasmanians Against the Pulp Mill, not to be disappointed that they were not included in a government round table meeting on forestry. I advised them that according to Martin's backfire model, official channels are best avoided. They accepted this advice in good faith. It transpired that the round table meetings were held in secret and included members of the Greens Party, Forestry Tasmania, the Wilderness Society and Environment Tasmania. The outcome was a proposed end to harvesting of native timber. Under conditions that proved contrary to the group's position, the round table gave tacit agreement to the proposed pulp mill. Given this outcome it is speculative as to whether their involvement would have produced a different result.

2.12 Approach to the DFTD scientific community

Unlike my involvement in the controversies where activists guided my actions, my approach to the scientific community was more systematic and impartial. In order to assess and understand the various roles of the scientists within the research community I undertook unstructured interviews. This process involved contacting the participants, initially via email, followed up with phone calls to make appointments for meetings. These meetings were generally informal conversations on the general theme of the devil disease and the scientist's role in that research.

Securing interviews with scientists often proved difficult. Rather than encountering openness and transparency many of my attempts to speak to the various participants in the DFTD research community resulted in responses that were guarded, clandestine and

on some occasions never took place. One group of scientists I was keen to interview worked at the DPIPWE Mt Pleasant laboratory in Launceston. My first line of communication was via emails, which were often not answered. I would then attempt to phone the individual. A principal scientist I tried to interview, and eventually conducted several lengthy interviews with, at first was refused permission by a manager to speak with me. She was told, as were others, they had first to seek DPIPWE approval. DPIPWE scientists informed me they were (incorrectly) told I was an oyster farmer looking to buy leases in Tasmania.

The scientists who were guarded may have been subject to the 'chilling effect' as described by Joanne Kempner, which often involves scientists engaging in self censorship.[80] Or, as described by Martin, their knowledge is 'suppressed' when scientists do not speak out because they are afraid they will be attacked if they do.[81] One scientist who had been the subject of suppression was extremely reluctant to speak with me but I was able to arrange a meeting through a mutual friend. This scientist provided me with valuable information but has reverted to being guarded, limiting further contact.

The younger post-graduate students were initially most forthcoming with their information and I gained valuable insights into the roles of the scientists within the community through these conversations. My first interview was with a young PhD student who had visited Tasmania from Brazil and joined the Save the Tasmanian Devil team. His research was critical in the immune studies into two devils, one of which

---

[80] Kempner J, 2008, The Chilling Effect, *PloS Medicine*, Vol **5**(11), pp 1517-1578
[81] Martin B, 1999, Suppression of Dissent in Science, *Research in Social Problems and Public Policy*, Vol **7**, pp 105-135

gained media attention when it was thought to be resistant to the cancer. I asked him the same question I asked of all the scientists – What is your role in the Tasmanian devil disease research? Another young PhD student told me that to say the devils are 'resistant' to the disease would in her opinion constitute 'scientific fraud'. These two young scientists who had initially given information freely, subsequently were no longer prepared to engage in conversations. At no stage have I made public any information given to me in these interviews.

Paradoxically, although scientists were warned not to speak to me, some scientists sought me out and freely gave me unpublished information and continue to do so. They are most critical of the DPIPWE's control over the scientific research and what they see as unusual practices. There is an internal DPIPWE controversy over the euthanasia of devils with DFTD. The veterinary scientists see the need to eliminate diseased devils from the environment so as to halt the spread, whereas zoologists undertaking population studies capture and release diseased devils. There are also those who are critical of the Tasmanian government's position on devil habitat, namely the lack of protection exacerbated by logging, plantation development and mining.

Scientists who delayed or deferred meeting with me to discuss their role in the research to save the Tasmanian devil have been the most problematic. However, following years of delay I finally did meet and had a number of very informative conversations with a scientist central to the disease research. Notwithstanding the obstacles to speaking to or obtaining meetings with scientists I continued to pursue these interviews in the course of my research.

2.13 Review of DFTD scientific literature

During my investigation of the scientific research, in an attempt to gain clarification and further insight into the studies, I continued to review the published scientific literature. Because the scientific research surrounding DFTD covered a new and novel disease I was able to cover its history from the beginning. The research is controlled by the Tasmanian DPIPWE hence it covers a single program created by an important hypothesis, which has been sustained by a continuing input of ideas, research and funding. As a non-scientist I have not tried to assess the validity of the scientific data or methodology of the papers but have focused on more general information, such as conveyed in the introductions and conclusions of the papers.

My analysis of the publicly available and published scientific literature on the Tasmanian devil facial tumour disease was undertaken along with a review of the media articles published in relation to the disease. By using the DPIPWE Save the Tasmanian devil website's list of published scientific articles and the *SCOPUS* and *Web of Science* databases I was able to review all the articles relating to DFTD. I also undertook a search of the UOW library's extensive database collection. Limitations may exist in that the databases may not cover all scientific journals worldwide but as mentioned above I was able to monitor all papers as they were published.

The DPIPWE *Save the Tasmanian devil* website contains a list of 65 articles published as of July 2011.[82] The list includes an article by Vetter et al published in *Rapid Communication in Mass Spectrometry* reporting the findings of the pilot study into the

[82] Tasmanian devil publications associated with Devil Facial Tumour Disease to July 2011. Available at: http://www.tassiedevil.com.au/tasdevil.nsf/file/82C18864F5819337CA2576CB0011569B/$file/DFTD_publications_Jul2011.pdf last accessed 19 November 2012

devil toxicology. It is the only peer-reviewed article relating to toxicology. No transmission studies to confirm that the devil cancer is contagious appear in this list. A detailed analysis of undone research into the Tasmanian devil cancer is documented in the following three chapters.

A further search of the *Factiva* database was undertaken to identify subject areas covered in the general media. The search was limited by date from 1 January 2003 to 24 March 2013, using the search terms 'Tasmanian devil' and 'devil facial tumour disease' and 'toxicology'. By using these methods of data collection I found that studies into toxicology and transmission were undone and were also not reported in the media.

2.14 Conclusion

Scientists are social beings whose views are affected by cultural, social and economic structures around them. Research has a political dimension via the role of vested interests in shaping scientific enquiry, particularly through funding. This politicalisation of science means that certain avenues of research receive preference over other areas, which leads to some research being abandoned or neglected – what Hess refers to as undone science. I have expanded this concept under the broader theories of ignorance and non-knowledge and developed a typology of practical and political reasons for undone research. In the following chapters I employ this typology to analyse the research into the Tasmanian devil cancer in depth and make comparisons with three other wildlife cancers.

My adoption of an approach using unstructured interviews or conversations on a theme provided various degrees of access to information. It has produced different outcomes with some participants guarded in their comments rather than open and cooperative in

discussing the devil disease. Whilst I attempted to conduct interviews with scientists conducting research into the Tasmanian devil cancer this proved problematic, as most of them were unwilling to speak to me. I tried numerous times over the course of my research to hold interviews but I found nearly all government-employed scientists to be secretive and evasive and unwilling to commit themselves to be interviewed. I did however speak to some of the more junior researchers, especially those engaged in PhD research, and they provided me with interesting insights into how the investigations were conducted. This latter information included unpublished reports and strategies, which added substantially to my thesis. Some interviews, including impromptu field trips, took place without the knowledge of interviewees' supervisors. I made one formal request to accompany scientists on a field trip; this was denied, even though there were public advertisements for volunteers. Meanwhile, my approach to activists encouraged them to share information and ideas allowing me to further develop evidence in support of my thesis.

To gain information about the devil disease research program I also researched and analysed the published literature on the Tasmanian devil cancer. The DPIPWE's website provides a full list of publications by researchers which I used as well as conducting a search of relevant databases. I also surveyed the local, national and international media for reports of conferences and meetings in relation to the devil disease.

My approach has been to use an informed non-specialist assessment of the issues to examine why a competing hypothesis that environmental carcinogens used in forestry plantations, such as pesticides, was delayed and then abandoned after an initial pilot

study. In the following chapters I analyse the devil cancer as an allograft, the selected research program and undertake an analysis of the toxicology studies.

Chapter 3 - The allograft theory

"A discovery is premature if its implications cannot be connected by a series of simple logical steps to canonical, or generally accepted, knowledge."[1]

3.1 Introduction

In 2005 the Tasmanian Department of Primary Industries, Parks, Water and Environment (DPIPWE) released a progress report *Research into the Devil Facial Tumour Disease (DFTD)* announcing ground breaking cytogenic studies.[2] These studies, undertaken at the DPIPWE Mt Pleasant laboratory, suggested that the devil cancer may have been a clone, that is a single cell line, passed from devil to devil through biting, which is the basis of the allograft hypothesis. An allograft, according to medical terminology, is the transplant of an organ or tissue from one individual to another of the same species but with a different genotype.[3] In June/July 2005 at the Wildlife Disease Association's International Conference, Cairns, Anne-Maree Pearse presented a paper titled 'Cytogenetic Support of the Allograft Theory of Transmission of Devil Facial Tumour Disease (DFTD) in Tasmanian Devils *(Sarcophilus Harrisii)'* outlining her initial findings.[4]

---

[1] Stent GS, 1972, Prematurity and uniqueness in scientific discovery, *Scientific American*, Vol 227, pp 84-93 in KE Studer & DE Chubin, 1980, *The Cancer Mission, Social Contexts of Biomedical Research,*Sage Publications, Beverly Hills, London, p 25

[2] Tasmanian Government, Department of Primary Industries, Water and Environment, 2005, *Research into the Tasmanian Devil Facial Tumour Disease (DFTD) Progress Report*, DPIWE, Hobart, p 4

[3] Definition of allograft, Medicinenet.com. Available at: http://www.medterms.com/script/main/art.asp?articlekey=30941 last accessed 23 January 2012

[4] Pearse AM, 2005, Cytogenetic support of the allograft theory of transmission of devil facial tumour disease (DFTD) in Tasmanian devils (*sarcophilus harrisii*), *Wildlife Disease Association International Conference Proceedings,* Cairns, Queensland, Australia

Pearse, along with her technical assistant, Kate Swift subsequently published these initial findings in the prestigious journal *Nature* in 2006.[5] The announcement that the cancer was contagious, an allograft, had occurred two years after the initial 2003 scientific meeting held by the DPIPWE to develop a strategy for dealing with the devil cancer. According to this strategy poisons or toxins were to be tested to assess their role in the devil cancer.[6] Coincidentally, the allograft hypothesis followed the publication in 2004 of the *Scammell Report* documenting a helicopter crash in the St Helens water catchment. The *Scammell Report* made a correlation in time and space between the introduction of plantation forests, chemical use and the outbreak of the devil cancer. The report called for the implementation of the precautionary principle, because of scientific uncertainty, to halt aerial spraying of chemicals until further studies could be undertaken.

Based on Pearse's preliminary observations, and despite the possibility that an alternative hypothesis existed, the Tasmanian devil research, under the guidance of the DPIPWE, chose a pathway that supported the proposed allograft hypothesis. This approach creates, according to David Hess, the science that flourishes on the vine. The result is that some studies are undertaken whilst other studies, supporting competing alternative hypotheses, are abandoned or neglected and left undone.

In this chapter I begin by interrogating the claims made by Pearse and Swift in the article published in *Nature* in 2006. I then undertake a comparison of the two published transmissible cancer programs, Canine Transmissible Venereal Tumour (CTVT) and DFTD. I then focus on an analysis of the DFTD research publications

[5] Pearse AM & Swift K, 2006, Transmission of devil facial-tumour disease, *Nature*, Vol. 439(2), p 549
[6] Tasmanian Government, Department of Primary Industries, Water and Environment, 2005, *Research into the Tasmanian Devil Facial Tumour Disease (DFTD) Progress Report*, DPIWE, Hobart, Appendix 1

including those listed on the DPIPWE *Save the Tasmanian Devil* website and those I have found in my search of *SCOPUS* and the Web of Science as described in the previous chapter.

3.2 The Pearse and Swift article

The Pearse and Swift article was published in the Brief Communications section of the scientific journal *Nature* in 2006.[7] It comprised a single page with few references and an online supplement. The Brief Communications section of *Nature* magazine, which has since been discontinued, was designed to announce to the general public, new and exciting preliminary discoveries. Despite the brevity of this preliminary paper, it has become the most cited in the DFTD research (see Appendix A). The word allograft did not appear in the *Nature* article title, but the hypothesis that the cancer is contagious has been referred to by writers in *Nature* and subsequent publications as the allograft theory. In the article Pearse and Swift supported the hypothesis that the devil cancer is transmissible by making two claims based on scientific observations and by presuming two precedents. In the next section, I undertake an analysis of the two claims and in the following section I will analyse the precedents.

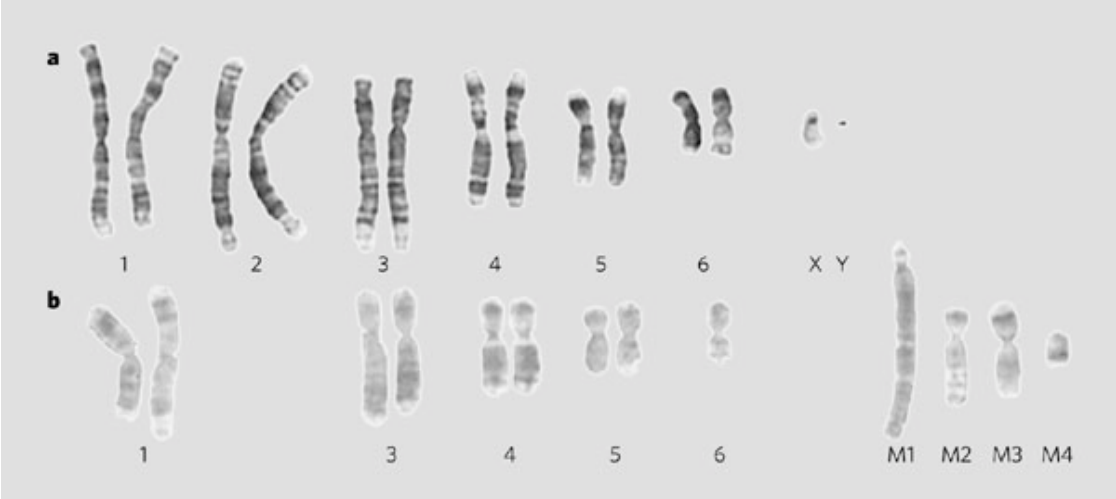
3.3 The two claims

Pearse and Swift's cytogenetic findings, shown in Figure 3.1 below, form the basis of the first claim. It is proposed that the devil tumour is transmissible because the 'similarity in the karyotype of these malignant tumours means that they could be infective' which is supported by the statement 'that the chromosomes in these tumours have undergone a complex rearrangement that is identical for every animal studied'.[8] It

[7] ibid.
[8] ibid.

is the basis for the proposal that 'an infectious cell line is passed directly between the animals through bites' and like the transmissible venereal sarcoma in dogs the tumour cells are clones.[9]

Figure 3:1 Chromosomes of facial tumours from Tasmanian devils



a, Normal karyotype for a male Tasmanian devil (14 chromosomes, including XY). **b,** Karyotype of cancer cells found in each of the facial tumours of all 11 animals studied (13 chromosomes, with no sex chromosomes, no chromosome-2 pair and only one chromosome 6; the long arm of one chromosome 1 was deleted; four additional marker chromosomes were present (M1–M4).[10]

This first claim, that the chromosome rearrangements in the tumours were identical, has since been challenged by two conflicting findings. The first appeared in July 2008, when it was revealed that the cancer was evolving into several different cytogenic strains.[11] The different strains, interpreted as different chromosomal rearrangements, conflict with the original claim that the chromosomal arrangements in every devil studied were identical. The second came in a recent paper by Deakin *et al* published in 2012 which provides the following explanation for the different strains:

---

[9] ibid.
[10] ibid.
[11] Save the Tasmanian Devil, *New Strains of DFTD Emerging*, Joint Initiative Tasmanian Government and the University of Tasmania. Available at: http://www.tassiedevil.com.au/research.html last accessed 10 December 2008

> Our observation of limited divergence into several strains and sub-strains implies that the basal tumour karyotype was established early in tumour evolution, and has remained extraordinarily stable over the subsequent fifteen years. Thus an alternative hypothesis is that all tumour strains are the same age and represent various subclones of an original, heterogenous tumour in the sentinel animal. However, subclones must have been all capable of self-renewal and tumour initiation, which seems rather unlikely as few cells independently acquire properties of CSCs [clonal stem cells].[12]

Deakin et al provide an alternative hypothesis in relation to the inconsistency in the strains. This however, rather than clarifying the situation, enhances the ambiguity by raising further uncertainties. The Deakin et al paper also revealed a further inconsistency by Pearse and Swift, with regards to the interpretation of the devil karyotype, for chromosomes 1 and 2.[13] This is discussed in more detail in section 3.8 below.

The second claim relied on the observation in a single devil of 'a pericentric inversion of chromosome 5 in its constitutional karyotype' being interpreted as different from the chromosomes in the cancer cells.[14] This interpretation was taken to mean that because the anomaly was in the devil's own cells and not in its cancer cells, the cancer had not arisen in the host devil's own tissue. Apart from research undertaken separately by Pearse at the DPIPWE Mt Pleasant laboratory and Janine Deakin at the Australian National University, the results of which have not been published, this claim has not

---

[12] Deakin JE, Bender HS, Pearse AM, Rens W, O'Brien PCM, Ferguson-Smith MAF, Cheng Y, Morris K, Taylor R, Stuart A, Belov K, Amemiya CT, Murchison EP, Papenfuss AT, Graves JAM, 2012, Genomic Restructuring in the Tasmanian Devil Facial Tumour: Chromosome Painting and Gene Mapping Provide Clues to Evolution of a Transmissible Tumour, *PLoS Genetics*, Vol 8(2), pp 1-16, p 11

[13] Cited Martin PG & Hayman DL, 1967, Quantitative comparisons between karyotypes of Australian marsupials from 3 different superfamilies, *Chromosoma*, Vol 20 pp 290-310 and CRES, 2006, *Sarcophilus harrisii* (Tasmanian devil) in SJ O'Brien, JC Menninger & WG Nash, (eds) *Atlas of Mammalian Chromosomes,* New York, Wiley p 30 in JE Deakin, HS Bender, AM Pearse, W Rens, PCM O'Brien, MA Ferguson-Smith, Y Cheng, K Morris, R Taylor, A Stuart, K Belov, CT Amemiya, EP Murchison, AT Papenfuss & JA Marshall Graves, 2012, Genomic Restructuring in the Tasmanian Devil Facial Tumour: Chromosome Painting and Gene Mapping Provide Clues to Evolution of a Transmissible Tumour*, PLoS Genetics*, Vol 8(2) pp 1-16

[14] ibid.

been scientifically verified.[15] There was also a trial breeding program conducted at the Trowunna Wildlife Park in Tasmania but the resulting devil offspring escaped before studies could be undertaken.[16]

The allograft hypothesis, which proposes that the devil cancer is transmissible, passed from devil to devil via biting, is an anomaly to the conventional theory of cancer as a non-contagious disease. It could therefore reasonably be argued that either the evidence is flawed or the data interpretations are wrong, but no independent scientific studies have either verified or discredited the claims. Possibly hampering independent studies is the listing of the Tasmanian devil as endangered under the *Tasmanian Protected Species Act* and the Federal *Environmental Protection and Biodiversity Conservation Act 1999 (EPBC Act)*. This status means it is an offence to 'take' the Tasmanian devil or any part of the devil because it is protected and strict regulations control its use, including its use in scientific studies. Two scientists who conveyed a desire to undertake toxicology tests on devil cells were dissuaded due to legal implications.[17] The exact nature of the studies undertaken into the devil cancer is the topic of section 3.6 below.

3.4 The precedents

The authors added further support to the claim that the devil cancer was contagious by proposing two precedents. The first precedent is the canine transmissible venereal tumour (CTVT), a sexually transmitted cancer in dogs, and the second is tumour transmission in humans through organ transplants.[18]

---

[15] Personal communication with AM Pearse, October 2012
[16] ibid.
[17] I was personally involved in discussions where these scientists debated the risks involved in procuring devil cells for research.
[18] The dog cancer is known by various names but in this thesis I will refer to it as Canine Transmissible Venereal Tumour (CTVT) its more recent title.

The human organ transplant tumour is not a particularly strong precedent as demonstrated in a review of human organ transplants published in 2002.[19] The results showed only rarely did tumours arise - approximately 0.04% of transplants resulted in tumours being established and of these, only one third were donor-transmitted tumours. This is in sharp contrast to DFTD where devil populations have been reduced by 90% in some areas.

The CTVT precedent on the other hand would appear to be stronger and is analysed in a comparison with DFTD in the following section.

3.5 A comparison between CTVT and DFTD allograft programs and undone science

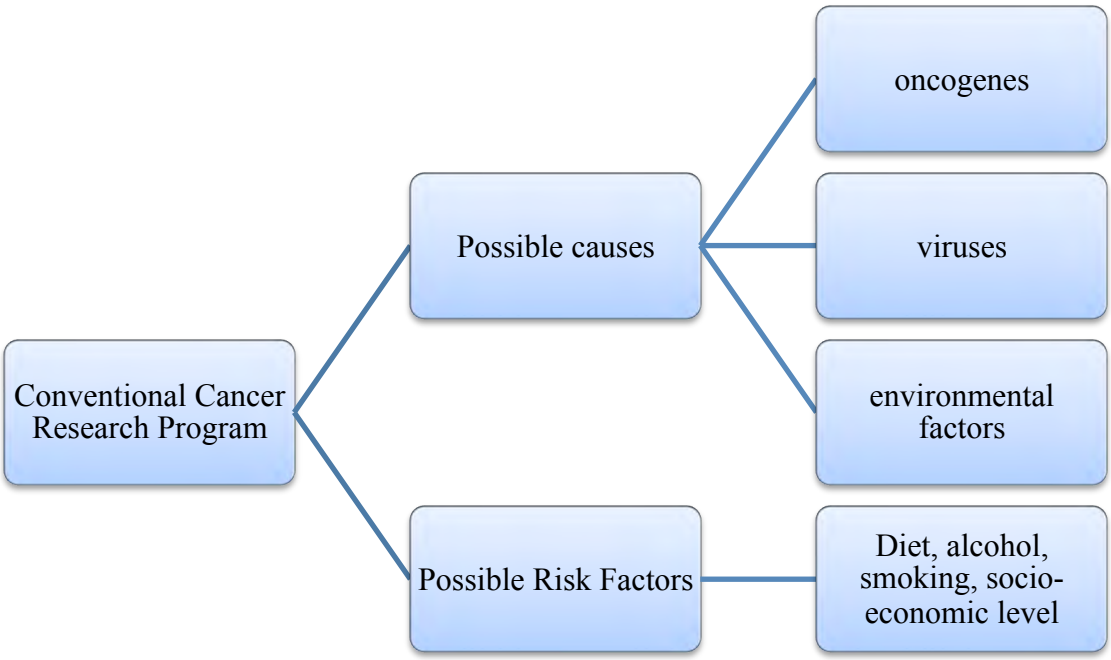
The scientists working on the Tasmanian devil disease have focused their studies on the hypothesis that the devil cancer is transmissible – an allograft. The second precedent cited by Pearse and Swift was the only other known transmissible cancer, the sexually transmitted dog tumour CTVT. In this section I compare the two programs based on a literature review of published studies. I do not attempt to assess the validity of the scientific methods or the findings of the research. However, through the comparison, I will identify whether studies in either program have been left undone and if so, determine if they have been abandoned or neglected for either practical or political reasons.

[19] Kauffman HM, McBride MA, Cherikh WS, Spain PC, Hanto DW & Delmonico FL, 2002, Donor-related malignancies, *Transplantation Reviews*, Vol 16(4), pp 177-191

The framework for my comparison incorporates the ideas of Lakatos's conventional progress of a research program and Hess's political sociology of science. According to Lakatos, a research program begins with a simple hypothesis or concept, based on an initial discovery.[20] This new discovery often takes place within an established research program and can initiate a whole new line of inquiry. As the new program develops it establishes a 'hard core' of theory, which can eventually be surrounded by auxiliary hypotheses. The dominant or 'hard core' theory of cancer causation, surrounded by auxiliary hypotheses, is shown in diagram Figure 3:2 below. Within this context, I have viewed the transmissible cancer theory as an auxiliary hypothesis to the 'hard core' orthodox or conventional theory of cancer causation. The transmissible cancer theory can also be viewed as an inconsistency or anomaly in relation to the hard core of the cancer research program. However, research programs can exist and progress irrespective of anomalies.[21] Lakatos's ideas are used as a guide to my chronological retracing of the published articles for both programs. A more political analysis of the development of the research program will be informed by Hess's concept that not only are certain studies selected by interest groups or elites for research but this then leads to some studies being left undone.

[20] Lakatos I, 1978, *The methodology of scientific research programmes*, Cambridge University Press, Cambridge
[21] ibid.

Figure 3:2 Conventional Cancer Research Program



Cancer research is extremely complex. The diagram above gives a simple schema where, for example, environmental factors include pharmaceuticals, biological agents, natural metals and radiation as well as chemicals agents. The dominant theory however, focuses on oncogenes, viruses and possible risk factors with less attention given to exposure to harmful environmental chemicals. The potential for environmental contaminants to cause cancer is documented in Robert Proctor's book *Cancer Wars*.[22] The allograft hypothesis is a new auxiliary theory of cancer causation whereby cancer is contagious, transmitted between unrelated hosts within a species.

The CTVT transmissible tumour theory is a mature research program strongly supported by both laboratory and genetic studies. In comparison, the DFTD studies

[22]Proctor RN, 1995, *Cancer Wars, How Politics Shapes What We Know and Don't Know About Cancer,* Basic Books, New York

attempting to prove that DFTD is also a transmissible tumour have been both speculative and ambiguous. In order to draw a comparison between the two transmissible cancer theories, I will first describe the development of the CTVT research program, followed by the DFTD research program. Elizabeth Murchison, a DFTD researcher, also published a comparison between the CTVT and DFTD research. A review of her paper is provided at the conclusion of this chapter.

3.5.1 CTVT research program

The CTVT research program is a logical progression that began with an observation of a tumour that is similar, occurring in dogs around the world, particularly in the warmer temperate areas. For my analysis I have focused on a number of recent reviews and the more significant studies from specialist journals covering over 1200 studies into CTVT. It is speculated that the tumour has evolved over a period of between 200 and 2,000 years. It was first proved transmissible by Novinski, a Russian veterinarian, in 1876 when he transplanted viable tumour tissue between unrelated dogs and found the tumour established in the new host.[23] Since the early 1900s many studies into CTVT have been undertaken to test for its transmissibility and its mode of transmission. There have also been many studies to try to identify the type of cancer, which have ranged from claiming it is a sarcoma, to a parasite. Recent sophisticated genetic studies have given support to the claim that the tumour is identical in all dogs. However, these studies are limited because they only rely on recent evidence; comparisons with earlier tumour cells are not possible, as they no longer exist.

[23] Novinski MA, 1876, Zur Frage uber die Impfung der Krebsigen Geschwulste. Zentralbl. Med. Wissensch, Vol 14, pp790-791 cited in C Murgia, JK Pritchard, SY Kim, A Fassati & RA Weiss, 2006, Clonal Origin and Evolution of a Transmissible Cancer, *Cell*, Vol 126, pp 477-487

Scientific studies into CTVT have been undertaken for over a century in numerous laboratories and universities across the world. Some of these institutions include: University of Zimbabwe; Utrecht University; the University of Agriculture and Technology in Nagar, India; University College London; University of Chicago and the University of Glasgow Veterinary School. Many of the funding sources are unknown but one study was funded from the Wellcome Trust and the Middlesex Hospital.[24] CTVT appears to have developed into a mature research program now operating autonomously and separate from conventional cancer programs. Whilst not all proposed studies have been undertaken into CTVT, the undone studies and anomalies have not undermined the CTVT transmissible cancer theory. At present there is no competing hypothesis, except possibly a viral aetiology, which is still subject to debate. Prior to 2006 CTVT was the only transmissible cancer research program. The majority of studies undertaken for CTVT fall into the following categories: transmission studies; genetic studies for chromosomal stability; molecular fingerprinting and diagnostic markers; and immunity. The following section provides a timeline of the more recent studies into CTVT.

3.5.2 A chronology of recent CTVT studies

In 2000 Utpal and Arup Das published a review of studies into Canine Transmissible Venereal Sarcoma (a variant name for CTVT). In relation to the CTVT studies as to its type, their review cites the following studies - the 1905 study by Bashford and colleagues who concluded that CTVT was not a sarcoma, but an infective granuloma; a study by Sticker in 1906 incorrectly calling it a 'contagious lymphoma'; and Feldman in 1929 associated the forceful nature of sexual intercourse between dogs and genital

[24] Murgia C, Pritchard JK, Kim SY, Fassati A & Weiss RA, 2006, Clonal Origin and Evolution of a Transmissible Cancer, *Cell*, Vol 126, pp 477-487

injury in both sexes with susceptibility to transplantation of the tumour cells.[25] Karlson and Mann are cited as providing proof of the transmissibility of the cancer when in 1952 they succeeded in following the passage of the tumour through 40 generations of dogs over a period of 17 years.[26]

During the course of the studies, it was noted that the dog tumours were found to develop at other sites, on the skin or in and around the mouth, but this was generally associated with a genital tumour. In 1966 Higgins provided an explanation, when he 'suggested that many of the cutaneous sites where these tumours are found represent lesions caused by biting and scratching, common in stray dogs, which predispose the skin to implantation of the tumour'.[27] According to Das and Das '[Higgins] observed scars in the skin above … tumours, suggestive of previous wounds.[28]

In 1970 Wright et al undertook genetic studies of the dog transmissible tumour cells and found there were usually between 58-59 chromosomes, whereas the normal number is 78 chromosomes in the somatic cells of dogs.[29] According to Das and Das these abnormal features of the tumour cells are consistent and unique, in that they have been observed in tumours of dogs across different continents.[30] Adams and Slaughter

---

[25] Feldman WH, 1929, So-called infectious sarcoma of the dog in an unusual anatomic situation, *American Journal of Pathology*, Vol 5, pp 183-194 cited in U Das & AK Das, 2000, Review of Canine Transmissible Venereal Sarcoma, *Veterinary Research Communications,* Vol 24, pp 545-556

[26] Karlson AG & Mann FC, 1952, The Transmissible venereal tumor of dogs: Observations on forty generations of experimental transfers, *Annals of the New York Academy of Sciences,* Vol 54, pp 1197-1213

[27] Higgins DA, 1966, Observations on the canine transmissible venereal tumour as seen the the Bahamas, *Veterinary Record*, Vol 79, pp 67-71 cited in U Das & AK Das, 2000, Review of Canine Transmissible Venereal Sarcoma, *Veterinary Research Communications,* Vol 24, pp 545-556, p 548

[28] Das U & Das, AK, 2000, Review of Canine Transmissible Venereal Sarcoma, *Veterinary Research Communications,* Vol 24, pp 545-556, p 548

[29] Wright DH, Peel S, Cooper EH & Huges DT, 1970, Transmissible venereal sarcoma of dogs: A histochemical and chromosomal analysis of tumours in Uganda*, European Journal of Clinical Biological Research* Vol 15, p 155 in ibid.

[30] Das U & Das, AK, 2000, Review of Canine Transmissible Venereal Sarcoma, *Veterinary Research Communications,* Vol 24, pp 545-556, p 549

confirmed the similarities between the features of the primary tumour and the secondary tumours, thus strengthening the evidence for the consistency of the abnormalities in the cells of CTVT.[31] The same chromosomal patterns are also maintained in cell culture.[32]

In 2006 Murgia et al undertook molecular fingerprinting to identify the defective gene responsible for the cancer and matched DNA sequencing in dogs from around the world confirming the tumours to be genetically identical.[33] They also identified three lines of observation they claimed confirmed CTVT as a transmissible cancer:

1. CTVT can only be experimentally induced by transplanting living tumour cells, and not by killed cells or cell filtrates;
2. Tumour karyotype is aneuploid but has characteristic marker chromosomes in tumours collected in different geographic regions
3. A long interspersed nuclear element (LINE-1) insertion near c-myc has been found in all tumours examined.

They also claim,

> [a]lthough the tumor is highly aneuploid, the karyotype is remarkably constant in tumors from the United States, Kenya and Japan. Therefore, its genome diversity at the chromosomal level appears to have stabilized early in its emergence as a transmissible parasite, and our studies revealed only moderate diversification of microsatellite DNA sequences.[34]

In terms of the instability of chromosomes they confirmed that CTVT does not appear to exhibit a mutator phenotype in terms of microsatellite instability, and neither does it exhibit progressive chromosome instability. They also stated that '[i]t is not evident

[31] Adams EW & Slaughter LJ, 1970, A canine venereal tumour with metastasis to the brain, *Pathologia Veterinaria,* Vol 7, pp 498-502 cited in ibid.
[32] Adams EW, Carter LP & Sapp WJ, 1968, Growth and maintenance of the canine venereal tumour in continuous culture, *Cancer Research*, Vol 28, pp 753-757 cited in U Das & AK Das, 2000, Review of Canine Transmissible Venereal Sarcoma, *Veterinary Research Communications,* Vol 24, pp 545-556
[33] Murgia, C, Pritchard JK, Kim SY, Fassati A & Weiss RA, 2006, Clonal Origin and Evolution of a Transmissible Cancer, *Cell*, 126, pp 477-487
[34] ibid, p 484

from our data whether the "infective dosage" is a single cell or a bolus of tumour tissue' but they suspect the latter.[35]

In concluding, Murgia et al note that a definitive analysis based on DNA markers for DFTD, such as used for CTVT, was awaited.[36] It remains to be determined if epigenetic factors affect the progressive and regressive phases of tumour growth for CTVT. The stable genome for CTVT has aided the host's survival and onward tumour transmission 'whereas the evolutionary dynamics of a "selfish", dead-end tumour typically progresses toward greater autonomy and malignancy'.[37] Hence DFTD contrasts with CTVT in that it is highly virulent - killing all of the affected animals. They propose that a similarity between DFTD and CTVT may be the initial facilitation of CTVT within a partially inbred population. But today it exists within mixed-breed dogs, particularly strays. Further, in 2006 David Dingli and Martin Nowak published an article in *Nature* on both CTVT and DFTD concurring with Murgia et al.[38] In 2009 Purohit in a review stated that CTVT is the only proven example of a naturally occurring tumour that is transmitted as an allograft by cell transplantation.[39]

CTVT is commonly found in dogs aged between two and five years that are sexually active. The dog cancer is benign, not fatal. CTVT appears to overcome the histocompatibility barriers to escape from the host's immune surveillance, however a

---

[35] ibid.
[36] Murgia, C, Pritchard JK, Kim SY, Fassati A & Weiss RA, 2006, Clonal Origin and Evolution of a Transmissible Cancer, *Cell*, 126, pp 477-487
[37] ibid, p 485
[38] Dingli D & Nowak MA, 2006, Infectious tumour cells, *Nature*, Vol 443, pp 35-36
[39] Purohit, GN, Canine Transmissible Venereal Tumour: A Review, 2009, *The Internet Journal of Veterinary Medicine,* Vol 6(1). Available at: http://archive.ispub.com/journal/the-internet-journal-of-veterinary-medicine/volume-6-number-1/canine-transmissible-venereal-tumor-a-review.html#sthash.mqPhdT27.dpbs last accessed 30 September 2013

response is eventually mounted and the cancer goes into remission.[40] This regression leads to tumour immunity that prevents successive occurrences.[41] However in immunocompromised animals and puppies there is metastasis (secondary tumours).[42]

In relation to a viral hypothesis Mukaratirwa and Gruys found CTVT has the ability to be transplanted to other members of the canine family such as foxes, coyotes and wolves, which suggests a viral cause.[43] But this hypothesis has been discounted elsewhere because it can only be experimentally induced by transplanting living tumour cells and not by dead cells or cell filtrates, so some scientists remain skeptical of the viral hypothesis. Meanwhile, Das and Das in two studies found oncogenic viral particles, that had not been seen through an electron microscope in the tumour cells, suggestive of the agent possibly being a type C retrovirus.[44] It would appear that the viral hypothesis is still to be resolved.

3.5.3 The DFTD research program

For the purpose of comparing the DFTD research program I have focused on the studies listed on the DPIPWE *Save the Tasmanian Devil* website (Appendix B). It is, however, not a complete list of all studies undertaken and published. I have included studies that do not appear on the list but are relevant to the analysis. Important omissions from the list include studies undertaken by independent scientists highlighting the need for research into a chemical aetiology for DFTD and these are described below.

---

[40] Mukaratirwa S & Gruys E, 2004, Canine transmissible venereal tumour: cytogenetic origin, immunophenotype, and immunobiology, *Veterinary Quarterly*, Vol 25, pp 101-111
[41] Powers RD, 1968, Immunologic properties of canine transmissible venereal sarcoma, *American Journal of Veterinary Research*, Vol 29, pp 1637-1645
[42] Cohen D, 1985, The canine transmissible venereal tumour: a unique result of tumour progression, *Advances in Cancer Research*, Vol 43, pp 75-112
[43] Mukaratirwa & Gruys, 2003, Canine Transmissible venereal tumour: cytogenetic origin, immunophenotype and immunobiology. A review. *The Veterinary quarterly,* 25(3), pp 101-111
[44] Das U & Das AK, 2000, Review of Canine Transmissible Venereal Sarcoma, *Veterinary Research Communications,* Vol 24, pp 545-556

It was the proposed identical nature of the tumour chromosomes that gave rise to the claim that CTVT was a precedent for DFTD as referred to by Pearse and Swift in their 2006 *Nature* article. Pearse and Swift claimed that 'the devil's cancer (like the dogs') is infective'. All DFTD research scientists in the *Save the Devil* program accept this claim. The DPIPWE *Progress Report* also likened the devil allograft to the sexually transmitted dog tumour whose cells are stable, constant and highly specific aberrations – therefore suggestive of a cellular mode of transmission.

Research programs according to Lakatos progress from an initial observation made outside the conventional research program and proceed to form a new program. Or alternatively, the observation is made within an existing research program – in this case the CTVT transmissible cancer research program, and progresses from there. In this case, Pearse's observed stability of the devil tumour chromosomes and observation of a peri-centric inversion in one devil led to the development of the hypothesis that DFTD is a transmissible cancer. The program would then adopt conventional scientific methods to test the new claim. A chronological analysis of the studies in the DFTD program will serve to reveal the progression of the research.

The DFTD research program commenced in 2003 when the Tasmanian government, convinced that the Tasmanian devil disease was threatening the survival of the species, convened a meeting of scientists, which was closed to the public. As mentioned in Chapter 1, an outcome of the meeting was that a number of initial studies were

undertaken including: Richmond Loh's Masters degree[45]; the DPIPWE's own reports[4647]; and an AusVet Report[48].

3.5.3.1 A chronology of DFTD studies

In 2003 and 2004 two early studies were published in relation to the genetic diversity of devils. At the time these were not related to DFTD but became important in later research. The studies were undertaken by Menna Jones and colleagues and published in the journal *Molecular Ecology*. Jones was also to become a key scientist in future DFTD research. The first paper was published in 2003 and according to the abstract '[devil] populations are impacted by habitat clearance and anthropogenic mortality and genetic studies could be of value in informing levels of genetic diversity, mating system, dispersal and effects of natural and anthropogenic landscape features on gene flow'.[49] The study revealed 'moderate genetic variability across the species range'.[50] The second study, published in 2004, again investigated genetic diversity, finding that the northwestern population was the more genetically distinct.[51] The abstract concluded with the observation that there appeared to be stronger population subdivisions within carnivorous marsupials such as devils than in their placental mammal equivalents.[52]

---

[45] Loh RC, 2006, *The Pathology of Devil Facial Tumour Disease in Tasmanian Devils (Sarcophilus harrisii)*, Master of Philosophy, Murdoch University, Perth, Western Australia
[46] Tasmanian Government Department of Primary Industries, Water and the Environment, 2005, *Research into the Tasmanian Devil Facial Tumour Disease (DFTD) Progress Report,* Department of Primary Industry, Water and Environment, Hobart, Tasmania
[47] Tasmanian Government Department of Primary Industries, Water and the Environment, 2005, *Devil Facial Tumour Disease Update*. Available at: http://www.dpiw.tas.gov.au/inter.nsf/Attachments/LBUN-6FC79N/$FILE/DFTDUpdate.Aug05.pdf last accessed 18 September 2007
[48] AusVet Animal Health Services Pty Ltd, 2005, *Tasmanian Devil Facial Tumour Disease Response, Technical Workshop 29-31 August 2005, Final Report to Department of Primary Industries, Water & Environment, Tasmania,* Hobart, Tasmania
[49] Jones ME, Paetkau,D, Geffen E & Moritz C, 2003, Microsatellites for the Tasmanian devil (Sarcophilus Laniarius), *Molecular Ecology Notes*, Vol 3, pp 277-279 p 277
[50] ibid.
[51] Jones, ME, Paetkau, D, Geffen E & Moritz C, 2004, Genetic diversity and population structure of Tasmanian devils, the largest marsupial carnivore, *Molecular Ecology*, Vol 13, pp 2197-2209
[52] ibid, p 2197

In 2005 Corey Bradshaw and Barry Brook[53] published in the journal *Ecography* results of a study relating to DFTD that first suggested a connection between facial lacerations and transmission.[54] In support of the connection they cited both Guiler's[55] and Kabat's[56] observations that '[a]gonistic [conflict] interactions often lead to severe facial lacerations [in devils] that may increase the transmission rate of pathogens between individuals'.[57] The reference to Guiler is a link that is incomplete, while the Kabat reference is a personal communication. They state '[o]ur models are still constrained by the lack of an explicit spatial component incorporating movement of infected individuals from disease-source regions to unaffected areas'.[58] These studies on genetic diversity, population dynamics and spatial movements of devils were to become the basis of the DFTD research program.

In 2006, along with the Pearse and Swift article published in February, a number of other articles were published, including a paper by Richmond Loh and colleagues in *Veterinary Pathology* on the definition of the devil cancer DFTD.[59] They confirmed the tumours to be a 'poorly differentiated malignant round cell neoplasm', qualifying the statement with 'the scarcity and primitive appearance of the desmosomes were not

---

[53] Both are now Professors of Ecology Evolution and Landscape Science, The University of Adelaide. Available at: http://www.adelaide.edu.au/directory/corey.bradshaw and http://www.adelaide.edu.au/directory/barry.brook last accessed 10 December 2012

[54] Bradshaw CJA & Brook BW, 2005, Disease and the devil: density-dependent epidemiological processes explain historical population fluctuations in the Tasmanian devil, *Ecography*, Vol 28(2), pp 181-190

[55] Guiler ER, 1992, *The Tasmanian devil*, St David's Park Publication with the online reference (?url_ver=Z39.88-2004&rft_val_fmt=info%3Aofi%2Ffmt%3Akev%3Amtx%3Abook&rft.genre=book&rft.btitle – this reference cannot be accessed.

[56] Cited personal communication.

[57] Bradshaw C & Brook B, 2005, Disease and the devil: dependent epidemiological processes explain historical population fluctuations in the Tasmanian devil, *Ecography*, Vol 2(2), pp 185-190, p 183

[58] ibid, p 188

[59] Loh R, Bergfeld J, Hayes D, O'Hara A, Pyecroft S, Raidal S & Sharpe R, 2006, The Pathology of Devil Facial Tumor Disease (DFTD) in Tasmanian Devils (Sarcophilus harrisii), *Veterinary Pathology*, Vol 43, pp 890-895

enough evidence to classify DFTD as a carcinoma'.[60] In concluding they stated that the '[t]ransmissibility of the tumor cells per se must be assessed to ascertain whether it satisfies Koch's postulates'.[61] Koch's postulates are four criteria formulated by Robert Koch and Friedrich Loeffler in 1884 to establish a causal relationship between an infectious microbe and a disease.[62] This study appears not to have been undertaken. A second paper published by Loh and colleagues again, in *Veterinary Pathology* in 2006, confirmed DFTD was consistent with cells of neuroectodermal[63] origin.[64] Noting there was little agreement on the cell type and classification of the neoplasm of DFTD, they stated,

> 'DFTD also shares some morphologic, immunohistochemical staining, and possibly epidemiologic features with canine [C]TVT, which is a round-cell tumor of the skin. However, [C]TVT is negative for S-100. Karyotyping by cytogenetic analysis has revealed complex chromosomal rearrangements in DFTD cells but the nature of the aneuploidy differed from that found in [C] TVT: DFTD cells were hypodiploid and contained chromosomal deletions and 4 complex marker chromosomes whose derivation was uncertain (A. Pearse, personal communication).'[65]

In concluding they further stated,

> '[a]n alternative explanation for the sudden occurrence of DFTD in multiple geographic locations across Tasmanian could be the occurrence of multiple concurrent epidemics owing to an unknown etiology. An epidemiologic analysis of DFTD should clarify this and may shed insights into the possible etiopathogenesis of the disease'.[66]

---

[60] Loh, R, Bergfeld, J, Hayes, D, O'Hara, A, Pyecroft, S, Raidal, S and Sharpe R, 2006, The Pathology of Devil Facial Tumor Disease (DFTD) in Tasmanian Devils (Sarcophilus harrisii), *Veterinary Pathology*, Vol 43, pp 890-895, p 894

[61] ibid, p 895

[62] Princeton University, Koch's postulates. Available at: http://www.princeton.edu/~achaney/tmve/wiki100k/docs/Koch_s_postulates.html last accessed 29 June 2013

[63] Dorland's Medical Dictionary for Health Consumers, 2007, Neuroectoderm - region of the early embryo that develops into the brain and spinal cord as well as into the peripheral nervous system, Saunders. Available at: http://medical-dictionary.thefreedictionary.com/neuroectoderm last accessed 29 June 2013

[64] Loh R, Hayes D, Mahjoor,A, O'Hara,A, Pyecroft S & Raidal S, 2006, The Immunohistochemical Characterization of Devil Facial Tumor Disease (DFTD) in the Tasmanian Devil (Sarcophilus harrisii), *Veterinary Pathology*, Vol 43, pp 896-903

[65] ibid, p 900

[66] ibid, p 902

This article also called for transmission trials to test Koch's postulates to confirm the allograft theory.

Claire Hawkins, Senior Scientist at DPIPWE, and colleagues also published the results of a study in 2006.[67] This study of devil population numbers was undertaken through regional spotlighting surveys and trapping studies to assess the decline in devil population numbers. They found '[n]o evidence for density dependence, or immunity, in DFTD'. They did find however, that in the northeast, prevalence remains high despite a reduction of 75-80% in the local population.[68] They advised that the Devil Disease Project Team would continue to analyse and to investigate changes in DFTD distribution, spread and impact, to identify any relationship between population density and DFTD prevalence.[69] They also found a significant decline (41%) in devil sightings since the first DFTD reports.

Hamish McCallum and Menna Jones, both part of the DPIPWE research team, also published a paper in *PLoS Biology* in October 2006, using DFTD as a case study for how to manage an emerging disease that is also a serious conservation threat.[70] They posed a number of questions, while at the same time claiming that the 'apparent spatial and temporal progression of the disease strongly suggests that it is infectious and that it is spreading'.[71] The article by McCallum and Jones concludes with the observation that

[67] Hawkins CE, Baars C, Hesterman H, Hocking GJ, Jones ME, Lazenby B, Mann D, Mooney N, Pemberton D, Pyecroft S, Restani M & Wiersma J, 2006, Emerging disease and population decline of an island endemic, *Biological Conservation*, Vol 131, pp 307-324
[68] ibid, p 319
[69] ibid.
[70] McCallum H & Jones ME, 2006, To lose both would look like carelessness: Tasmanian Devil Facial Tumour Disease, *PLoS Biology*, Vol 4(10), pp 1671-1674
[71] ibid, p 1671

'[t]he question of the nature of the transmission dynamics …might be important…but it is unlikely to have much short- to medium-term impact on devising appropriate management strategies. Selective culling is likely to be far more effective … [however] the likely key periods for disease transmission during the mating season are outside human control'.[72] The focus of this paper appears to be possible conservation measures rather than an attempt to understand the devil cancer itself.

In 2007 a number of articles on DFTD were published including those that appeared in a special September issue of a new journal *EcoHealth*. In this issue with a special focus on the devil decline there were four articles by DPIPWE researchers and two supporting articles. A paper by Menna Jones and colleagues from DPIPWE was included, which was on the conservation management of the Tasmanian devils. It reported encouraging preliminary results of the first suppression trials on Freycinet Peninsula on the east coast of Tasmania.[73] It however recognized that limiting spread or suppressing the disease on a large scale was not feasible. The trials on the peninsula were later abandoned.[74] In the same issue McCallum and colleagues, including Jason Wiersma from the Forest Practices Board, had an article titled 'Distribution and Impacts of Tasmanian Devil Facial Tumor Disease'.[75] The abstract describes a mark-recapture analysis and a preliminary epidemiological model. The authors concluded '[a]s

[72] ibid, p 1674
[73] Jones, ME, Jarman PJ, Lees, CM, Hesterman H, Hamede, RK, Mooney NJ, Mann D, Pukk, CE, Bergfeld J & McCallum, H, 2006, Conservation Management of Tasmanian Devils in the Context of an Emerging, Extinction-threatening Disease: Devil Facial Tumour Disease, *EcoHealth*, Vol 4(3), pp 326-337
[74] University of Tasmania, nd. Selective culling can't save the devils. Available at: http://www.utas.edu.au/tools/recent-news/news/selective-culling-cant-save-the-tasmanian-devil last accessed 9 December 2012
[75] McCallum, H, Tompkins, DM, Jones, ME, Lachish,S, Marvanek, S, Lazenby, B, Hocking, G, Wiersma, J & Hawkins CE, 2007, *EcoHealth*, Vol 4(3), pp 318-325

transmission appears to occur by biting, much of which happens during sexual encounters' and further speculates that this 'means that transmission is likely to be frequency-dependent with no threshold density for disease maintenance'.[76] It would appear from these modeling studies it had become accepted that stopping the spread of the devil disease was impossible. However, this again raises the question, referred to in previous studies, how does the disease spread in areas where there is severely reduced devil population numbers?

Stephen Pyecroft and colleagues, in the same issue of *EcoHealth,* claimed that cytogenetic analysis of tumour tissue, together with evidence from Major histocompatibility (MHC) gene analysis, provides 'significant evidence to confirm the tumour is a transmissible neoplasm'.[77] At the time, the 'evidence' on the MHC genes was unpublished.[78] A further article, in the same issue, by Professor Woods and colleagues on the immune system of the Tasmanian devil claims there is evidence that the devil has a competent immune system and 'the most likely explanation for devil-to-devil transmission of DFTD is that the tumor is not recognized by the devil as "non-self" because of the limited genetic diversity.'[79] It concluded that '[w]ith its consistent morphology and relatively stable genome, this tumor would provide a reasonable target for a vaccine approach, provided the immune system can be coaxed into recognizing the tumor as "non-self"'.[80] Also included in the issue was an Editorial by Andy Dobson

[76] ibid, p 318
[77] Pyecroft, SB, Pearse, AM, Loh R, Swift, K, Belov, K, Fox, N, Noonan, E, Hayes, D, Hyatt, A, Wang, L, Boyle, D, Church, J, Middleton D & Moore, R, 2007, Towards a Case Definition for Devil Facial Tumour Disease: What is it? *EcoHealth*, Vol 4(3), pp 346-351
[78] It would however be published by Siddle et al in *Immunogenetics* in the same month, August 2007.
[79] Woods GM, Kreiss A, Belov K, Siddle HV, Obendorf DL & Muller KH, 2007, The Immune Response of the Tasmanian Devil (*Sarcophilus harrisii*) and Devil Facial Tumour Disease, *EcoHealth*, Vol 4(3), pp 338-345, p 338
[80] ibid.

titled 'Sympathy for the Devil'[81] and an article by Peter Daszak and Aleksei Chmura titled 'Cover Essay: John Gould and a Devil's Despair'.[82]

In August 2007 PhD students Hannah Siddle, Claire Sanderson and their supervisor Katherine Belov published in *Immunogenetics* the results of the first genetic library for the Tasmanian devil.[83] They claimed that the 'MHC genes described here are …an important first step for studying MHC diversity and immune response in the devil'.[84] This equivocal statement is the source, at the time unpublished, referred to above by Pyecroft et al in *EcoHealth,* claiming confirmation of the transmissibility of the DFTD tumour.

In October 2007 Siddle and Belov together with the DFTD research scientists published an article in *PNAS* on the MHC genes in the Tasmanian devil.[85] They noted that '[t]he most common mechanism of immune evasion by tumors is down-regulation of classical cell surface MHC molecules', which is the case for CTVT but not for the devil cancer.[86] They claimed a lack of MHC diversity, verified by genotyping, provided a 'conclusive link between a loss of MHC diversity and spread of a disease'.[87] They further claimed '[h]ere we provide conclusive multilocus genetic evidence for the allograft theory of DFTD transmission, confirming that this disease is a clonal rogue cell line'.[88] This

---

[81] Dobson AP, 2007, Sympathy for the Devil, *EcoHealth* Vol 4(3), pp 241-243
[82] Daszak P & Chmura A, 2007, Cover Essay: John Gould and a Devil's Despair, *EcoHealth,* Vol 4(3), pp 367-368
[83] Siddle HV, Sanderson C & Belov K, 2007, Characterization of major histocompatibility complex class I and class II genes from the Tasmanian devil, *Immunogenetics* Vol 59, pp 753-760
[84] ibid, p 753
[85] Siddle, HV, Kreiss A, Eldridge MDB, Noonan, E, Clarke, CJ, Pyecroft, S, Woods GM & Belov K, 2007, Transmission of a fatal clonal tumor by biting occurs due to depleted MHC diversity in a threatened carnivorous marsupial, *PNAS,* Vol 104(41), 16221-16226
[86] ibid, p 16221
[87] ibid.
[88] Siddle, HV, Kreiss A, Eldridge MDB, Noonan, E, Clarke, CJ, Pyecroft, S, Woods GM & Belov K, 2007, Transmission of a fatal clonal tumor by biting occurs due to depleted MHC diversity in a threatened carnivorous marsupial, *PNAS,* Vol 104(41), 16221-16226, p 16224

finding was to be later found to be false, when it was revealed that tissue grafts between devils had been rejected, indicating that the lack of genetic diversity in the devils MHC was not responsible for the transmission of the cancer.[89] Subsequently, in an interview with Rachel Carbonell on the Australian Broadcasting Commission's *The World Today*, Kathy Belov stated 'I suppose all of science is about testing hypotheses. In this case, it turns out our hypothesis wasn't correct'.[90] In the introduction to the program Eleanor Hall stated:

> [s]cientists investigating the deadly facial tumours decimating the Tasmanian devil population have just disproved their original theory and are now in a race against time to identify the cause of the cancer.

Also in 2007 Shelly Lachish, Jones and McCallum published a study on the impact of DFTD on devil population growth.[91] From their observations they found strong evidence that the rate of DFTD infection in the target population was increasing and that the epidemic was not declining. Meanwhile, they also state, '[a]t this site, DFTD prevalence remains high (33%) despite a reduction in population size from approximately 7 individuals per square kilometer to just 0.18 individuals.[92] They conclude given this decline in population numbers 'local population extinction seems likely'.[93]

---

[89] All eastern devils tested in 'in vivo' allograft experiments – total of 8 animals – all showed host-graft or graft-host rejection. 22 March 2009 Kreiss & Woods laboratory notes (Appendix C).
[90] Carbonell R, 2012, Tasmanian devil facial tumour theory debunked, Australian Broadcasting Commission, The World Today with Eleanor Hall. Available at: http://www.abc.net.au/worldtoday/content/2012/s3523185.htm last accessed 9 December 2012
[91] Lachish, S, Jones, M & McCallum H, 2007, The impact of disease on the survival and population growth rate of the Tasmanian devil, *Journal of Animal Ecology*, Vol 76, pp 926-936
[92] ibid, p 935
[93] ibid.

Authors independent of the DPIPWE also published a paper titled 'Update on the devil facial tumour in Tasmania' in the *European Journal of Oncology* in 2007.[94] The authors were Neil McGlashan[95] from the School of Geography, University of Tasmania (UTAS), David Obendorf, a veterinary pathologist, and Jack S Harington,[96] a cancer researcher. It reported on the forum of research scientists held in Hobart in February 2007 revealing that transmission experiments to support the allograft cell transfer theory had been attempted but the results had not been published. Stephen Pyecroft from the DPIPWE Mt Pleasant laboratory presented an abstract to the Forum on his transmission trials which stated '[t]rial animals injected with cell lines and receiving surgical implants of tumour tissue developed actively developing cancers at the treatment sites, to a variable degree'.[97] No further studies have been undertaken.

In 2008 Obendorf and McGlashan published a paper, titled 'Research priorities in the Tasmanian devil facial tumour debate', in the *European Journal of Oncology* proposing that two aspects of the devil research were 'under-rated and under-funded' and called for further research into these areas.[98] The first was the possibility of immunogenic resistance to DFTD in a separate western devil population. The second, more importantly, sought an investigation into what is described as an 'all but neglected' area of research stating:

---

[94] McGlashan ND, Obendorf DL & Harington JS, 2007, Update on the devil facial tumour in Tasmania, *European Journal of Oncology,* Vol 12(2), pp 75-80

[95] Neil McGlashan was a former member of the staff of the Cancer Research Unit and a member of the International Geography Union's Commission for Medical Geography.

[96] Dr Jack Harington was a senior member of the Cancer Research Unit at the South African Institute for Medical Research Johannesburg South Africa (Source: Harington JS & McGlashan ND, 1976, Migrant Workers and Cancer Patterns in Southern Africa, *Journal of Southern African Studies*, Vol 3(1), pp 92-101

[97] Pyecroft SB, 2007, Transmission trials: Devil Facial Tumour Disease, Devil Facial Tumour Disease, *Senior Scientist's Scientific Forum*, 20-22 February 2007, University of Tasmania, Hobart

[98] Obendorf DL & McGlashan ND, 2008, Research priorities in the Tasmanian devil facial tumour debate, *European Journal of Oncology*, Vol 13(4), pp 229-238

> that the genesis and effective transmission of this disease was the fateful culmination in a cascade of anthropogenic land-use activities and can more specifically be linked to a toxin-related aetiology occurring in a wild, carrion-feeding marsupial…[99]

Both of these papers appear on the DPIPWE List of Publications as at July 2011. However, other papers by these authors, who raise the issue of competing hypotheses, do not appear. These include a paper published in 2005 by Harington and McGlashan titled 'The Tasmanian Devil Facial Tumour Disease (DFTD) – a problem unresolved' in *Annals of the Australasian College of Tropical Medicine.*[100] In it they noted,'[w]hilst no viral aetiology has yet been established, direct spread by biting and transfer of allograft cells is currently favoured speculation.'[101] They further suggested that '[b]ecause of the lesion's visual similarity with Kaposi's sarcoma in humans, a form of Devil AIDS (DAIDS) or Devil HIV (DHIV) also merits consideration.'[102]

A further two papers were published in 2006 which are not cited in the DPIPWE List. The first, a letter by McGlashan, Obendorf & Harington titled 'Researching the Tasmanian devil facial tumour', drew attention to the need to consider '[t]he capacity of highly toxic new-generation agents to be mutagenic, genotoxic or oncogenic needs consideration'. The second, by McGlashan, Obendorf and Harington, was again published in the *European Journal of Oncology* and titled 'Aspects of the fatal malignant disease among the Tasmanian devil population'. In this paper the authors again raise the possibility that, as the Tasmanian devil is the top carnivore at the head of a native herbivorous marsupial food chain, the 'role of bioaccumulated persistent

---

[99] ibid, p 230
[100] Harington JS & McGlashan ND, 2005, The Tasmanian Devil Facial Tumour Disease (DFTD) – a problem unresolved. *Annals of the Australasian College of Tropical Medicine,* Vol 6(2), p 34
[101] Harington & McGlashan, 2005, p 34
[102] Harington JS & McGlashan ND, 2005, The Tasmanian Devil Facial Tumour Disease (DFTD) – a problem unresolved. *Annals of the Australasian College of Tropical Medicine,* Vol 6(2), p 34

organic pollutants and possibly genotoxic chemicals requires investigation as do conventional infectious pathogens such as exogenous and endogenous viruses…'.[103]

The papers published prior to 2007 were also not cited in the *EcoHealth* issue in 2007 mentioned above. According to Obendorf the then DFTD Manager, Alistair Scott asked to see a draft of the first devil paper before submission to the journal as the Tasmanian government and its scientists had 'a right to contact the journal's editor and get the opportunity to referee or veto this paper'.[104]

Under pressure in 2008 a paper was published on a preliminary pilot study into the role of chemicals in the devil cancer. Walter Vetter and his colleague, Roland von der Recke both from the University of Hohenheim in Stuttgart, Germany, Robert Symons from the Australian National Measurement Institute and Stephen Pyecroft from the DPIPWE published a paper in *Rapid Communications in Mass Spectrometry*.[105] This paper reported findings of residues of chemicals PBBs (flame retardants) and PBDEs in devil tissue. A full analysis of this paper and the lack of studies following these initial findings is given in Chapter 5.

In 2008 a number of studies were also published on the Tasmanian devil immune system. It has been shown elsewhere that CTVT cancer down regulates the dogs' immune system in order to establish in the new host; this is not the case in the devil

[103] McGlashan ND, Obendorf DL & Harington JS, 2006, Aspects of the fatal malignant disease among the Tasmanian devil population (*Sarcophilus laniarius*), *European Journal of Oncology*, Vol 11(2), pp 95-102, pp 95-96
[104] Email from Obendorf to me (Appendix D).
[105] Vetter W, Recke R, Symons R & Pyecroft S, 2008, Determination of polybrominated biphenyls in Tasmanian devils (*Sarcophilus harrisii*) by gas chromatography coupled to electron capture negative ion tandem mass spectrometry or electron ionization high-resolution mass spectrometry, *Rapid Communications in Mass Spectrometry*, Vol 22, pp 4165-4170

cancer. The devil immune system was said to be functionally competent and a lack of genetic diversity, particularly in the MHC genes, was proposed as the reason the devil cancer established in its new host.[106] As noted above this hypothesis has since been found to be false, however, an analysis of the studies undertaken in relation to the immune system and the MHC is the focus in the next chapter.

Other papers published in 2008 related to the results of studies undertaken on devil populations. A paper published in *PNAS* by the Tasmanian devil researchers proposed that DFTD may have caused changes in reproductive behavior in female devils, resulting in breeding at an earlier age.[107] Rodrigo Hamede, then a PhD student, and his supervisors, McCallum and Jones, published a paper suggesting transmission is likely to be frequency dependent, as the mating season appeared to be the key period for transmission.[108] In 2009 Hamede et al suggested that there was limited potential for control of the cancer, as devils were highly connected thus permitting spread of the disease from any single infected devil.[109] However, in a more recent paper by Hamede et al published in 2012 suggests that there is not a super spreader devil but that some devils are super receivers.[110] The idea is that the more aggressive devils do not get bitten but bite the tumours of less aggressive devils.

---

[106] Kreiss A, Fox N, Bergfeld J, Quinn SJ, Pyecroft S & Woods GM, 2008, Assessment of cellular immune responses of healthy and diseased Tasmanian devils (*Sarcophilus harrisii*), *Developmental and Comparative Immunology,* Vol 32, pp 544-553

[107] Jones ME, Cockburn A, Hamede R, Hawkins C, Hesterman H, Lachish S, Mann D, McCallum H and Pemberton D, 2008, Life-history change in disease-ravaged Tasmanian devil populations, *PNAS*, Vol 105, pp 10023-10027

[108] Hamede RK, McCallum H & Jones M, 2008, Seasonal, demographic and density-related patterns of contact between Tasmanian devils (*Sarcophilus harrissi*): Implications for transmission of devil facial tumour disease, *Austral Ecology*, Vol 33, pp 614-622

[109] Hamede RK, Bashford J, McCallum H & Jones M, 2009, Contact networks in a wild Tasmanian devil (*Sarcophilus harrisii*) population: using social network analysis to reveal seasonal variability in social behavior and its implications for transmission of devil facial tumour disease, *Ecology Letters*, Vol 12, pp 1147-1157

[110] Hamede RK, McCallum H, & Jones M, 2013, Biting injuries and transmission of Tasmanian facial tumour disease, *Journal of Animal Ecology*, Vol 82(1), pp 182-190

In 2009 many of the studies published related to conservation and population dynamics but Elizabeth Murchison published a paper on a comparison between the dog and the devil cancers in *Oncogene*.[111] This paper is discussed more fully in section 3.6 below. A study published in 2010 suggested that the original DFTD cell was a Schwann cell based on genetic studies and also claimed confirmation 'that DFTD is a monophyletic[112] clonally transmissible tumor'.[113] In 2011 a study was published on the development of a mouse model for the study of DFTD.[114] Two recent studies on the different strains of DFTD published in 2012 are included in the discussion below.

3.5.4 Summary of comparison between CTVT and DFTD

Many of the studies documented above concern the conservation and population dynamics of the Tasmanian devil but these are not considered within the allograft research program. Hence, only those relative to a comparison between CTVT and DFTD are summarised in Table 3:1 below.

Table 3:1 Comparison between CTVT and DFTD

CTVT	DFTD
Sexually transmitted	Transmission via biting
Spread due to 'popular sire effect' of dogs ('superspreader')	Devils are 'super receivers' – transmission through biting the tumours of less aggressive devils or any single infected devil
Infiltration of lymphocytes, plasma cells and macrophages	No infiltration of lymphocytes, plasma cells and macrophages
Do not express type I and II MHC antigens	No studies of DFTD cells for antigen markers

[111] Murchison E, 2009, Clonally transmissible cancers in dogs and Tasmanian devils, *Oncogene*, Vol 29, pp S19-S30

[112] Monophyletic according to the Merriam-Webster Dictionary means – developed from a single common ancestral form. Available at: http://www.merriam-webster.com/dictionary/monophyletic last accessed 10 December 2012

[113] Murchison EP, Tovar C, Hsu, A, Bender HS, Kheradpour P, Rebbeck CA, Obendorf D, Conlan C, Bahlo M, Blizzard CA, Pyecroft S, Kreiss A, Kellis M, Stark A, Harkins TT, Graves JAM, Woods GM, Hannon GJ & Papenfuss AT, 2010, *Science*, Vol 327, pp 84-87, p 84

[114] Kreiss A, Tovar C, Obendorf DL, Dun K & Woods GM, 2011, A Murine Xenograft Model for a Transmissible Cancer in Tasmanian Devils, *Veterinary Pathology*, Vol 48(2), pp 475-481

Commonly occurs in 2-5 year old dogs – sexually active	Occurs in adult devils; rare in devils under 2 years of age
Regression leads to tumour immunity which prevents successive occurrences	Fatal in all cases, no immunity
58-59 chromosomes with 13-17 metacentric and 42 acrocentric - stable	Chromosomes unstable number varies across 13 different strains
Possibly viral	No virus found
Benign – except in immunocompromised animals	Malignant – metastatic
Identified the molecular fingerprint of the cancer – insertion near c-myc was found in all tumours, found in a different location to normal canine DNA and used as a diagnostic marker for CTVT.	No studies for genetic marker
Transmission studies by Novinski 1876	Transmission studies abandoned following variable results.
Competing hypothesis – none.	Competing hypothesis – chemical aetiology.

As is shown in the comparison in Table 3:1 above, the research programs followed a similar pathway but with somewhat different results. Most significant is the lack of transmission studies in DFTD and the existence of a competing hypothesis, that chemicals in the environment may have contributed to the cancer, which has also not been adequately investigated.

3.6 Review of comparisons between CTVT and DFTD by Murchison

Elizabeth Murchison, a researcher at the Cancer Genome Project, Wellcome Trust Sanger Institute in Cambridge, undertook a review of CTVT and DFTD by contrasting and comparing the two cancers with a focus on biology.[115] Murchison produced a table, reproduced in Table 3:2 below, of her comparison between the two allografts.

[115] Murchison EP, 2009, Clonally transmissible cancers in dogs and Tasmanian devils, *Oncogene,* Vol 27, pp S19-S30,

Table 3:2 Comparison between DFTD and CTVT[116]

	DFTD	CTVT
Host species	Tasmanian devil	Dog
Species of origin	Tasmanian devil	Wolf or dog
Distribution	Mainland Tasmania (excluding northwest)	Worldwide
Time of origin	15-20 years ago	7800-78000 years ago
Body location	Face, oral cavity	External genitalia
Mode of transfer	Biting	Coitus
Histogenesis	Neuroendocrine	Myeloid
Metastasis	Common	Common in immune-compromised animals
Spontaneous regression	0%	Common in experimentally inoculated CTVT, prevalence in naturally occurring CTVT unknown
Mortality	100%, within 6-12 months after appearance of symptoms	Rare in experimentally inoculated CTVT, prevalence in naturally occurring untreated CTVT unknown
Treatment	None	Chemotherapy, radiation therapy
Effect on host population	Host population decline/possible imminent extinction	Probably little effect

It appears from Murchison's comparison that the two cancers differ in all listed aspects. It is noted that metastasis, although common in devils is uncommon in dogs, except in strays and pups that are immunocompromised. Murchison cites both the Pyecroft et al paper published in 2007 and the Obendorf and McGlashan paper published in 2008 as evidence of experimental transmission of DFTD as an allograft.[117]

As discussed above, the transmission experiments undertaken by Pyecroft were incomplete with ambiguous results and have to date have not been published in full. Meanwhile, the evidence supporting the hypothesis that DFTD is transmissible in the Obendorf and McGlashan paper was the result of a study at Androo Kelly's Trowunna

[116] ibid, p S26
[117] ibid, S20

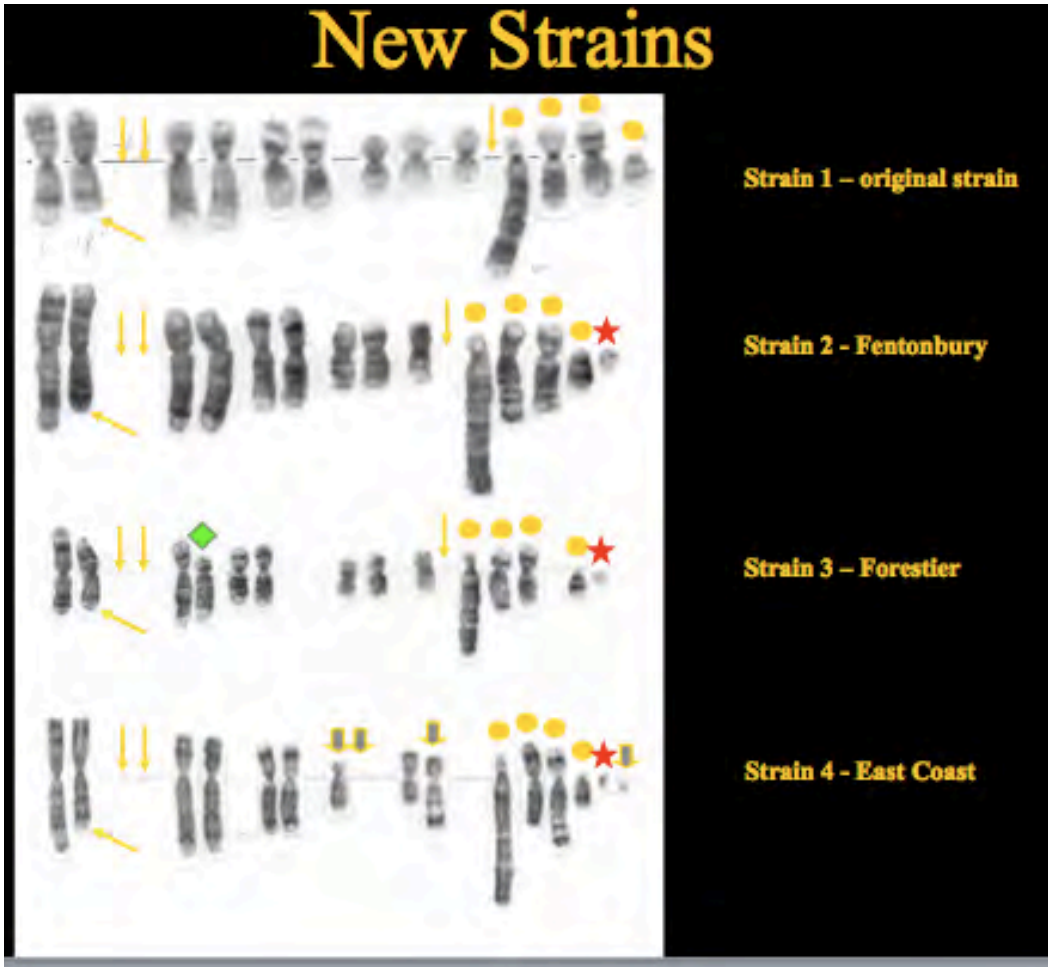
Wildlife Park. This evidence is speculative at best, as it is based mainly on anecdotal evidence.[118] The speculation is that a devil escaped from the Park, had an encounter with a DFTD-affected devil (when it was bitten) and it subsequently returned to the Park. The devil developed DFTD and passed it onto another devil in the Park. The Trowunna Wildlife Park has since had many more devils with DFTD. In an interview with Androo Kelly he assured me his perimeter security fence had been strengthened since the initial DFTD case and it was unlikely that the incident had been repeated. Trowunna Wildlife Park is however in close proximity to plantation forests, which are aerially sprayed on a regular basis with chemicals to prevent predation by pests. The Liffy Creek from which water is sourced for the Park has also been contaminated with chemicals used in plantation forestry. A full analysis of the potential for chemicals to be a contributing factor in DFTD, as mentioned above, is given in Chapter 5.

3.7 Different strains of DFTD

Pearse and Swift had continued to observe the DFTD tumour cells at the DPIPWE Mt Pleasant laboratory and continued to observe increasing instability in the chromosomes of the DFTD tumour cells.[119] They also observed that devil tumour cells from particular locations on the island shared the same chromosomal abnormalities. These sets of chromosomes (strains 2–4) were different from the original DFTD chromosomes (strain 1) published in the *Nature* article as shown in the illustrations Figures 3:3 and 3:4 below.

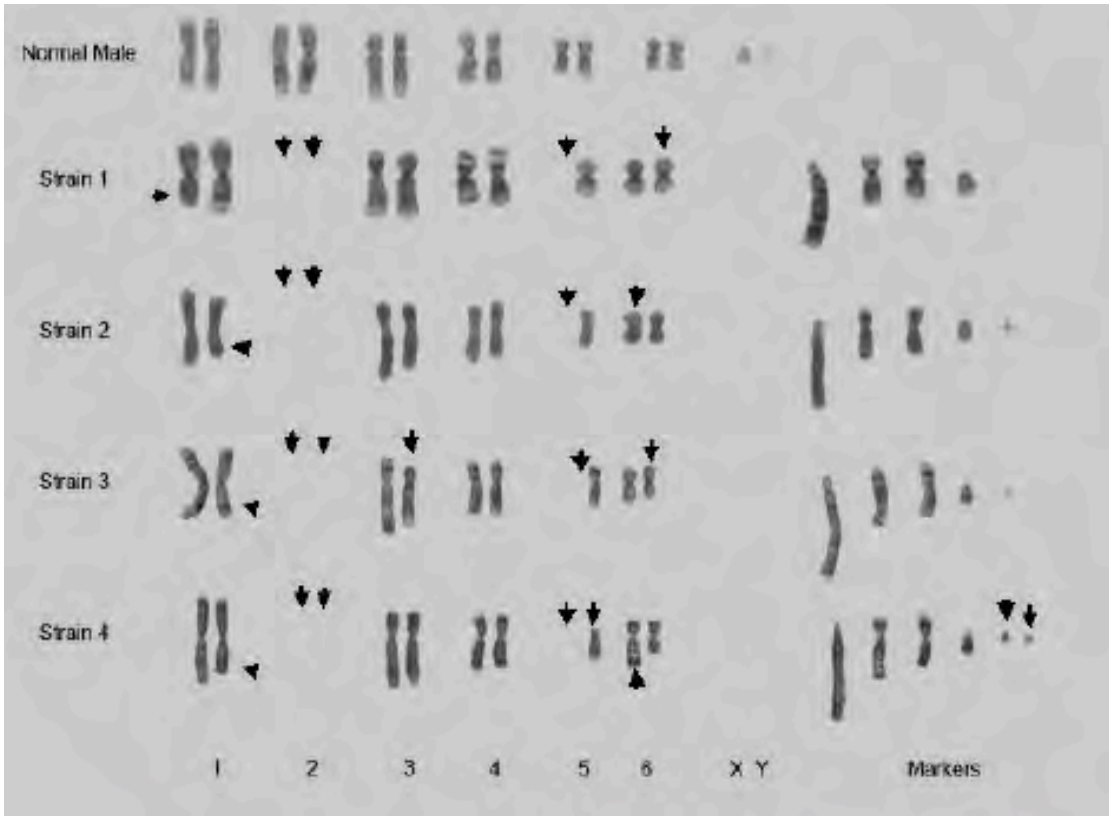
[118] Obendorf DL & McGlashan ND, 2008, Research priorities in the Tasmanian devil facial tumour debate, *European Journal of Oncology*, Vol 13(4), pp 229-238, p 231
[119] Personal communication.

Figure 3:3 Different Strains of DFTD from different locations[120]



[120] Pearse, 2011, Presentation to DPIPWE devil research team.

Figure 3:4 Devil Chromosomes [121]



The images in Figure 3:4 above are accompanied by the following explanation. The figure

> …compares a normal male karyotype with the karyotypes of 4 DFTD strains. Anne-Maree Pearse (DPIPWE) has characterized at least 9 transmissible DFTD strains (A strain is defined as a karyotype of consistent chromosomal constituents that has been identified in multiple geographically proximate individuals and is therefore transmissible). Some interesting features of DFTD strains are emerging. Firstly, primary tumours appear highly stable, with little variation in chromosome numbers and conformations. Metastases are more variable karyotypically and contain variants exhibiting aneuploidy and aneusomy. These variants are not transmitted. There is also evidence that some strains are more successful than others – e.g. strain 2 has overtaken strain 1 as the most prominent strain. Tetraploid strain 1 has become more common than diploid strain 1. Some strains appear to have died out – e.g. strain 4 has only been seen in 5 individuals on the east coast. Tumour evolution also occurs in culture.[122]

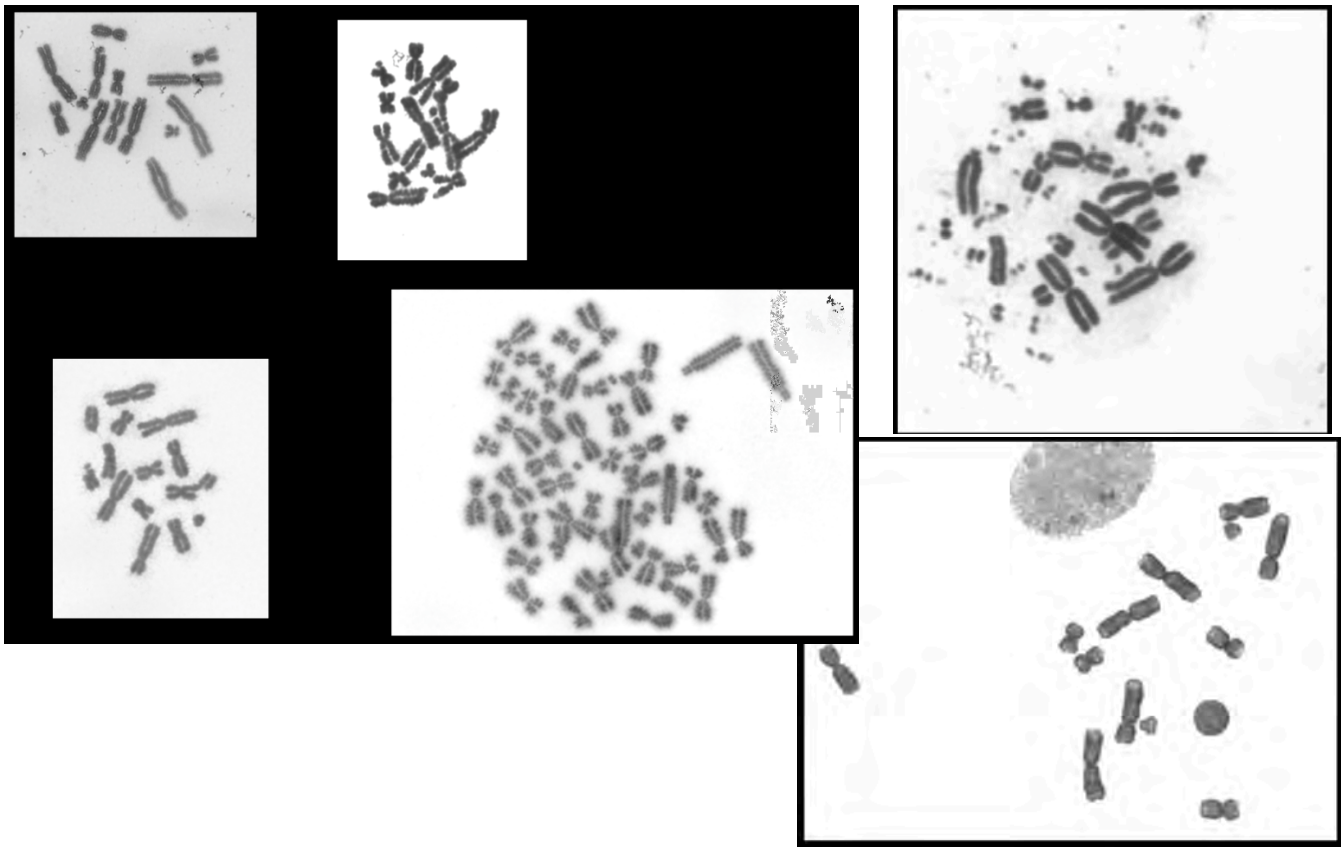
[121] Pyecroft, 2010, Internal DPIPWE Report
[122] Pyecroft, 2010, Internal DPIPWE Report, p 43

The claim for transmission of DFTD appears to have narrowed to the highly stable primary tumours with variants unable to be transmitted. It also appears to be a deviation from CTVT where it was found that all tumours including primary, metastatic and cell cultures are similar as noted above. These observations also vary from the original claim made by Pearse and Swift in *Nature* –

> …these anomalies were the same in the facial tumours of every animal (*n*=11). These rearrangements are complex, but no intermediate stages were found between normal and tumour chromosomes, even in small primary cancers.[123]

The observations were not limited to those above but also included extreme instability as shown in the three images in Figure 3:5 below.

Figure 3:5 Images of DFTD chromosomes[124]



[123] Pearse & Swift, 2006, p S49
[124] Personal communication with Pearse, 2011, Presentation to DPIPWE devil research team.

Pearse proposed that the new theory of epigenetics, genetic changes due to environmental factors, may explain the different abnormalities in the different locations in the DFTD cells.[125] Pearse prepared a paper detailing her observations but she told me her original article was rejected by *Cell.*[126] At the time of our meeting, she was in the process of re-writing it for submission to another journal. However, since then two articles with Pearse as a co-author have since been published.

The first was published in *Cell* and the second in *PLoS Genetics,* which appear to cover Pearse's different strains. The Murchison et al article published in *Cell* in 2012 claims '[p]revious studies have indicated that the cancer is derived from the cells of one devil (the DFTD founder) and has subsequently spread through the devil population as a clone' citing Pearse's and Swift's proposed hypothesis in *Nature* in 2006.[127] In the discussion they state '[o]ur analysis of the genomes of two geographically distant DFTD subclones has indicated that DFTD is continuing to acquire new variations in its karyotype, genomic copy number and DNA sequence'.[128] The second, Deakin et al's

[125] Personal communication on 18 March 2010 in Launceston

[126] Animal Health Laboratories/Diagnostic Services Branch Deliverables to the STTDP, 2010, Notes on publications – Anne-Maree Pearse, Katherine Belov, Hannah V. Siddle, Kate Swift, Erin Noonan, Stephen Pyecroft and Mark D.B. Eldridge, Chromosome evoluation in Tasmanian Devil Facial Tumour Disease: a contagious cancer. Status Submitted, Rejected by Cell To be submitted to "Cancer Genetics and Cytogenetics". p 22

[127] Murchison EP, Schulz-Trieglaff OB, Ning Z, Alexandrov LD, Bauer MJ, Fu B, Hims M, Ding Z, Ivakhno S, Stewart C, Ng LB, Wong W, Aken B, White S, Alsop A, Becq J, Bignell GR, Cheetham RK, Cheng W, Connor TR, Cox AJ, Feng ZP, Gu Y, Crocock Rj, Harris SR, Khrebtukova I, Kingsbury Z, Kowarsky M, Dreiss A, Luo S, Marshall J, McBride, DJ, Murray L, Pearse AM, Raine K, Rasolonjatovo I, Shaw R, Tedder P, Tregidgo C, Vileila AJ, Wedge DC, Woods GM, Gormley N, Humphray S, Schroth G, Smith G, Hall, K, Searle SMJ, Carter NP, Papenfuss AT, Futreal PA, Campbell PJ, Yang F, Bentley DR, Evers DJ & Stratton MR, 2012, Genome Sequencing and Analysis of the Tasmanian Devil and Its Transmissible Cancer, *Cell,* Vol 148, pp 780-791, p 782

[128] Murchison EP, Schulz-Trieglaff OB, Ning Z, Alexandrov LD, Bauer MJ, Fu B, Hims M, Ding Z, Ivakhno S, Stewart C, Ng LB, Wong W, Aken B, White S, Alsop A, Becq J, Bignell GR, Cheetham RK, Cheng W, Connor TR, Cox AJ, Feng ZP, Gu Y, Crocock Rj, Harris SR, Khrebtukova I, Kingsbury Z, Kowarsky M, Dreiss A, Luo S, Marshall J, McBride, DJ, Murray L, Pearse AM, Raine K, Rasolonjatovo I, Shaw R, Tedder P, Tregidgo C, Vileila AJ, Wedge DC, Woods GM, Gormley N, Humphray S, Schroth G, Smith G, Hall, K, Searle SMJ, Carter NP, Papenfuss AT, Futreal PA, Campbell PJ, Yang F, Bentley DR, Evers DJ & Stratton MR, 2012, Genome Sequencing and Analysis of the Tasmanian Devil and Its Transmissible Cancer, *Cell,* Vol 148, pp 780-791, p 787

2012 paper in *PLoS Genetics* again cites the Obendorf and McGlashan's speculation from the Trowunna Wildlife Park claiming '[t]his observed pattern of intra-tumour chromosome variability is consistent with observations that the tumour is passed from animal to animal by biting, during which many clumps of tumour cells are dislodged from the mouth of the affected animal'. [129] Deakin et al conclude '[w]e provide further confirmation of the clonal transmission of DFTD and tentatively identify the sentinel animal as a female devil'.[130] The editor of *PLoS Genetics* is Stephen J. O'Brien and the study was funded by the Australian Research Council, the Dr Eric Guiler Tasmanian Devil Research Grants and DPIPWE. Both articles claim that although it appears through G-banding that the chromosomes are unstable, chromosome painting and gene mapping show that the chromosomes remain stable. However, in undertaking these studies they did not identify, as Murgia et al did in CTVT, the marker gene to confirm DFTD like CTVT is transmissible.

Pearse's initial observation conflicts with the now recognized different strains, as shown here in the *Conservation Magazine*:

> When she stained the nuclei of tumour cells from several different devils, she saw that the chromosomes were abnormal. The "leg and arms" of the chromosomes looked as if they had been cut off and glued back together in arbitrary places. This was not too surprising; lots of tumor cells have rearrangements in their chromosomes. But what was surprising was that all the tumor cells, whether from one devil or another, had exactly the same rearrangements – the bizarre rearrangements were identical.[131]

---

[129] Deakin JE, Bender HS, Pearse AM, Rens W, O'Brien PCM, Ferguson-Smith MA, Cheng Y, Morris K, Taylor R, Stuart A, Belov, K, Amemiya CT, Murchison, EP, Papenfuss AT & Graves JAM, 2012, *PLoS Genetics*, Vol 8(2), pp 1-16, p 13

[130] ibid.

[131] Mills C, 2008, Cancer on a Whole Species, The gruesome disease ravaging Tasmanian devils is unlike anything we've seen before, *Conservation Magazine*, Vol 9(1). Available at: http://conservationmagazine.org/2008/07/cancer-on-a-whole-species/ last accessed 2 October 2013

The research into the different strains of cancer may be important but it has not provided proof that the cancer is contagious; in fact if anything, it has weakened the evidence. Instability is the hallmark of cancer.[132]

3.8 Conclusion

The selection of studies has steered the research along the genetic pathway, successfully avoiding relevant research such as transmission and toxicology studies. It is also apparent from the studies in the comparison above, that definitive identification of specific marker genes, as identified in the dog cancer, have not been undertaken. Practical reasons, such as lack of theoretical concepts or technology, do not explain why this research has not been done, hence political reasons should be considered. It would appear that ignorance, in the form of negative non-knowledge, may be a contributing factor in the continuing demise of the Tasmanian devil.

The CTVT research program has followed a scientific pathway that has led to discoveries about the cancer and confirmed the probability that it is transmissible although there are still some skeptics who think a virus may be involved. The DFTD research program appears to have followed the existing CTVT allograft program closely. Firstly, Pearse and Swift likened DFTD to CTVT in their original article because of the identical chromosomal rearrangements unique to the cancers and to a particular change in centromeres[133]. Secondly, both studies have researched the MHC genes in order to better understand the immune system role in the cancer. It was found that CTVT down-

[132] Pardee AB & Stein GS, (Eds), 2009, *The Biology and Treatment of Cancer: Understanding Cancer*, John Wiley & Sons, Hoboken, New Jersey.
[133] Centromeres are the organizing center in cells during division. Centrosome defects have been implicated in disease and tumour progression. Lingle WL & Salisbury JL, 1999, Alterned centrosome structure is associated with abnormal mitoses in human breast tumors, *American Journal of Pathology,* 155(6), pp 1941-51

regulates the dog's immune system, with the dog eventually developing resistance and the cancer going into remission, on rare occasions it becoming malignant. This does not occur in the devils. A lack of diversity in the devil MHC was proposed as the reason DFTD could establish in a new host, but this has now been revealed to be incorrect.

There are however studies undertaken by the DFTD researchers that were not undertaken by CTVT researchers. For example, studies to understand the apparent instability in the devil tumour cells were done. DFTD researchers also undertook studies into the spread of the devil cancer as shown in Table 3:3 below, which reveal nothing consistent or conclusive.

Table 3:3 Studies into spread of DFTD

Year	Authors	Findings
2006	Hawkins et al[134]	No evidence for density dependence as prevalence high, even with 80% decline in population
2006	McCallum & Jones[135]	Mating key to transmission
2007	McCallum et al[136]	Transmission during sexual encounters – frequency dependent
2009	Hamede, et al[137]	Transmission – frequency dependent
2009	Hamede, et al[138]	Devils highly connected spread likely results from single infected devil
2012	Hamede, et al[139]	No 'super spreader' but a 'super receiver' due to aggressive behaviour

[134] Hawkins CE, Baars C, Hesterman H, Hocking GJ, Jones ME, Lazenby B, Mann D, Mooney N, Pemberton D, Pyecroft S, Restani M & Wiersma J, 2006, Emerging disease and population decline of an island endemic, *Biological Conservation*, Vol 131, pp 307-324, p 319

[135] McCallum H & Jones ME, 2006, To lose both would look like carelessness: Tasmanian Devil Facial Tumour Disease, *PLoS Biology*, Vol 4(10), 1671-1674, p 1674

[136] McCallum, H, Tompkins, DM, Jones, ME, Lachish,S, Marvanek, S, Lazenby, B, Hocking, G, Wiersma, J & Hawkins CE, 2007, *EcoHealth*, Vol 4(3), pp 318-325, p 318

[137] Hamede RK, McCallum H & Jones M, 2008, Seasonal, demographic and density-related patterns of contact between Tasmanian devils (*Sarcophilus harrissi*): Implications for transmission of devil facial tumour disease, *Austral Ecology*, Vol 33, pp 614-622

[138] Hamede RK, Bashford J, McCallum H & Jones M, 2009, Contact networks in a wild Tasmanian devil (*Sarcophilus harrisii*) population: using social network analysis to reveal seasonal variability in social behavior and its implications for transmission of devil facial tumour disease, *Ecology Letters*, Vol 12, pp 1147-1157

[139] Hamede RK, McCallum H, & Jones M, 2013, Biting injuries and transmission of Tasmanian facial tumour disease, *Journal of Animal Ecology*, Vol 82(1), pp 182-190

Some of the studies undertaken in CTVT, such as the transmission studies by Novinski in 1876, were abandoned in DFTD after variable results. The identification of a set of genes that occur in all dog tumours across a number of countries has not been identified in the devil tumours. There has been a lot of expensive and highly technical research into DFTD genetics but the basic studies, to prove the cancer is transmissible, still remain undone.

Further, it would appear that some articles published in support of the allograft theory of DFTD, rather than being a genuine representation of the scientific experiments undertaken to confirm that the devil cancer is indeed transmissible, falsely assume or imply the theory has already been proved. This is particularly evident in the issue of *EcoHealth* containing several articles relating to DFTD. Whilst there is nothing unusual in focusing on a particular topic for an issue, all articles reference the Pearse and Swift *Nature* article as confirmation of the allograft theory of DFTD, which is clearly not the case. There are also other assumptions made confirming the transmission of the devil cancer that are premature and claims not supported by evidence as will be shown in the following chapters.

In both research programs today's sophisticated knowledge creation relies on methods and the latest, often prohibitively expensive technology, resulting in those with the most funds having the most access; the study of genetics is one such area. In the case of CTVT these studies have been independently explored and supported. In contrast the Tasmanian Government through the DPIPWE, which operates the Mt Pleasant laboratory in Launceston, has controlled the DFTD studies. At the laboratory devil

samples are prepared and experiments are undertaken with access to expensive genetic testing equipment. The Tasmanian devil, listed as 'endangered' under the *EPBC Act 1999,* is a protected species making it illegal to 'take' without specific authority. This arrangement, whereby one government department has control over the research, the funding and the endangered species specimens, constitutes a capture of the scientific research. The outcome has been the effective silencing of any competing alternative hypotheses. Consequently, the impression is given that the dominant research community, with access to sophisticated equipment, is pursuing the only genuine science.[140]

The CTVT research program has a long history of independent studies directed to solving and understanding the dog transmissible tumour. By using this research program as the benchmark for the DFTD research program it is evident that not all the relevant and important studies have been undertaken. The transmission studies undertaken as early as 1876 to confirm that the dog cancer was transmissible have still not been completed for the devil cancer. Genetic studies identifying the mutated genes in CTVT also remain undone in the devil research. These are not the only studies to have been left undone, research into an alternative hypothesis that chemicals used in plantation forests may have played a role in the devil cancer, was abandoned following a pilot study. Before analyzing this aspect of the research the next chapter explores the DFTD research selected for study that has steered the research priorities in particular directions. These include a search for why, when the devil immune system is claimed to be functionally competent, the cancer can establish in a new host.

---

[140] Hess, DJ, 2007, *Alternative Pathways in Science and Industry, Activism, Innovation, and the Environment in an Era of Globalization*, The MIT Press, Cambridge, Massachusetts, p 24

Chapter 4 – The science selected for study

4.1 Introduction

The Tasmanian devil research program is committed to the allograft theory that argues the cancer is transmissible. Any competing hypothesis for the disease is ignored (I address this in the next chapter) and adjustments are made for anomalies. Theories are seldom abandoned because of anomalies. According to Hess adjustments are made to accommodate the data, or as Lakatos suggests, rather than discard a useful theory because of apparently contradictory evidence, attempts are made to harmonise the findings. In this chapter I will interrogate an apparent anomaly, that the devils' immune system is competent, to discover if this anomaly to the allograft theory has been problematic for the research program. Evidence shows that environmental toxins, including pesticides, can suppress the normal responses of the immune system to invading viruses, bacteria, parasites and tumours, resulting in immune suppression.[1] Closely aligned with the immune studies is the search to develop a vaccine for the devil cancer, which also forms part of the analysis in this chapter.

In most diseases, including cancer, it is the failure of the body's defences to recognize and eliminate foreign invaders that allows a disease to progress.[2] Many chemicals used in the environment have damaging effects on the body's immune system compromising

[1] Repetto R & Baliga S, 1996, *Pesticides and the Immune System: The public health risks*, World Resources Institute, Washington, DC. Available at: http://www.wri.org/publication/content/8344 last accessed 30 June 2013

[2] United States Department of Health and Human Services National Institutes of Health, 2007, *Understanding the Immune System, How it Works*. Available at: http://www.niaid.nih.gov/topics/immunesystem/documents/theimmunesystem.pdf last assessed 13 December 2012

its ability to ward off diseases.[3] Hence, it is becoming increasingly evident that a lack of immune system competence is a high risk for malignancy in cancer.[4] This view is supported by the high risk of malignancy in patients receiving immunosuppressive medications, such as organ transplant patients[5], and in patients with autoimmune diseases like AIDS with underlying immune system abnormalities.[6] Alternatively, cancer cells can proliferate by effectively avoiding the surveillance of the immune system, as proposed in the dog transmissible tumour CTVT. It is proposed that CTVT in dogs has evolved to avoid immune surveillance by down-modulating major-histocompatability (MHC) complex antigen expression.[7]

There has been no suggestion that the devil immune system is artificially suppressed by chemicals, nor has it been found that the cancer has evolved, like the dog cancer, to avoid immune system surveillance. The question then is how do the devil cancer cells establish in a new host devil? The DFTD researchers proposed the devils' lack of genetic diversity, particularly in the MHC genes, was the most likely reason that devils succumb to DFTD. The researchers suggested a similar lack of genetic diversity in a population of African cheetah as a precedent for this proposal. An analysis of the cheetah precedent is included in this chapter. Before continuing however the following

[3] Vos JG & Dean JH, 1990, Methods for Assessing the Effects of Chemicals on the Immune System in P Bourdeau, E Somers, GM Richardson & JR Hickman, (Eds), 1990, *Short-term Toxicity Tests for Non-gegenotoxic Effects, SCOPE 41, IPCS Joint Symposia 8,* Wiley, Chichester

[4] Whiteside TL, 2005, Immune suppression in cancer: Effects on immune cells, mechanisms and future therapeutic intervention, *Seminars in Cancer Biology,* pp 1-13. Available at: http://www.aimath.org/WWN/tumorimmune/WhitesideImmuneSuppression.pdf last accessed 3 October 2013

[5] Gutierrez-Dalmau A & Campistol JM, 2007, Immunosuppressive therapy and malignancy in organ transplant recipients: a systematic review, *Drugs*, Vol 67(8), pp 1167-1198

[6] Mueller N, 1998, Overview: Epidemiology of Malignancy in Immune-suppression, *Journal of Acquired Immune Deficiency Syndromes & Human Retrovirology,* Vol 17(4), p A38

[7] Murgia C, Pritchard JK, Kim SY, Fassati A & Weiss RA, 2006, Clonal Origin and Evolution of a Transmissible Cancer, *Cell*, Vol 126, pp 477-487

section is a very brief description from the US National Cancer Institute (NCI) of the function of the immune system.

4.2 The immune system

The immune system is a network of cells, tissues, and organs that has evolved to defend the body against foreign invasion.[8] The function of the immune system is briefly described as follows:

> The targets of the immune system are infectious organisms – bacteria, parasites and viruses. The function of the immune system therefore is to distinguish between "self" and "non-self". There is a set of unique markers on living cells called the major histocompatibility complex (MHC). There are two classes: Class 1 proteins, which are on all cells, and MHC Class II proteins, which are only on certain specialised cells. An immune response is triggered by an antigen and the distinctive markers on an antigen that triggers an immune response is called an epitope. When tissues or cells from another individual enter the body carrying such antigenic non-self epitopes, the immune system will mount a response.[9]

As described above the immune system is the body's defence mechanism against foreign non-self cells or tissues, intruders such as bacteria or viruses or cells transformed by cancer.[10] The immune system therefore is an important part of the body's defence mechanism and when it is compromised it leads to disease, cancer and possibly death. As noted in the previous chapter only dogs that are immune-compromised, the young and stray animals, have malignant cancers, whereas in devils, DFTD is a malignant cancer in all cases.

---

[8] National Cancer Institute, Understanding Cancer: The Immune System. Available at: www.cancer.gov/cancertopics/understandingcancer/immunesystem last accessed 23 July 2009

[9] ibid.

[10] Schindler L, Kerrigan D, Kelly J & Hollen B, nd, Understanding Cancer and Related Topics Understanding the Immune System, National Cancer Institute, p 14. Available at: http://www.cancer.gov/cancertopics/understandingcancer/immunesystem/immune.pdf last accessed 12 April 2013

4.3 The Tasmanian devil immune system studies

According to the Tasmanian devil scientific research literature no immune system studies of the Tasmanian devil had been undertaken prior to the detection of DFTD. Richmond Loh in his initial research into the devil cancer found that devils with DFTD did not mount an immune response, stating '[i]n most DFTD tumours there is little evidence of a cell mediated immunological reaction with only 7% containing any evidence of lymphocyte infiltration'.[11] Loh's research found more than 95% of devils with DFTD were between the ages of 2 and 4 years, which he found puzzling, and he recommended immune system studies on devils with DFTD.[12] Following Loh's observations and recommendations two studies on the devil immune system were undertaken. The devil samples were provided by the DPIPWE from their own captive breeding program and the studies were funded by the DPIPWE. Associate Professor Greg Woods and his then PhD student Alex Kreiss of the Menzies Research Institute undertook studies at the Royal Hobart hospital laboratory to assess firstly, the devils' immune structure and function and secondly, test for the possible development of a DFTD vaccine.

In 2008 Kreiss and colleagues concluded that the Tasmanian devils have a fully functioning immune system.[13] This result in devils was contrary to findings in other studies on a range of marsupial species, which had indicated a poorly developed immune system.[14] In concluding their article Kreiss et al ambiguously state:

---

[11] Loh RC, 2006, *The Pathology of Devil Facial Tumour Disease in Tasmanian Devils (Sarcophilus harrisii)*, Master of Philosophy, Murdoch University, Perth, Western Australia, p 90
[12] ibid, p 94
[13] Kreiss A, Fox N, Bergfeld J, Quinn SJ, Pyecroft S & Woods GM, 2008, Assessment of cellular immune responses of healthy and diseased Tasmanian devils (*Sarcophilus harrisii*), *Developmental and Comparative Immunology,* Vol. 32, pp 544-553
[14] ibid, p 551

> [t]here was no difference in immune responses between healthy and susceptible animals, but the need for high concentrations of mitogens may suggest that induction of immunity requires a strong stimulus. Importantly, susceptibility to DFTD is not a consequence of severely impaired cell-mediated immunity. However, as the variation in responses was large one may hypothesise that devils undergo transient periods of immunosuppression, potentially during periods of high stress, such as during mating season, and at this stage could be more susceptible to DFTD.[15]

Further they state '[i]f immune suppression is an important contributing factor to the transmission of DFTD, it was not due to an inability to induce lymphocyte stimulation and proliferation'.[16] This finding appears contrary to Loh's finding of little evidence of lymphocyte infiltration in the DFTD tumours, as discussed on the previous page.

Kreiss et al did not undertake a study of devil macrophages because they claimed it was deemed that the extraction process of these cells would be too invasive.[17] In a review of the book *The Macrophage* (2nd Ed.) published in the *British Journal of Cancer* in 2003, the reviewers state 'macrophages are part of the innate immune system which allows organisms to distinguish between self and non-self as opposed to the adaptive immune system comprising B and T lymphocytes; in relation to cancer, macrophages form a significant proportion of the total cell population in a vast majority of tumour tissue'.[18] A study of devil macrophages in relation to DFTD remains undone.

---

[15] Kreiss A, Fox N, Bergfeld J, Quinn SJ, Pyecroft S & Woods GM, 2008, Assessment of cellular immune responses of healthy and diseased Tasmanian devils (*Sarcophilus harrisii*), *Developmental and Comparative Immunology,* Vol. 32, pp 544-553
[16] ibid, p 552
[17] ibid, p 552
[18] Embleton MJ, 2003, Book Review, Burke, B & Lewis CE, 2002, *The Macrophage* (2nd Edn), Oxford University Press, Oxford, *British Journal of Cancer*, Vol 89, p 421

Kreiss and colleagues published the results of a second study of the devil immune system in 2009.[19] This study was limited because of the lack of availability of a statistically significant number of devils due to restrictions under the *Tasmanian Threatened Species* legislation. A permit (TFA 08088) granted by the DPIPWE was however issued to take a restricted number of devils for scientific purposes.[20] These devils included: 4 wild devils (roadkill); 2 captive devils; and a three-week old pouch young (mother died from DFTD). All were claimed to appear healthy and DFTD-free. Notwithstanding the limited number of study specimens Kreiss et al concluded, 'Tasmanian devil lymphoid tissues have all the structural elements required for effective T- and B-cell immune responses to disease.'[21] However, this claim was qualified by the statement '[t]here were some minor variations between the samples studied (data not shown) because of the opportunistic nature of the sampling, but it was beyond the scope of this article to compare different animals.'[22] They admitted 'it is not yet clear why DFTD-affected devils fail to develop effective immunological rejection for the facial tumor allograft…' but speculated that the 'paucity of lymphocyte infiltration in association with tumors' reported by Loh 'may be explained by low MHC diversity in the devil populations where high prevalence of DFTD has been detected.'[23] In this article the authors accept Loh's observation and propose a new explanation for the lack of lymphocyte infiltration in the tumours. The evidence for the explanation, that low MHC diversity may be the cause of the lack of lymphocytes in the tumours, is in

[19] Kreiss A, Obendorf DL, Hemsley S, Canfield PH & Woods GM, 2009, A Histological and Immunohistochemical Analysis of Lymphoid Tissues of the Tasmanian Devil, *The Anatomical Record*: *Advances in Integrative Anatomy and Evolutionary Biology*, Vol. 292(5), pp 611-620
[20] ibid, p 612
[21] Kreiss A, Obendorf DL, Hemsley S, Canfield PH & Woods GM, 2009, A Histological and Immunohistochemical Analysis of Lymphoid Tissues of the Tasmanian Devil, *The Anatomical Record*: *Advances in Integrative Anatomy and Evolutionary Biology*, Vol. 292(5), pp 611-620, pp 615-616
[22] ibid, p 616
[23] ibid, p 619

reference to an initial study published in 2007 by Siddle et al.[24] The Siddle et al study published in October 2007 in *PNAS* makes the claim 'DFTD is a transmissible tumor that spreads through a population due to a lack of histocompatibility barriers.'[25] This hypothesis, that a lack of histocompability barriers was the reason for transmissibility of the devil cancer, was later proven false.

At the time, however, this hypothesis formed the basis for further studies to determine if the Tasmanian devil immune system had the ability to recognise foreign cells. This study, published in 2009, was undertaken by Kreiss, Wells and Woods and tested antibody responses in devils over 40 weeks.[26] These experiments were undertaken in both *in vitro*[27] and *in vivo*[28] to evaluate the humoral immune response[29] of the Tasmanian devil. Again it was also noted that due to the endangered status of the devils only four devils, all of which were maintained by DPIPWE, were used in the experiments. Their findings indicated that Tasmanian devils are able to mount a humoral immune response as well as a memory response following two types of injections. However, cytotoxic T lymphocytes responses were not evaluated. According

[24] Kreiss A, Obendorf DL, Hemsley S, Canfield PH & Woods GM, 2009, A Histological and Immunohistochemical Analysis of Lymphoid Tissues of the Tasmanian Devil, *The Anatomical Record*: *Advances in Integrative Anatomy and Evolutionary Biology*, Vol. 292(5), pp 611-620
[25] Siddle, HV, Kreiss A, Eldridge MDB, Noonan, E, Clarke, CJ, Pyecroft, S, Woods GM & Belov K, 2007, Transmission of a fatal clonal tumor by biting occurs due to depleted MHC diversity in a threatened carnivorous marsupial, *PNAS,* Vol 104(41), 16221-16226, p 16224, p 16225
[26] Kreiss A, Wells B & Woods GM, 2009, The humoral immune response of the Tasmanian devil (Sarcophilus harrisii) against horse red blood cells, *Veterinary Immunology and Immunopathology,* Vol 130, pp 135-137
[27] In vitro: literally in glass; as in a test tube. Available at: http://www.medterms.com/script/main/art.asp?articlekey=4033 last accessed 17 August 2010
[28] In vivo: in the living organism, as opposed to in vitro (in the laboratory). Available at: http://www.medterms.com/script/main/art.asp?articlekey=4034 last accessed 17 August 2010
[29] Humoral refers to the non-cellular components of the blood, such as plasma and lymphatic fluid. The humoral immune response denotes immunologic responses that are mediated by antibodies. Available at: http://www.uptodate.com/contents/the-humoral-immune-response last accessed 30 December 2012

to Ito and Seishima '[c]ytotoxic T lymphocytes (CTLs)[30] constitute a distinct lymphocyte sub-population, and are induced by several diverse stimuli including major histocompatibility antigens... CTLs are involved in adaptive immune responses and are key players in mediating immunity against pathogens and tumors.'[31] Kreiss et al were aware that 'a successful anti-DFTD vaccine should also induce cytotoxic T cell activity, as this is the traditional immune response against tumours'.[32] It would appear that again this study lacked sufficient devil numbers to provide statistically significant results and critical studies were left undone.

Despite the inconclusive nature of the findings of the Tasmanian devil immune system studies, DFTD researchers continue to claim that the devils' immune system is not compromised. In order to demonstrate that immune competence is an anomaly in the Tasmanian devil malignant cancer, a comparison is given in the next section between four wildlife species, including the Tasmanian devil, threatened with extinction from cancer.

4.4 Wildlife cancers and immune systems

Tasmanian devils are not the only wildlife species threatened by a deadly cancer; three other small, localized populations of larger populations in various parts of the world are also threatened. These are the California sea lions in the San Francisco Bay, United States; the Beluga whales in the St Lawrence Estuary, Canada; and the green sea turtles in Moreton Bay, Australia and in other sub-tropical locations around the world. All

[30] Cytotoxic T Lymphocytes (CTLs) are cells that have the ability to directly kill cells. Cardiff University, 2009, T-cell Modulation Group, Cytotoxic T Lymphocytes, Cardiff University. Available at: http://www.tcells.org/scientific/killer/ last accessed 30 June 2013

[31] Ito H & Seishima M, 2010, Regulation of the Induction and Function of Cytotoxic T Lymphocytes by Natural Killer T Cell, *Journal of Biomedicine and Biotechnology*, Vol 2010, pp 1-8

[32] Kreiss A, Wells B & Woods GM, 2009, The humoral immune response of the Tasmanian devil (Sarcophilus harrisii) against horse red blood cells, *Veterinary Immunology and Immunopathology,* Vol 130, pp 135-137, p 137

inhabit environments that are heavily polluted with toxins, mainly from chemicals used in agriculture, although industrial and domestic toxins have also been detected. All, except the Tasmanian devil, have immune systems that are compromised or suppressed.

No experiments have been undertaken in any of the cases, including the Tasmanian devil, to assess the effects of toxins, including those detected in the environment or within the bodies of the various animals, on the immune system.[33] However, Guillette et al have called for further investigations into the role of endocrine disrupting contaminants (EDC) in the reproductive, immune and nervous systems in wildlife species, including the Beluga whale, exhibiting symptoms of EDC exposure.[34] A more detailed analysis of the wildlife cancers is provided in Chapter 6.

Studies into the devil immune system, as mentioned above, were undertaken with the result that it was found to be competent. With this result the research then turned to the role of genetics, in particular the MHC set of genes, as a reason for the ability of the tumour to transmit from devil to devil.

4.5 Is the lack of genetic diversity in devils a reason for cancer transmission?

In 2003 Menna Jones and colleagues had published a paper on devil genetics claiming 'moderate genetic variability across the species range'.[35] However, they concluded that random mating[36] occurs in all subpopulations.[37] In 2004 Jones and colleagues published

[33] Herbst, LH & Klein, PA, 1995, Green Turtle Fibropapillomatosis: Challenges to Assessing the Role of Environmental Cofactors, *Environmental Health Perspectives*, Vol. 103 Supplement 4, pp 27-30
[34] Guillette LJ, Crain DA, Rooney AA & Pickford DB, 1995, *Environmental Health Perspectives*, Vol 103, Supplement 7, pp 157-164
[35] Jones ME, Paetkau D, Geffen E & Moritz C, 2003, Microsatellites for the Tasmanian devil (Sarcophilus laniarius), *Molecular Ecology Notes* Vol. 3, pp 277-279, p 277
[36] Random mating. A population mating system in which every female gamete has an equal opportunity to be fertilized by every male gamete. The American Heritage Medical Dictionary, 2007, Houghton

a further paper this time suggesting that low genetic variation in devils is consistent with a founder effect, stating '[i]sland effects and repeated periods of low population density may also have contributed to the low variation'.[38] Despite this claim two distinct populations for Tasmania were identified, a well-connected eastern population and a smaller northwestern population. The population at Marrawah (west coast) was quite different genetically from all of the eastern Tasmanian populations. There was also some genetic variation in eastern populations, the Freycinet devils being quite different from those at Little Swanport as were the populations at Pawleena and Narawntapu.[39] In concluding the article they stated, '[r]ecent trends of population growth in devils indicate that survival and reproduction is not invariably compromised by low-moderate diversity in this species'.[40]

Contrary to these observations it was announced in 2008 by Hamish McCallum, Senior Scientist with the Save the Devil Program, that devils 'are so similar genetically that they have been described as having 'functionally identical MHC types'.[41] Menna Jones in an Australian Broadcasting Commission (ABC) *Science Show* interview with Robyn Williams claimed devils' 'genetic diversity is around about that of the cheetah or just slightly higher than the cheetah'.[42] This claim was supported by a further announcement on *ABC News* that the devil may be doomed because of inbreeding.[43] Kathy Belov, a

---

Mifflin Company. Available at: http://medical-dictionary.thefreedictionary.com/random+mating last accessed 30 June 2013

[37] Jones ME, Paetkau D, Geffen E & Moritz C, 2003, Microsatellites for the Tasmanian devil (Sarcophilus laniarius), *Molecular Ecology Notes* Vol. 3, pp 277-279, p 279

[38] Jones, ME, Paetkau, D, Geffen, E & Moritz, C, 2004, Genetic diversity and population structure of Tasmanian devils, the largest marsupial carnivore, *Molecular Ecology,* Vol 13, pp 2197-2209, p 2197

[39] ibid, p 2201

[40] ibid, p 2206

[41] Mc Callum H, 2008, Tasmanian devil facial tumour disease: lessons for conservation biology, *Trends in Ecology and Evolution*, Vol 23(11), pp 631-637

[42] *ABC The Science Show*, Tasmanian devil facial tumour disease, 3 June 2006. Available at: http://www.abc.net.au/rn/scienceshow/stories/2006/1652688.htm last accessed 9 August 2010

[43] Ogilvie, F, 2007, Inbred tassie devils face extinction: study, *ABC News*. Available at: http://www.abc.net.au/news/stories/2007/10/03/2050359.htm?section=australia 9 August 2010

geneticist from the University of Sydney's School of Veterinary Science, is quoted as saying 'even highly inbred populations tend to have a bit of genetic diversity in the MHC genes'. In 2004 Jones et al claimed the lack of genetic diversity may be the result of a founder or island effects or population crashes due to disease and genetic bottlenecks.[44] However, they observed '[n]o genetic signature of recent reductions (genetic bottleneck) or expansions in effective population size were found in any of the subpopulations…or the total population'.[45]

Meanwhile, Shelly Lachish, PhD zoology researcher at the University of Queensland and part of the *Save the Devil* program, reported to Matthew Denholm of *The Australian* newspaper that "[w]e did pre(DFTD) and post(DFTD) tests and basically there were elevated levels of a measure of inbreeding", but that '[w]hile markers for inbreeding and relatedness showed up, there was no evidence this had resulted in reduced genetic diversity'.[46] In 2011 Lachish et al claimed that a decline in genetic diversity was due to inbreeding subsequent to the DFTD outbreak: '[w]e observed a significant increase in inbreeding…in devil populations after just 2-3 generations of disease arrival, but no detectable change in genetic diversity.'[47] Research from the Schuster laboratory in the United States claims that while genomic diversity in the Tasmanian devil is low, it has not decreased much over the last century.[48]

---

[44] Jones, ME, Paetkau, D, Geffen, E & Moritz, C, 2004, Genetic diversity and population structure of Tasmanian devils, the largest marsupial carnivore, *Molecular Ecology,* Vol 13, pp 2197-2209, p 2197

[45] ibid, p 2201

[46] Denholm M, 2009, Tasmanian devils face new peril: inbreeding, *The Australian.* Available at: http://www.theaustralian.com.au/news/tasmanian-devils-face-new-peril-inbreeding/story-e6frg6no-1225764165914 last accessed 17 December 2012

[47] Lachish S, Miller KJ, Storfer A, Goldizen AW & Jones ME, 2011, Evidence that disease-induced population decline changes genetic structure and alters dispersal patterns in the Tasmanian devil, *Heredity*, Vol 106, pp 172-182, p 172

[48] Voss K, nd, Tasmanian Devil Genome Project: Press Release. Available at: http://tasmaniandevil.psu.edu/press_release.html last accessed 17 December 2012. Miller W, Hayes VM, Ratan A, Petersen DC, Wittekindt NE, Miller J, Walenz B, Knight J, Qi J, Fangping Z, Wang Q, Bedoya-Reina OC, Katiyar N, Tomsho LP, Kasson LMcC, Hardie RA, Woodbridge P, Tindal EA, Bertelsen MF, Dixon D, Pyecroft S, Helgen KM, Lesk AM, Pringle TH, Patterson N, Zhang Y, Kreiss A, Woods GM,

Much ambiguity exists in the results of the testing of the hypothesis that genetic diversity and inbreeding is a reason for the transmissibility of DFTD, and whether a genetic bottleneck existed prior or subsequent to DFTD. The cheetah, with its low genetic diversity, was cited as a precedent and a brief discussion of the literature on this topic, much of which has been written by Stephen J O'Brien, Chief of the Laboratory of Genomic Diversity with the Center for Cancer Research, follows.

4.5.1 The Cheetah precedent

Regardless of the uncertainty surrounding the role of genetic diversity in DFTD, the dominant hypothesis, first proposed by Menna Jones in an interview, became devils, like cheetahs, are inbred. Serengeti cheetahs at some stage in the past, through a population bottleneck mated with closest relatives, resulting in genetic uniformity.[49] In devils it is proposed inbreeding enables DFTD to be transplanted between devils.[50] In the Serengeti cheetah lack of genetic diversity results in their susceptibility to a virus that threatens the survival of the population from a wasting disease. In his book *Tears of the Cheetah* Stephen O'Brien documents the experiments used to test the hypothesis that a lack of genetic diversity in Serengeti Cheetahs might be the cause of vulnerability to this wasting disease.[51] A brief outline of these studies follows.

O'Brien's scientific observations and the twelve different experiments undertaken to determine the cheetah population's genetic diversity involved: autografts, the transplanting of tissue from the cheetah's own body; allografts, transplanting of tissue

---

Jones ME & Schuster SC, 2011, Genetic diversity and population structure of the endangered marsupial Sarcophilus harrisii (Tasmanian devil), *PNAS*, Early Edition, pp 1-6. Available at: www.pnas.org/cgi/doi/10.1073/pnas.1102838108 last accessed 18 December 2012

[49] O'Brien, SJ, 2003, *Tears of the Cheetah*, St Martin's Press, New York

[50] Price M, 2011, For Conservationists, the (Tasmanian) Devil Is in the details, *Science*. Available at: http://news.sciencemag.org/sciencenow/2011/06/for-conservationists-the-tasmani.html last accessed 29 December 2012

[51] O'Brien SJ, 2003, *Tears of the Cheetah*, St Martin's Press, New York

between unrelated cheetahs but of the same species; and xenografts, transplanting of tissue between unrelated species. It was found that the autografts and the allografts took in all cases.[52] These experiments confirmed that cheetahs were extremely inbred and lacked genetic diversity. Support for the finding was provided by observations of asymmetry in cheetahs, something particular to inbred species. Hence, when compared to the skulls of leopards, they 'certainly looked very inbred'.[53]

Zookeepers were the first to notice a problem in cheetahs when they encountered difficulty in breeding them in captivity. It was hypothesised that a lack of genetic diversity might be the problem. Subsequently a number of experiments on the captive cheetahs proved their immune system did not recognise tissue transplants as non-self, and so they appeared to be inbred.[54] The experiment was repeated in Pretoria, South Africa on an eastern population of wild cheetahs with the result that seven out of ten allografts, between seemingly non–related cheetahs, were accepted.[55] The same experiment was then undertaken on the western population and they found similar results. The cheetah's MHC genes were analysed to determine if a lack of diversity in this most diverse set of genes could be the problem. It was found that cheetahs did lack genetic diversity probably due to a previous bottleneck in the population.

The bottleneck is proposed to have occurred around 12,000 years ago during an ice age that resulted in a large number of animal extinctions.[56] The cheetah apparently escaped extinction very narrowly and it is possible only one female and her cubs survived to re-breed and populate. The species, based on these few survivors, successfully bred to the

[52] ibid, p 25
[53] ibid, p 2
[54] ibid.
[55] ibid.
[56] ibid, p 34

present large numbers. Although it was shown beyond doubt that cheetahs are very closely related, the decline in the species may not be due to a lack of genetic diversity, but to human activities such as habitat destruction.[57]

The highly respected scientist Edward O Wilson in his book *The Future of Life* states 'if the species manages to pass through a bottleneck of very low population size and still survive, the depression may in the course of the passage "clean out" the defective genes.[58] Such a genetic purge evidently occurred in the cheetah.'[59] He goes on to say they 'did not perish from genetic defects, as might be immediately suspected' but 'the principal causes instead were predation by lions and spotted hyenas, along with abandonment by the mothers during periods of food scarcity'.[60] According to Wilson a very small or very local population is most vulnerable to demise from a natural disaster, such as storm, fire or drought.

The Tasmanian devil researchers have not undertaken the exhaustive experiments described above to prove the devils' genetic diversity is similar to that found in the cheetah population. Despite this the DFTD research team did not deviate from the belief that the devils' lack of diversity in its MHC genes is the reason the cancer is transmissible. Stephen O'Brien visited Hobart on an invitation from DPIPWE and although the meeting was publicized in local newspapers, no reports of his views on the devil disease were made public. However, Pearse in a personnel communication informed me that those who attended were asked to think of 'pot-stirring' questions. She herself asked two questions. The first: are Dasyurids (the family to which the devils

[57] ibid.
[58] Wilson EO, 2002, *The Future of Life*, Abacus, London, p 57
[59] ibid, pp 56-57
[60] ibid, p 57

belong) at the end of their natural existence? To which O'Brien replied – 'rubbish'. The second question was, if vaccines were created against the devil cell-lines isn't there the danger of the devils developing an autoimmune disease? O'Brien replied – 'sure'.

Regardless of the lack of studies the devil researchers remain committed to the allograft hypothesis and to the conviction that a lack of genetic diversity in the devils' MHC genes was why the cancer could establish in a new host.

4.5.2 The role of the majorhistocompatibility (MHC) genes in the devil cancer

The MHC comprises the most diverse genes in all vertebrate species.[61] The MHC is not only the most diverse set of genes - it also controls the immune function in all animals.[62] The studies into the devil MHC genes were also groundbreaking research. In 2006 two papers were published on the MHC of a marsupial - the gray, short-tailed opossum (*Monodelphis domestica*).[63] The first was published in the journal *Cytogenet Genome Research* providing an analysis of genetic organization and chromosome localization of the MHC of this marsupial. The authors noted that until this research 'no chromosomal location and physical arrangement of the various classes of MHC genes has been undertaken for any marsupial genome'.[64]

The second paper published in *PLoS Biology* by Kathy Belov and colleagues constructed the first map of the marsupial gray, short-tailed opossum.[65] Kathy Belov

---

[61] Knapp A, 2007, Selection on MHC: A matter of form over function, *Heredity*, Vol. 99, pp 241-242
[62] Janeway DAJr, Travers P, Walport M, et al, 2001, *Immunobiology: The Immune System in Health and Disease,* 5th Ed, Garland Science, New York
[63] Gouin N, Deakin JE, Miska KB, Miller RD, Kammerer CM, Graves JAM, VendeBerg JL & Samollow PB, 2006, Linkage mapping and physical localization of the major histocompatibility complex region of the marsupial [*Monodelphis domestica*], *Cytogenet Genome Research*, Vol. 112 pp 277-283
[64] ibid, p 278
[65] Belov K, Deakin JE, Papenfuss AT, Baker ML, Meiman SD, Siddle HV, Gouin N, Goode DL, Sargeant TJ, Robinson MD, Wakefield MJ, Mahony S, Cross JGR, Benos PV, Sammollow PB, Speed,

was also to undertake the first study of the Tasmanian devil MHC genes with her PhD student Hannah Siddle and research assistant Claire Sanderson; it was published in *Immunogenetics* in August 2007.[66] In this latter paper they stated '[w]e have made the first genetic library for the Tasmanian devil, a spleen cDNA library, and have isolated and characterized full-length MHC Class I and Class II genes'.[67] It explains the methodology; 'RNA and DNA was extracted from spleen, blood, kidney and liver from a single male Tasmanian devil' and 'DNA was extracted from the blood of five additional devils'.[68] It concludes

> [t]his study has provided the fundamental information required to study the MHC biology of Tasmanian devils in relation to DFTD. We have isolated Class I and Class II DAB sequences, which are likely to be involved in immune response and antigen presentation, and have developed markers to study MHC diversity in wild populations. Extensive polymorphism studies of the classical Class I and Class II MHC loci are now in progress in our lab.[69]

At the same time, August 2007, Woods et al published in *EcoHealth* a paper stating '[a] lack of MHC expression is unlikely to account for the failure of the devil's immune system to reject the DFTD allografts because the tumor cells, which were analyzed by constructing a cDNA library, all expressed MHC class Ia and Class II genes.[70] They proposed an alternative explanation 'that there is a lack of genetic diversity within the devil population and the "cancer graft" MHC types are identical to those of the host' concluding that a 'lack of diversity at MHC genes' results in a 'failure of the DFTD tissue to be recognized as "non-self" by the host's immune system.'[71] They cite as

---

TP, Graves JAM & Miller RD, 2006, Reconstructing an Ancestral Mammalian Immune Supercomplex from a Marsupial Major Histocompatibility Complex, *PLoS Biology*, Vol. 4(3), pp 0317-0328

[66] Siddle HV, Sanderson C & Belov K, 2007, Characterization of major histocompatibility complex class I and class II genes from the Tasmanian devil, *Immunogenetics*, Vol. 59, pp 753-760

[67] ibid, p 753

[68] ibid, p 754

[69] ibid, p 759

[70] Woods GM, Kreiss A, Belov K, Siddle HV, Obendorf DL & Muller KH, 2007, The Immune Response of the Tasmanian Devil (*Sarcophilus harrisii*) and Devil Facial Tumour Disease, *EcoHealth*, Vol 4(3), pp 338-345, p 343

[71] ibid.

evidence the August 2007 paper by Siddle et al, which claims samples were taken from a single, male Tasmanian devil (Individual I) and DNA was extracted from the blood of five additional devils.[72] There is no mention of taking samples from devil tumor cells for analysis. The Siddle et al paper also does not confirm that the lack of MHC is the reason why the tumours proliferate.

However, in October 2007, Siddle, her supervisor Kathy Belov and DPIPWE devil researchers published a further paper in *PNAS* (referred to in the previous chapter in section 3.3).[73] In this article they claimed '[t]his novel disease arose as a direct result of loss of genetic diversity...'.[74] In 2008 Wood confirmed that the DFTD cells had not been examined for MHC markers.[75] There are still no studies published indicating an investigation of MHC markers on the devil DFTD cells.

On the *Save the Tasmanian Devil* website in 2007 Belov is quoted as saying "[i]n the case of devils from eastern Tasmania, genetic diversity at the MHC is so low, and the MHC type of tumour and host are so alike, that the host does not see the tumour as 'non-self'".[76] Woods is also quoted as saying 'we now have a tool to measure immune response genes and we are now in search of devils whose MHC might be different from the MHC of the tumour'.[77]

---

[72] Siddle HV, Sanderson C & Belov K, 2007, Characterization of major histocompatibility complex class I and class II genes from the Tasmanian devil, *Immunogenetics*, Vol. 59, pp 753-760, p 754
[73] Siddle HV, Kreiss A, Eldridge MDB, Noonan E, Clarke CJ, Pyecroft S, Woods GM & Belov, K, 2007, Transmission of a fatal clonal tumor by biting occurs due to depleted MHC diversity in a threatened carnivorous marsupial, *PNAS,* Vol 104, No. 41, pp 16221-16226
[74] ibid, p 16221
[75] Personnal interview with Greg Woods in Hobart, 14 November 2008
[76] Anon, 2007, A lack of genetic diversity, *Save the Tasmanian Devil*. Available at: http://www.tassiedevil.com.au/tasdevil.nsf/TheProgram/BFD7236BFF934B62CA2576D200174180 last accessed 17 August 2010
[77] ibid.

In 2010 Siddle, Belov, Jones and colleagues from the University of Sydney's Faculty of Veterinary Science published a paper in the *Proceedings of the Royal Society B* journal. In this study they undertook a comprehensive screen of MHC diversity in devils and concluded overall levels were low. In an apparent about turn they conclude '[c]ounterintuitively, we postulate that the immune system of devils with a restricted MHC repertoire may recognize foreign MHC antigens on the surface of the DFTD cell.'[78] A subsequent media report in May 2011 in *The Australian* newspaper stated '[w]ith almost identical DNA across the whole population, Tasmanian devils are like 'walking zombies" spreading cancer by biting each other, University of Adelaide researchers say'.[79]

However, the hypothesis that a lack of genetic diversity in the devil MHC genes is the reason why the cancer could establish in a new devil host was eventually abandoned. In a 2012 interview Kathy Belov told Rachel Carbonell on the ABC program *The World Today* 'that in trying to prove the theory her team instead debunked it'.[80] It had been made public by Assistant Professor York that the devils' MHC was not involved as discussed below.

Assistant Professor Ian York of Microbiology and Molecular Genetics at Michigan State University had provided a credible challenge to the hypothesis. Posted on his website he relates an encounter with Elizabeth Murchison (the young Tasmanian scientist who struggled to get access to the devils cell lines for experiments at Cold

[78] Siddle HV, Marzec J, Cheng Y, Jones M & Belov K, 2010, MHC gene copy number variation in Tasmanian devils: implications for the spread of a contagious cancer, *Proceedings of the Royal Society B*, published online. Available at: www.rspb.royalsocietypublishing.org last accessed 12 March 2010
[79] Peddie C, 2011, Lack of genetic diversity contributes to Tasmanian devil cancer deaths, *The Australian*. Available at: www.theaustralian.com.au/news/breaking-news/devils-in-the-lack-of-diversity/story-fn3dxity-1226054415704 last accessed 13 May 2011
[80] Carbonell R, 2012, Tasmanian devil facial tumour theory debunked, *ABC The World Today*. Available at: http://www.abc.net.au/worldtoday/content/2012/s3523185.htm last accessed 31 December 2012

Springs in the US).[81] She had informed him that tissue transfers undertaken between devils were unsuccessful. On reflection Professor York posted the following on his website:

> Murchison told me that Tasmanian Devils — even those in the same sub-population — vigorously reject each others' skin grafts. This is what's supposed to happen with skin grafts, of course. It implies that the Devils do not, in fact, have the same MHC; and in my opinion it's a much stronger experiment than those in the original homogenous-MHC paper. If Devils reject skin grafts from each other, then they ought to reject tumors from each other — in other words, even if the tumor can take in one individual, then it should be rejected in another, so the tumor should not spread throughout the population. The skin graft finding hasn't, as far as I know, been published, but if it holds up, it's a strong argument against homogenous MHC.[82]

York concludes that the devils' MHC is not involved in the transmission of the cancer. DPIPWE research notes (Attachment C) confirm Murchison's claim stating '[a]ll eastern devils tested 'in vivo' allograft experiments - total of 8 animals - all showed host-graft or graft-host rejection'. It further states:

> [t]here is diversity present in the MHC class II but only one family of genes has been examined; and then, there's class III. This class II diversity gives validation for Kreiss's uniform host-graft rejections in the skin graft experiments. MHC class II are found on the immunologically competent stem cells and their progenitors - they help to recognise exogenous antigens (microbiological/parasitological/viral).

At the time of writing, April 2013, the MHC research appears to have been abandoned but the search for a vaccine or a resistant population, possibly on the west coast, continues.

4.6 Resistance and the search for a vaccine

Carina Dennis in an article in *Nature* in 2006 was the first to moot devil resistance to DFTD publicly when she referred to Stephen Pyecroft's work at the DPIPWE's Mount

[81] Bryan E, Devil DNA to go to New York for testing, *The Examiner*. Available at www.examiner.comau/print.asp?ip=372048 last accessed 21 November 2006
[82] York I, 2009, Vertical transmission of tumors. Available at: http://www.iayork.com/MysteryRays/index.php?s=tasmanian+devil last accessed 1 July 2013

Pleasant Laboratory, where he was looking for genetic variation, which could confer resistance.[83] Resistance was also referred to by Nick Mooney, Wildlife Officer with DPIPWE, when he stated "I mean the history of diseases like this is that some animals are resistant to a disease – I mean it doesn't matter what infectious disease it is usually,...".[84] Adhering to the conventional practice of seeking a way to prevent the spread of a contagious disease the devil scientists focused on finding a vaccine. It is extremely unlikely that a vaccine would be found for a number of reasons but the fact that the cancer was evolving into different strains was also a significant hurdle. The vaccine trials were undertaken on a number of devils the most public being the trials on two devils named Clinky and Cedric. The results of the study have not been peer reviewed or published formally but were reported through the popular media and the DPIPWE's *Save the Tasmanian devil* website.[85]

4.7 Cedric and Clinky

The Devil researchers proposed that as devils on the east coast of Tasmania have succumbed to DFTD, it is possible that the DFTD-free devils on the west coast could be a resistant population. The research pathway dominated by the allograft theory, that the cancer was transmissible, meant that the competing hypothesis, that the cancer was caused by increasing use of pesticides in plantation forests on the eastern side of Tasmania, was ignored. The proposal that west coast devils could be resistant to the cancer was the basis for the vaccination trials on devils Cedric and Clinky. It was anticipated that resistance in a devil, or the development of a vaccine, could lead to a way to prevent for the Tasmanian devil cancer. Matthew Denholm published news of a

[83] Dennis C, 2006, Time to Raise the Devil, *Nature*, Vol. 439, p 530
[84] Breakthough in Tasmanian Devil Disease, *Bio-Medicine*, 2006, http://www.bio-medicine.org/medicine-news/Breakthrough-In-Tasmanian-Devil-Disease-7497-1/ 10 May 2009
[85] There are a number of print, television, radio and electronic publications covering the story of Cedric and Clinky both national and international.

possible resistant devil in *The Australian* newspaper on 31 March 2008.[86] The article stated that the devil Cedric had shown an immune response to DFTD, that he was the first devil to do so, and it was expected he would remain disease free. It was also suggested that devils with similar genes to Cedric could also be resistant to DFTD or capable of responding to a vaccine.

According to the immunogenic studies research notes (Appendix D) three devils from the western population were used in a trial to assess if devils could develop an immune response to a DFTD vaccination.[87] The three devils were named Cedric and Klinky (aka Clinky) half-brothers and their mother Christine. Christine was a female devil from a Woolnorth population on the west coast of Tasmania. The research notes state Cedric was a male offspring from a wild mating, while Clinky was a male offspring from a captive mating with an Arthur River (west coast) male.[88]

Christine, according to the notes, developed no detectible immune response to a DFTD[89] vaccination but developed tumours 16 weeks after being inoculated with DFTD cells. She was subsequently reimmunised against 4 different strains[90] of DFTD and again developed tumours, which were removed. Following an examination in week 70 which showed no palpable tumours she was later found comatose and was euthanased. Cedric had developed no immune response to a DFTD vaccination at week

[86] Denholm M, Hope of cure for dying Tasmanian devils, *The Australian,* 31 March 2008. Available at http://www.theaustralian.com.au/news/nation/hope-of-cure-for-dying-tasmanian-devils/story-e6frg6nf-1111115929033 last accessed 3 September 2010

[87] These notes varied from the information provided to me in conversation with Alex Kreiss in Hobart on 14 November 2008. He was later to suggest that he had misunderstood my questions but my only question to him was – what is your role in the scientific research into the devil cancer?

[88] These notes differ from the media reports where it is Cedric that is the offspring from a captive mating and who appeared to be resistant to the cancer.

[89] Although it is termed Devil Facial Tumour (DFT) in the notes it is the same disease as Devil Facial Tumour Disease (DFTD) and I have maintained DFTD for consistency.

[90] The inoculation of different strains of cancer would suggest that DFTD is in fact different cancers and is unstable previously referred to in Chapter 4.

41 and was subsequently challenged with strains 2 and 3 of the DFTD cancer. In week 90 Cedric also developed two facial tumours.[91] Clinky, on the other hand developed a strong immunity to a DFTD vaccination when he was challenged at week 8. He was challenged again in week 30 with no obvious antibody response detected. In week 41 he was challenged this time with strain 2 and at week 53 developed tumours at inoculation sites.

The apparent resistance in one devil was claimed as a breakthrough.[92] On the *Save the Tasmanian Devil* website in 2008, Associate Professor Greg Woods explained 'this male devil (Cedric) was injected with dead DFTD tumour cells' and subsequently 'Cedric produced an immune response as his body recognised the cancer cells as foreign'.[93] Woods further explained '[d]evils don't produce immune responses to DFTD because the diseased cells are too similar to their own cells' he continues '[b]ut what we've found is that Cedric's MHC is sufficiently different to the tumour or the diseased cells to be recognised as foreign'.[94] It is further proposed that a west coast 'group may be so genetically different that they are naturally resistant to the disease'.[95] However, it was reported in the media on 17 December 2008 that Cedric had developed DFTD.[96] It was not revealed until September 2010 that Cedric had been euthanised when X-rays revealed he had lung tumours.[97]

---

[91] Sharman A, Welcome, *Devil News*, March 2009, DPIPWE, Hobart, p 2
[92] Hayden EC, 2009, Genome scan may save Tasmanian devils from cancer, *Nature News*. Available at: http://www.nature.com/news/2009/090303/full/news.2009.132.html last accessed 31 December 2012
[93] Anon. 2008, Cedric's life inheritance, *Save the Tasmanian Devil*. Available at: http://www.tassiedevil.com.au/tasdevil.nsf/TheProgram/49364AFDF5B41207CA2576D2000DD302 last accessed 17 August 2010
[94] ibid.
[95] ibid.
[96] Serious setback in race to save the Tasmanian devil, 17 December 2008. Available at http://origin-www.thewest.com.au/default.aspx?MenuID=28&ContentID=113744 last accessed 14 August 2010
[97] *The World Today with Eleanor Hall*, ABC Radio National, Cedric the Tasmanian devil dies, 1 September 2010. Available at: http://www.abc.net.au/worldtoday/content/2010/s2999435.htm last accessed 3 September 2010

These experiments were to test whether or not the DFTD researchers could develop a vaccine, which would enable these devils to mount a resistance to DFTD. However, all three devils succumbed to the disease, which suggests the experiment failed. The results of the experiment have never been published and the cause of the devils' cancers has not been explained. It is also not known if there were experimental controls on variables such as contaminants in the food and water or the environment of the devils in the experiment.

The discovery of resistance within the devil population or the development of a vaccine for DFTD does not appear likely within the foreseeable future. New researchers have now embarked on the sequencing of the entire devil genome to meet the challenges and enable a conservation project to maximize devil genetic diversity.

4.8 Sequencing of the devil genome - a conservation project

The Tasmanian Devil Genome Project aims to help scientists understand identify and establish an "insurance population".[98] According to the Save the Tasmanian Devil website 'the ultimate aim is to establish targets to generate an immune response in infected animals, or to possibly produce a vaccine'.[99] In a media release dated 22 September 2008 the Children's Cancer Institute Australia (CCIA) stated that '[r]esearchers from CCIA, together with US collaborators, are aiming to undertake the huge task of generating a complete DNA sequence of the Tasmanian Devil. This sequence will be used to develop markers to breed a healthier and stronger Tasmanian

[98] Tasmanian Devil Genome Project: Our Research. Available at: http://tasmaniandevil.psu.edu/our_research.html last accessed 25 April 2013
[99] Save the Tasmanian Devil Newsletter, March 2008, DPIPWE, Hobart, p 2

Devil population that is resistant to this infection'.[100] It is to be undertaken at the Schuster Lab at Penn State University, US, by an Australian scientist Dr Vanessa Hayes and Professor Stefan Schuster. Dr Hayes is group leader for the Cancer Genetics Group of the Children's Cancer Research Institute (CCRI) and Adjunct Professor of Biology, Pennsylvania State University.[101] Hayes is currently working on the effect of DNA variation on prostate cancer risk in Australia and was recruited to CCIA in 2008 to establish a state-of-the-art genomics laboratory with new generation sequencing technologies. Hayes in an *ABC* interview with Felicity Ogilvie said that the 'reason why these animals cannot fight the cancer is because it hasn't got enough genetic diversity'.[102] The plan is to 'create as much diversity as we can'.[103]

The sequencing of the devil genome began when Elizabeth Murchison requested DNA samples from the Tasmanian devil so she could research DFTD at the Cold Spring Harbor Laboratory (CSHL). After originally being denied access to the devil material by the DPIPWE, the Tasmanian government, following strong criticism, conceded samples would be sent.[104] The CSHL research team formed a collaboration with 454 Life Sciences to sequence parts of the devil genome. In an interview the director of research at the CSHL, David L Spector, said "[o]ur efforts to sequence the devil's genome mark the first time anyone has attempted to use the technology for exploring this particular type of cancer biology" and further stated "[w]hen we have a complete

---

[100] Media Release, 22 September 2008, *Kids Raise Money to save the Tassie devil and find a cure for children's cancer,* Children's Cancer Institute Australia, Sydney, Australia
[101] Australian-Canadian Prostate Cancer Research Alliance, Dr Vanessa Hayes. Available at: http://www.aus-canprostatealliance.org/Members/vhayes-40ccia.unsw.edu.au last accessed 31 December 2012
[102] Ogilvie F, 2008, Tassie devil may help human cancer research, *Australian Broadcasting Commission The World Today*, 18 December. Available at: http://www.abc.net.au/worldtoday/content/2008/s2450069.htm last accessed 25 April 2013
[103] ibid.
[104] *ABC Online*, 2006, Devil DNA to be sent to US for facial tumour research. Available at: http://www.abc.net.au/newsitems/200611/s1794013.htm last accessed 2 June 2007

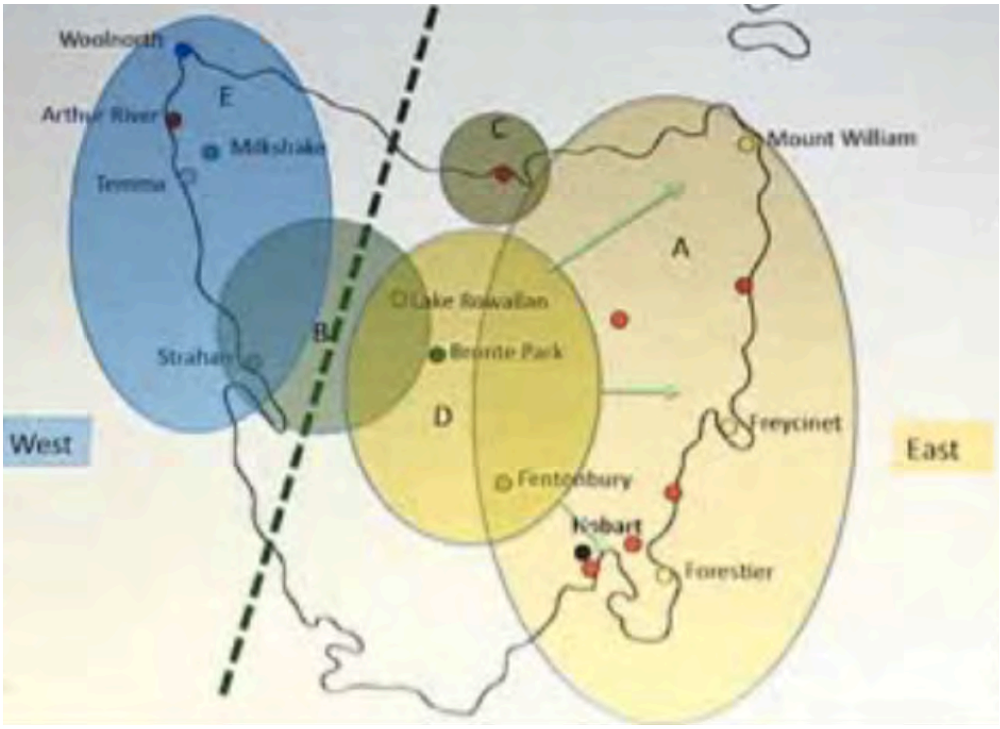
view of the devil tumor genes, scientists will be able to identify the cancer causing genes, which may lead to the development of therapies and vaccines".[105] 454 Life Sciences is a Roche company and according to their website a center of excellence of Roche Applied Science.

On the *Save the Tasmanian Devil* website under the heading 'Using genetics to guide selective breeding' Hayes is reported to have said 'the cheetah is a perfect comparison to the devil'.[106] In support of the conservation effort she is quoted as saying "The cheetah was headed for extinction due to in-breeding and low genetic diversity until genetics was used to guide selective breeding".[107] The genetic studies had now shifted from studies of low MHC diversity as a possible reason for the transmission of the cancer, to finding enough genetic diversity to save the species.

On 18 June 2009 the ABC program *Catalyst* ran an update on the progress of the sequencing of the devil genome.[108] Hayes appeared on the program with a map, Figure 4:1 below, showing the genetic diversity in nearly 200 devils from across Tasmania. There are five groups, A to E, shown in different colours.

[105] Bono J, 2008, Cold Spring Harbor Laboratory Researchers Race Against Time to Save Tasmanian Devils, Cold Springs Harbor Laboratory. Available at: http://www.cshl.edu/public/releases/08_save_taz.html last accessed 10 May 2009
[106] Save the Tasmanian Devil, 2008, Using genetics to guide selective breeding, DPIPWE, Hobart. Available at: http://www.tassiedevil.com.au/tasdevil.nsf/0/e7e180ed50a05a5dca2576d200179c84!OpenDocument&Click= last accessed 3 October 2013
[107] ibid.
[108] Australian Broadcasting Commission Television, *Catalyst*, Devil in the Detail, 18 June 2009. Available at: http://www.abc.net.au/catalyst/stories/2601084.htm# last accessed 11 April 2012

Figure 4:1 Genetic variation in Tasmanian devil populations[109]



This map confirms Menna Jones' and colleagues' research findings published in 2003 that a distinct population exists on the west coast and three or more genetically different groups also exist on the east coast.[110] Professor Woods and colleagues had also considered the west coast devils genetically different enough from the east coast devils to be used in their experiments with devil resistance when they used Cedric a devil bred from a west coast male.

The Tasmanian devils have probably moved through population bottlenecks in the past reducing their genetic diversity but in 1996, when the first devil with DFTD was photographed, the population numbered approximately 150,000. The devils had retained sufficient genetic diversity to breed successfully and re-populate to such an extent that they were at the time considered a pest.

---

[109] ibid.

[110] Jones M, Paetkau D, Geffen E & Moritz C, 2003, Microsatellites for the Tasmanian devil (*Sarcophilus harrissi*) in *Molecular Ecology Notes*, Vol 3(2), pp 277-279

4.9 Conclusion

Within the framework of conventional cancer theory malignant tumours are not considered transmissible. According to Assistant Professor Ian York of the Microbiology and Molecular Genetics Department of the Michigan State University tumours are unique, arising independently each time and when their host dies, the tumour also dies.[111] This is in contrast to infectious pathogens which are not unique, may or may not be killed by their host, and survive to be transmitted to a new host. According to York 'tumours can't do this, for the same reason that skin grafts are rejected by unrelated animals - tumours are essentially unrelated grafts'.[112]

It is claimed that the devil cancer is a clonal cell line transmitted from devil to devil through biting. In order for this to occur either the devils' immune system must be compromised (it is said to be competent), its immune system does not mount a response to the cancer (according to Loh) or the MHC antigen receptors on the tumour cells are inactivated or are not there. According to the research notes from the Kreiss and Woods immunogenic studies (Appendix D) MHC class II antigen receptors are absent from the tumour cells – but no further information is given. This remains a research problem that has not been investigated.

The absence of MHC antigen receptors may possibly be linked to toxins in the environment. The triazine chemicals atrazine and simazine together with other chemicals used in plantation forestry are known to cause immunosuppression or inactivate MHC antigens on cells. Atrazine exposure caused a dose-dependent removal

[111] York IA, 2009, Why aren't most tumors transmissible?. Available at: http://www.iayork.com/MysteryRays/2009/08/13/why-arent-most-tumors-transmissible/ last accessed 9 August 2010
[112] ibid.

of surface MHC-1 in a study by Pinchuck *et al*.[113] The possible role of atrazine in DFTD and three other wildlife cancers will be discussed in Chapter 6.

The devils have a cancer that is fatal in all cases leading to the possible extinction of the species. The cause of the initial devil cancer, aside from the hypothesis that it is spread via biting, remains unknown. The DPIPWE first thought either a carcinogen or a virus was the logical cause. As a virus has been discounted the next hypothesis to investigate would seem to be a chemical carcinogen. According to *Scammell Report* the increase in plantations, the increase in the devil disease and the oyster abnormalities all in the northeast were all correlated in time and space.

The allograft theory points to devil behavior that has evolved as a ritualistic display to maximize their potential as a species now working against them to extinguish the species. Its mating habits - the male dragging the female into its den and inflicting injury, (although this is mainly to the back of the neck where the skin has thickened); its eating habits when congregated at a carcass, biting each other (although their whiskers protrude so they can sense their neighbour hence avoiding direct physical contact); the failure of its immune system to recognise foreign or abnormal cells; and finally the failure of its MHC to alert the devil against mating with its next of kin,[114] are all supposed to have set it up for the transmission of the cancer.

The anomaly in the immune system, that it is competent, did not divert the researchers from the allograft theory. Adjustments were made to the theory, however, and a search

[113] Pinchuk LM, Lee SR & Filipov NM, 2007, In vitro Atrazine Exposure Affects the Phenotypic and Functional Maturation of Dendritic Cells, *Toxicology and Applied Pharmacology*, Vol 223(3) pp 206-217
[114] Eldridge M, 2009, *Characterisation of the marsupial Major Histocompatibility Complex (MHC),* Australian Museum. Available at: http://australianmuseum.net.au/research/Characterisation-of-the-marsupial/ last accessed 1 July 2013

for a lack of genetic diversity in the devil MHC guided the research. This search has also since been abandoned. The research funding and the scientists still support the allograft theory and the search for a vaccine and genetic diversity to support a conservation program continue. All this investigation of why the cancer grows does not provide evidence confirming the allograft theory.

Questions still remain - Why did the cancer emerge in the mid-1990s in correlation with an increase in plantations and their use of chemicals and why on the east coast and not on the west coast? The research into a competing hypothesis – that toxins in the environment contributed to the initiation or progression of the cancer - will be explored in the next chapter.

Chapter 5 – DFTD toxicology studies - the undone science

5.1 Introduction

There is a compelling alternative hypothesis for the devil cancer that warrants thorough investigation before it can be concluded that DFTD is a transmissible disease. Are environmental toxins, either singularly or in synergy, a contributing factor in the devil cancer? David Obendorf and Neil McGlashan's request for 'a truthful investigation of the local environmental conditions that preceded the index outbreak' in the devil population was also ignored because the research pathway was dominated by the allograft theory.[1] The extent to which chemicals from either mining, agriculture or forestry industries have contributed to the devil cancer needs to be addressed given the history of contamination of soil and water from these activities in Tasmania.[2] Comprehensive toxicology studies were outlined in the DPIPWE's DFTD Disease Management Strategy in 2005. The strategy recommended:

- identify target toxins
- determine exposure levels
- do invitro studies
- investigate correlations between use of toxins and disease areas.[3]

Despite the acknowledged need for the studies they were first delayed and later, following an initial pilot study, abandoned. A pilot study did find flame retardants in devil tissue, which prompted further requests for toxicological investigations. However, to date no further studies have been undertaken. It is currently unknown if toxins in the

[1] Obendorf DL & McGlashan ND, 2008, Research priorities in the Tasmanian devil facial tumour debate,
[2] Tasmanian Government, *State of the Environment Tasmania*. Available at: http://soer.justice.tas.gov.au/2003/lan/2/issue/92/ataglance.php last accessed 2 January 2013
[3] Department of Primary Industries, Water and Environment, 2005, *Tasmanian Devil Facial Tumour Disease (DFTD) Disease Management Strategy*, Department of Primary Industries, Water and Environment, Hobart, Tasmania

environment, either heavy metals or agricultural chemicals, singularly or in synergy, are responsible for the devil cancer. However, two recent scientific developments concerning the role of endocrine disrupters (chemicals that mimic hormones) and epigenetic effects (environment-induced expression or suppression of genes) indicate the need for further investigations. The relevance of these developments in relation to the Tasmanian devil cancer is discussed in chapter 7. In this chapter, using the concept of undone science, I analyse the research into devil toxicology studies. This analysis follows my finding of only one peer-reviewed and published article on devil related toxicology. As background to the analysis the following sections give an overview of why it might be necessary to undertake these studies.

5.2 Why test for chemicals?

Tasmania is a small island with a population of approximately 512,875 persons as at 31 March 2013.[4] The Tasmanian economy relies heavily on mining, its largest source of income, and forestry. The mining industry is worth $A1.3 billion a year to the economy whilst Tasmania supplies half of all Australian exports of woodchips, newsprint and writing paper, worth half a billion Australian dollars a year to the Tasmanian economy.[5] Historically the mining and agricultural industries have contributed, through their practices, to the contamination of both soil and water in Tasmania. However, the more recent increase in eucalypt plantation forests, particularly in the northeast of the state, has, through its reliance on pesticides to protect seedlings and trees, substantially added to the contamination problem. In plantation forests there is also an increased need for aerial application, thus dispersing chemicals over a much wider area at greater heights,

[4] Tasmanian government, Department of Treasury and Finance, 2013, Population. Available at: http://www.treasury.tas.gov.au/domino/dtf/dtf.nsf/LookupFiles/Population.pdf/$file/Population.pdf last accessed 2 December 2013

[5] Top 10 contributors to the Tasmanian economy. Available at: http://www.tasmaniatopten.com/lists/economic_contributors.php last accessed 2 January 2013

with an increased potential for drift to non-target areas.[6] There is also a higher maximum allowable rate for chemicals on plantations – e.g. atrazine: (8kg/hectare) compared to crops (2.5 kg/hectare).[7] Whilst it is acknowledged that both mining and agriculture may have historically contributed to environmental contamination in Tasmania, it is the more recent and substantial increase in plantation forests and their reliance on chemicals that is the focus here. This more narrow focus is also in response to the *Scammell Report,* which made a correlation in time and space between the increase in plantation forests, an increase in oyster health problems and the spread of devil cancer.

Public pressure to conserve native and old-growth forests, and the implementation in 1997 of *Plantations for Australia: The 2020 Vision,* has driven the ever-expanding plantation forest estate in Tasmania.[8] Gunns Limited, the largest forest products company in Australia, has alone developed over 200,000 hectares of plantations in Tasmania over the last 25 years.[9] A more recent driver of plantation forests was the plan to build a $2 billion pulp mill in the north of the state. The then Tasmanian Premier Paul Lennon engaged in undisclosed talks with Gunns Limited for its construction in 2003.[10] It was to be the largest in the southern hemisphere and would have relied predominantly on plantation timber.[11] [12]

---

[6] Primary Industries Standing Committee, 2002, *Spray Drift Management, Principles. Strategies and Supporting Information*, CSIRO Publishing, Collingwood Victoria. Available at: http://www.publish.csiro.au/Books/download.cfm?ID=3452 last accessed 2 January 2013

[7] Jenkin BM & Tomkins B, 2006, *Pesticides in Plantations*, Forest and Wood Products Research and Development Corporation, Melbourne, Victoria, Australia

[8] Planatations 2020, *Plantations for Australia: The 2020 Vision*. Available at: http://www.plantations2020.com.au/vision/ last accessed 6 May 2013

[9] Gunns Limited, About Gunns. Available at: http://gunns.com.au/about-us/ last accessed 5 October 2013

[10] *ABC News*, 2012, Timeline: The rise and fall of Gunns. Available at: http://www.abc.net.au/news/2012-09-25/gunns-timber-company-rise-fall-timeline/4235708 last accessed 6 January 2013

[11] Gunns Limited has since gone into receivership and administrators have been appointed. *ABC News*, 2012, Timeline: The rise and fall of Gunns. Available at: http://www.abc.net.au/news/2012-09-

Plantation forests are now located in 44 of the 48 river water catchments in Tasmania.[13] These plantations are monocultures of eucalypts, which rely on synthetic fertilizers and pesticides to maintain high yields.[14] Establishing the eucalypt plantations is dependent on the use of poisons to control browsing mammals, herbicides to control weeds, fungicides to control pathogens and insecticides to control insect attack.[15] Chemicals used in Tasmanian plantation forests are included in an 18-page list of products registered by the national regulator, the Australian Pesticides and Veterinary Medicines Authority (APVMA).[16] However even this extensive list omitted terbuthylazine, fluazifop and 1080, all known to be used in Tasmanian plantation forests.[17] The chemical compound 1080 (sodium monofluoroacetate) is used as a poison in baits distributed in plantations to protect the eucalypt seedlings from browsing native animals. Although the Tasmanian devil lethal dose of 1080 is high compared to other native species, researcher Helen L Statham noted that marsupial carnivores are the first native

---

25/gunns-timber-company-rise-fall-timeline/4235708 last accessed 3 January 2013 The outcome for the proposed pulp mill, which was vehemently opposed by many Tasmanians, is currently unknown.

[12] In the Supreme Court of Victoria at Melbourne Commercial and Equity Division Commercial Court List G, 2012, In the matter of Gunns Plantations Limited. Available at: http://www.gunns.com.au/Content/uploads/documents/Court%20Orders%2019%20December%202012.pdf last accessed 2 January 2013

[13] Bendor M, Parr I & Goninon C, 2008, *The Tasmanian River Catchment Water Quality Initiative: The development and evaluation of a methodology for identify the nature and extent of chemical pesticide usage in Tasmanian river catchments,* Tasmania, Department of Primary Industries and Water, Hobart, Tasmania

[14] Altieri MA, nd. Modern Agriculture: Ecological impacts and the possibilities for truly sustainable farming. Available at: http://nature.berkeley.edu/~miguel-alt/modern_agriculture.html last accessed 10 May 2013

[15] Green G, 2004, *Plantation Forestry in Tasmania*, Timber Workers for Forests. Available at: http://www.twff.com.au/documents/research/pftpt4.pdf last accessed 2 January 2013

[16] Australian Government Senate Rural and Regional Affairs and Transport Legislation Committee, Answers to Questions on Notice, Budget Estimates May 2009, Agriculture, Fisheries and Forestry, Australian Pesticides and Veterinary Medicines Authority, Response to Question on Notice, Question: APVMA06 Attachment 1, *Hansard*. Australian Government Senate, Canberra.

[17] ibid.

species to show signs of 1080 poisoning.[18] The long-term effects of the poison on Tasmanian devils have not been studied.

Other chemicals designed to kill target species are also known to cause harm, such as endocrine disruption and cancer, to non-target species. Chemicals of particular concern include the triazine herbicides - atrazine, simazine and terbuthylazine - and the chemical paraquat, all used to kill weeds. Atrazine is a known endocrine disrupter in frogs[19] and a suspected carcinogen in humans.[20] Simazine and terbuthylazine, with almost identical chemical structures to atrazine, are suspected of having the same harmful effects although these suspicions are supported by fewer studies.[21] The US EPA in a report on triazine cumulative risk, grouped atrazine, simazine, propazine and the metabolites desethyl-s-atrazine (DEA), desisopropyl-s-atrazine (DIA) and diaminochlorotriazine (DACT) as a group of chemicals with a common mechanism of toxicity i.e. 'they act in the same way in the body – that is, the same toxic effect occurs in the same organ or tissue by essentially the same sequence of major biochemical events'.[22] This assessment is based on their ability to cause neuroendocrine and endocrine-related developmental, reproductive and carcinogenic effects. Paraquat, on the other hand, is acknowledged as the cause of serious ill health and even death in humans.[23] In November 2012 an

---

[18] Statham HL, 1996, *Impact of 1080 on non-target species and priorities for research.* A report to the Browsing Animal Research Council, Hobart, Tasmania

[19] Hayes TB, Haston K, Tsui M, Hoang A, Haeffele C & Vonk A, 2003. "Atrazine-induced Hermaphroditism at 0.1ppb in American Frogs (Rana pipiens): Laboratory and field evidence." *Environmental Health Perspectives* 111(4), pp 568-576

[20] MacLennan PA, E Delzell, N Sathiakumar, SL Myers, H Cheng, W Grizzle, VW Chen & Wu XC, 2002, Cancer Incidence Among Triazine Herbicide Manufacturing Workers, *Journal of Occupational and Environmental Medicine* 44(11), pp 1048-1058

[21] US Environmental Protection Agency, 2006, Pesticides: Health and Safety 2006, *Triazine Cumulative Risk Assessment and Atrazine, Simazine and Propazine Decisions; June 22, 2006*, Available at http://www.epa.gov/oppsrrd1/cumulative/triazine_fs.htm last accessed 7 March 2010

[22] US Environmental Protection Agency Office of Pesticides Programs Health Effects Division, 2006, *Cumulative Risk from Triazine Pesticides*, US EPA, Washington, DC, p 2

[23] Madeley J, 2002, *Paraquat - Syngenta's controversial herbicide*. Available at http://www.evb.ch/en/p1300.html last accessed 29 August 2009.

Australian farmer died as a consequence of being sprayed with paraquat.[24] Both atrazine and paraquat have been the focus of heated controversies between the manufacturer Syngenta and those who seek to minimise harm.

The consequence of this widespread use of chemicals in plantations has meant the implementation by DPIPWE of the Pesticide Water Monitoring Program, which tests for 16 pesticides at 47 sites every two months.[25] Results of the findings are also published every two months. The first evidence of the potential for pesticides to contaminate surface water in Tasmania was made in the findings of a study led by Professor Peter Davies from the University of Tasmania in 1994.[26] The authors found that between 1989 and 1992, 20 of the sampled 29 streams draining plantation forests contained detectable residues of the chemicals atrazine and simazine. Supporting these findings is the report *Pesticide Use in Australia,* which states that streams draining forestry land generally contain more pesticides than agricultural streams. [27] Contamination of surface and drinking water in Tasmania is ongoing with four pesticides detected in the latest survey.[28]

---

[24] McKenna K, 2012, Lifelong farmer dies from toxic weedkiller, *The Courier-Mail,* 16 November 2012, p9

[25] Tasmanian Government Department of Primary Industries, Parks, Water and the Environment, Water, Pesticide Monitoring. Available at: http://dpipwe.tas.gov.au/water/water-monitoring-and-assessment/pesticide-monitoring last accessed 23 April 2014

[26] Davies PE, Cook LSJ & Barton JL, 1994, Triazine Herbicide Contamination of Tasmanian Streams: Sources, Concentrations and Effects on Biota, *Australian Journal of Marine and Freshwater Resources* Vol 45, pp 209-226

[27] Radcliffe JC, 2002, *Pesticide Use in Australia*. Australian Academy of Technological Sciences and Engineering, Parkville, Victoria

[28] Tasmanian Government Department of Primary Industries, Parks, Water and the Environment, Water, Latest Pesticide Water Monitoring Results. Available at: http://dpipwe.tas.gov.au/water/water-monitoring-and-assessment/pesticide-monitoring/pesticide-water-monitoring-program/aschem-latest-results last accessed 23 March 2014

Chemicals used in Tasmanian plantation forests are registered for use by the APVMA, which also determines the use label. However, it is the responsibility of state governments to monitor and regulate chemical use, and in Tasmania this is the role of DPIPWE. Dr Marcus Scammell, marine ecologist, who investigated the contamination in Georges Bay (described in more detail in Chapter 9), in an interview with the *Australian Broadcasting Commission's (ABC's)* reporter Jocelyn Nettlefold, suggested that water contamination is perhaps the main way animals absorb chemicals.[29]

Reports of water contamination continue unabated in Tasmania and it is still a critical public and environmental health issue.[30] Therefore, it would be appropriate for scientific research to be undertaken into the potential effects chemicals used in the environment have on Tasmanian devils. This would seek to determine if one or more chemicals, acting singularly or in synergy, are involved in the aetiology of the cancer. A full analysis of the regulation and use of chemicals in forestry plantations is given in Chapter 9. The potential for non-target species, such as the Tasmanian devil, to be harmed by environmental contaminants is discussed in the next section.

5.3 Possible toxic impacts on wild and captive Tasmanian devils

According to the allograft hypothesis, devils in captivity that are isolated from the contagious cancer transmitted through biting would be less susceptible to DFTD than wild devils. Alternatively, it could also be proposed that devils in the wild compared to

[29] *7.30 Report*, 2004, *Australian Broadcasting Corporation*, Sydney, 19 July 2004. Available at www.abc.net.au/7.30/content/2004/s1157381.htm last accessed 14 August 2007

[30] Tasmanian Government Department of Primary Industries, Parks, Water and the Environment, Water, Latest Pesticide Water Monitoring Results. Available at http://dpipwe.tas.gov.au/water/water-monitoring-and-assessment/pesticide-monitoring/pesticide-water-monitoring-program/aschem-latest-results last accessed 23 April 2014

captive devils, are potentially more vulnerable to toxic chemicals, through contamination of the water they drink, the food they consume and more directly from agricultural and plantation forestry chemical spraying practices. The DPIPWE project to develop a captive breeding program on mainland Australia is another indication that devils may be safer removed from the Tasmanian environment. In fact DPIPWE actively use methods to avoid environmental toxins coming into contact with their captive devils. In one study by Kreiss et al it was noted that devils kept in captivity were 'fed once a day with road-killed wallabies or possums from non-diseased areas'.[31] Other captive devils' food is sourced as frozen meat from the northwest non-diseased areas.[32] Obviously careful measures are taken by DPIPWE staff to maintain healthy captive devils. It could be inferred from these practices that they were concerned about the chemical contamination of the environment. These measures however have not completely protected captive devils.[33]

At the Riverside-based Tasmanian Zoo one devil from a group of eight, after 10 months at the Zoo, was found to have DFTD.[34] The devils had been raised by the *Devils in Danger Foundation*, part of a conservation program set up to help save the species. The DPIPWE had also experienced DFTD in their captive breeding devils at Cressy.[35] Devils in captivity at Trowunna Wildlife Park at Mole Creek also contracted DFTD. The cause of the DFTD at the first two locations has not been established but the cancer

[31] Kreiss A, Fox N, Bergfeld J, Quinn SJ, Pyecroft S & Woods GM, 2008, Assessment of cellular immune response of healthy and diseased Tasmanian devils (Sarcophilus harrisii), *Developmental and Comparative Immunology* Vol 32, pp 544-553 p 545
[32] Personnel communication.
[33] Dadson M, 2012, Blow to devil rescue plan, *The Examiner Newspaper*. Available at http://www.examiner.com.au/news/local/news/general/blow-to-devil-rescue-plan/2531554.aspx last accessed 1 May 2012
[34] ibid.
[35] Personal communication from staff at the Mt Pleasant laboratory.

in devils in captivity at Trowunna Wildlife Park would seem to point to an external source other than contact with a DFTD infected devil.

5.3.1 Devils at Trowunna Wildlife Park, Mole Creek, Tasmania

Androo Kelly, the owner and operator of the Trowunna Wildlife Park, has successfully bred captive devils for over 25 years. Since the outbreak of DFTD, however, Kelly has encountered the disease in his devils on six separate occasions. On the first occasion, May 2006, when a devil with DFTD was identified at the Park, it was proposed by Obendorf and McGlashan that it had escaped, encountered and was bitten by a DFTD infected devil and was later recaptured and subsequently developed the cancer[36] (as previously mentioned in Chapter 2). The suggestion by the DPIPWE was that the Park's perimeter fencing and devil pens were not secure.[37]

However, since the initial case four more devils contracted DFTD, the last in August 2007, but Kelly insisted following the first case he had secured his boundary fences and pens.[38] In 2009 Kindred a devil at the Park, shown being examined by a veterinary officer in Figure 5.1 below, was suspected of having DFTD. The tumour appears as a small red lump under the tongue. DFTD was later confirmed at the DPIPWE Mt Pleasant laboratory in Launceston.

[36] Obendorf DL & McGlashan ND, 2008, Research priorities in the Tasmanian devil facial tumour debate, *European Journal of Oncology*, Vol 13(4), pp 229-238
[37] *ABC News*, 2009, Biosecurity audit sought for devil parks. Available at: http://www.abc.net.au/news/2009-04-02/biosecurity-audit-sought-for-devil-parks/1638568 last accessed 12 October 2013
[38] Personal communication with Androo Kelly, 2 April 2009

Figure 5.1 Kindred's suspected DFTD tumour under the tongue[39]

In 2009 I accompanied the DPIPWE veterinary officer to the Park where devils displaying obesity and enlarged lymph glands were examined. Also reported on the visit were low offspring survival rates and the case of an intersex devil (having both male and female reproductive organs). These health problems, including the cases of DFTD, could possibly indicate affects from toxins in the environment, especially endocrine disruption. Plantation forests are within sight of the Park and the water to the Park is sourced from Mole Creek.[40]

In the Mole Creek and Chudleigh region there have been claims of major breaches of the *Forestry Practices Act*.[41] The Trowunna Wildlife Park is situated at Mole Creek below the Gog Range as shown in Mole Creek Drainage Map in Figure 5:2 below. The

[39] Scott L, 2009, Breakthrough test for devil facial tumour, *The Examiner*. Available at: http://www.examiner.com.au/story/496852/breakthrough-test-for-devil-facial-tumour/ last accessed 12 May 2013
[40] Personal communication with Androo Kelly, April 2009
[41] Godfrey, P, 2006, The Chudleigh Report: Complaint to Forest Practices Board of Breaches of Forest Practices Code of Tasmania, unpublished.

Trowunna Wildlife Park (located north of the Mole Creek Holiday Village on map) is downstream from plantation forests and within the vicinity of possible spray drift from aerial spraying of pesticides as shown in Figure 5:3 below.

Figure 5:2 Mole Creek drainage map

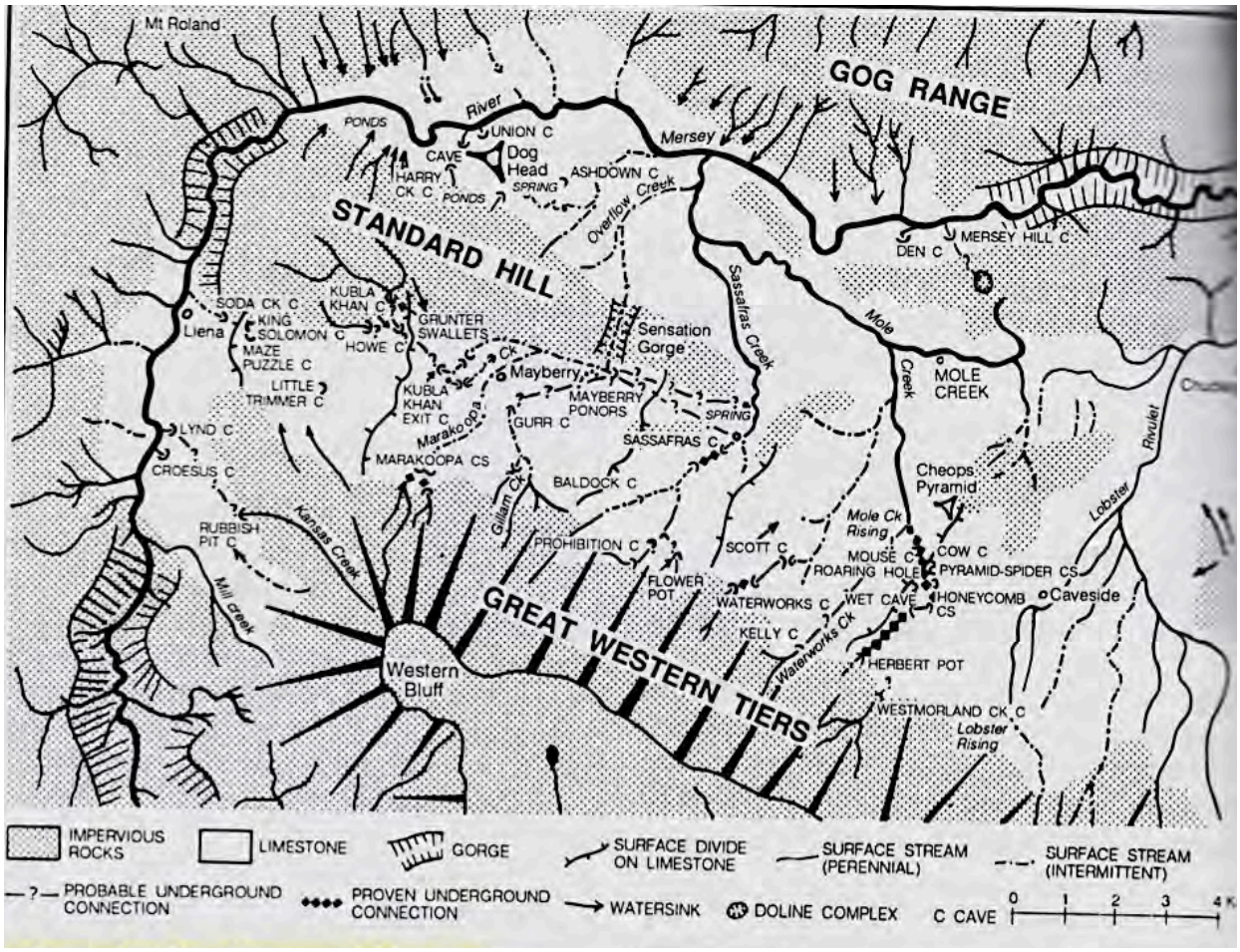
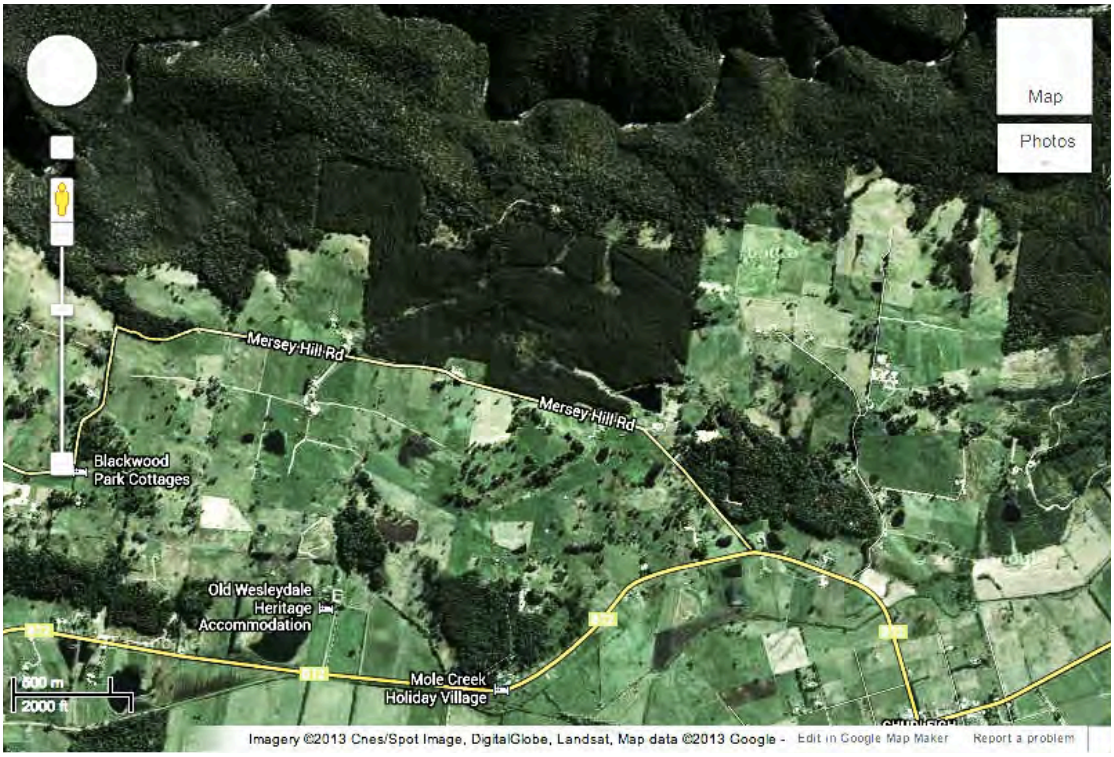


Figure 5:3 Map showing the location of Trowunna Wildlife Park (centre front) with plantations along Mersey Hill Road (centre)[42]



Further evidence of destructive practices is shown in the following images:

- Figure 5:4 below shows the results of cable logging on steep slopes.
- Figure 5:5 shows the results of Gunns Limited and Forestry Tasmania operations in clear felling a coupe[43] in the Gog Range.

[42] Google Maps Plantations. Available at: https://maps.google.com.au/maps?hl=en&q=Trowunna+wildlife+park+%2B+map&bav=on.2,or.r_cp.r_qf.&biw=1188&bih=649&um=1&ie=UTF-8&sa=N&tab=wl last accessed 1 July 2013

[43] Coupe – a small management area of a forest in which harvesting and forest regeneration may occur. Glossary, Department of Agriculture, Fisheries and Forestry. Available at: http://www.daff.gov.au/rfa/glossary last accessed 6 January 2013

Figure 5:4 Cable logging on steep slopes in the Gog Ranges

Figure 5:5 Logged slope with plantations in the middle ground

These practices lead to turbidity in local streams, loss of topsoil, habitat destruction for native wildlife and loss of biodiversity. Combined with the use of pesticides, which leads to contamination of surface and ground water and the hazards of aerial spraying to non-target species, the need for a full investigation of the role of environmental toxins in the devil cancer DFTD, especially at the Park, would seem warranted.

5.4 Support for toxicology studies

In February 2005 the DPIPWE released the *Tasmanian Devil Facial Tumour Disease (DFTD) Disease Management Strategy,* which reported a consensus amongst the researchers that the cancer was a neuro-endocrine tumour of unknown origin.[44] In the same year DPIPWE published a *Progress Report* identifying key areas for investigation, the relevant fields being: haematology; blood biochemistry; immunology; endocrinology; and the identification of the aetiology (cause) of the disease.[45] A viral aetiology was discounted because a test for virus particles had proved negative but a trial to test for a range of chemical toxins was proposed.

In 2006 the novel hypothesis that the devil cancer is a transmissible tumour, an allograft, based on cytogenic research by Anne Maree Pearse conducted at the Tasmanian Government DPIPWE Mt Pleasant laboratory in Launceston, was proposed. This hypothesis was proposed prior to undertaking the toxicology studies.

---

[44] Cited in Loh RC, 2006, *The Pathology of Devil Facial Tumour Disease in Tasmanian Devils (Sarcophilus harrisii)*, Master of Philosophy, Murdoch University, Perth, Western Australia

[45] Tasmanian Government Department of Primary Industries, Water and the Environment, 2005, *Research into the Tasmanian Devil Facial Tumour Disease (DFTD) Progress Report,* Department of Primary Industry, Water and Environment, Hobart, Tasmania

The need for toxicology studies to determine the possible role of a carcinogen in the aetiology of the cancer has been identified on a number of occasions:

- the initial DPIPWE *Progress Report* see previous page;
- Pearse and Swift in their article in *Nature*[46];
- David Obendorf and Neil McGlashan (see p 156);
- Vetter et al paper following the pilot study (see next section); and
- Professor Michael Moore and Dr Tony Ross in reviews of the results of the pilot study (see section 5.6 below).

To date comprehensive studies into the role of an environmental toxin acting as a carcinogen have not been completed or published. My analysis of the published scientific research into the devil cancer, discussed in Chapter 2, revealed only one paper that published by Vetter *et al*.[47] In this chapter I analyse the research leading to the publication of that paper.

5.5 Toxicology studies into DFTD

In 2004 a National Dioxins Program accessed the concentrations of PCDD/PCDFs and PCBs in Australian fauna but it did not include Tasmanian devils amongst the marsupials studied.[48] In the same year Robert Symons and colleagues from the Australian Government Analytical Laboratories (AGAL) published figures on levels of brominated flame retardants, in particular polybrominated diphenyl ethers (PBDEs) in Australian fauna.[49] It reported detectable levels of PBDEs in all eight Tasmanian devils studied.[50] The Tasmanian devil samples had been supplied by Dr Menna Jones.

---

[46] Pearse AM & Swift K, 2006, Transmission of devil-facial-tumour disease, *Nature*, Vol.439(2), p 549
[47] Vetter W, Recke R von der, Symons R & Pyecroft S, 2008, Determination of polybrominated biphenyls in Tasmanian devils (Sarcophilus harrisii) by gas chromatography coupled to electron capture negative ion tandem mass spectrometry or electron ionization high-resolution mass spectrometry, *Rapid Communications in Mass Spectrometry*, Vol 22, pp 4165-4170
[48] Correll R, Muller J, Ellis D, Prange J, Gaus C, Shaw M, Holt E, Bauer U, Symons R & Burniston D, 2004, *Dioxins in Fauna in Australia, National Dioxins Program Technical Report No. 7*, Australian Government Department of the Environment and Heritage, Canberra
[49] Symons R, Burniston N, Piro N, Stevenson G & Yates A, 2004, A study of the presence of brominated flame retardants in Australian fauna, *Organohalogen Compounds* Vol 66, pp 3959-3965.
[50] Jones, M (unpublished) in T Ross, 2008, *Persistent Chemicals in Tasmanian Devils,* DPIW, Hobart, accessed 17 August 2009 http://tassiedevil.com.au/research.html

In 2005 the DPIPWE *Progress Report* identified as necessary the establishment of a pilot study of a statistically valid number of tissue samples to test for a range of toxins to determine the aetiology of the devil disease.[51] The *Progress Report* also recommended that following the pilot study, normal devil cell cultures should be exposed to ten of the most commonly isolated toxins in amounts similar to those found in affected devils. Positive effects of the toxins on the cell cultures would indicate a need for a much larger project.

In April 2007 Simon Bevilacqua, a journalist with the *Sunday Tasmanian,* in an email dated 23 April, requested information about the toxicology studies for an article he wished to publish. Despite the 2004 results and the acknowledged need for toxicology studies, pilot studies had still not commenced. In the following month, prompted by Bevilacqua's request, devil tissue was sent for toxicological analysis. The samples from 8 diseased devils and 8 non-diseased devils were sent from the DPIPWE Mt Pleasant laboratory to three separate laboratories. All the laboratories were accredited through the National Association of Technical Authorities (NATA), a private body, which is Australia's government-endorsed national authority. At the time Stephen Pyecroft, Principal Veterinary Pathologist at the DPIPWE Mt Pleasant laboratory, was also on the NATA Veterinary Testing Accreditation Advisory Committee.[52] The laboratories included the National Measurement Institute (NMI) in Sydney, the Alan Fletcher Research Station in Brisbane Queensland and Analytical Services Tasmania (AST) in Hobart.

---

[51] Tasmanian Government Department of Primary Industries, Water and the Environment, 2005, *Research into the Tasmanian Devil Facial Tumour Disease (DFTD) Progress Report,* Department of Primary Industry, Water and Environment, Hobart, Tasmania

[52] Bailey N, 2007, Veterinary Testing, *NATA News*, Issue 125, p 29. Available at: http://www.nata.com.au/phocadownload/publications/Annualreport_newsletter/Newsletter/NN_Sept07rev2.pdf last accessed 6 January 2013

Whilst full results of these studies have never been published, Matthew Denholm of *The Australian* newspaper did obtained the results through a Freedom of Information request. A limited version is now available on a *SourceWatch* website.[53] The NMI results were published by Vetter *el al* in the journal *Rapid Communications in Mass Spectrometry* in 2008, the only paper reporting the results of the studies.[54] The results from the other laboratories were not published. There were however two official reviews of the results given by qualified scientists and published on the *Save the Tasmanian Devil* website, which are discussed below. The results from the various laboratories are summarized in Table 5.1 below.

Table 5.1 Results of toxicology studies

Laboratory	Chemicals tested	Date of Study	Conclusions
National Measurement Institute (NMI)	Dioxins – PCDD/PCDF, PAHs, PBDEs, organic pollutants, PBBs - fat samples	May 2007	Need for more studies into the reasonable levels of PBB residues (flame retardants) in devil samples.[55]
Alan Fletcher Research Station	Sodium Fluoroacetate (1080) poison	May 2007	1080 residue not detected in any devil samples
Analytical Services Tasmania (AST)	Inorganic (arsenic, lead and mercury),Organo-chlorines & metabolites,Organo-phosphates,Triazine herbicides (atrazine and simazine) – liver samples	May 2007	Inorganic analysis (arsenic, lead, mercury) - less than 1ppm detected,Organo-chlorines & metabolites - one devil above detection range (limit <0.20 ppb),Organo-phosphates and triazine herbicides (atrazine and simazine) – not detected

[53] Water Pollution in Tasmania published a limited version of the toxicology results. Available at: http://www.sourcewatch.org/index.php?title=Water_pollution_in_Tasmania last accessed 29 August 2009.
[54] Vetter W, Recke R von der, Symons R & Pyecroft S, 2008, Determination of polybrominated biphenyls in Tasmanian devils (Sarcophilus harrisii) by gas chromatography coupled to electron capture negative ion tandem mass spectrometry or electron ionization high-resolution mass spectrometry, *Rapid Communications in Mass Spectrometry*, Vol 22, pp 4165-4170
[55] There was no significant difference between the levels of toxins found in diseased and non-diseased devils. Concerns raised over devil disease findings, *ABC News*, 22 January 2008. Available at: http://www.abc.net.au/news/2008-01-22/concerns-raised-over-devil-disease-findings/1019328 last accessed 1 July 2013

5.5.1 The National Measurement Institute (NMI) and dioxin testing

The NMI is the institute responsible for Australia's national infrastructure in analytical, biological, chemical and physical measurements. The NMI has the capability of carrying out what it terms on its website as 'environmental analysis' into dioxins, organic pollutants, pesticide contaminants as well as metal pollutants, microbiological contaminants and water analysis.[56] Devil samples sent to the NMI were to be tested for a limited range of chemicals. The tests requested by DPIPWE to be carried out were for dioxins (PCDD/PCDF in I-TEQ, USEPA method 1668A – Isotype dilution), polycyclic aromatic hydrocarbons (PAHs) (indicator benzio-a-pyrene PBDEs), and polybrominated biphenyls (PBBs).[57] Symons, who would be conducting the analyses at NMI, had arranged for co-authorship of the results, which were reported in the paper by Vetter *et al* in *Rapid Communications in Mass Spectrometry* in September 2008.[58] Interestingly, the paper claimed that the Tasmanian devils were endangered due to a virus epidemic.

The results found concentrations of PBB153 in the range 0.3-11ng/g lipids in all but two devil samples. There was no significant difference between healthy and diseased devils. Levels were significantly lower than those causing toxic effect but 'PBB concentrations were one level or even higher than PBDEs' found in the National Dioxin Program 2004 study by Symons and colleagues.[59] The paper also highlighted the need

---

[56] Australian Government National Measurement Institute. nd, *Environmental Testing*. Available at: http://www.measurement.gov.au/Services/EnvironmentalTesting/Pages/default.aspx last accessed 7 September 2011

[57] Email from DPIPWE to NMI, dated 11 April 2007

[58] Vetter W, Recke R von der, Symons R & Pyecroft S, 2008, Determination of polybrominated biphenyls in Tasmanian devils (Sarcophilus harrisii) by gas chromaography coupled to electron capture negative ion tandem mass spectrometry or electron ionization high-resolution mass spectrometry, *Rapid Communications in Mass Spectrometry*, Vol 22, pp 4165-4170

[59] ibid, 2008:4165

for more detailed environmental PBB residue studies in devils.[60] PBBs have been shown to cause cancer in rats and the International Agency for Research on Cancer (IARC) has determined that PBBs are possibly carcinogenic to humans.[61]

The impact of these results is made clear in comments by Mariann Lloyd-Smith, co-chair of the International Persistent Organic Pollutants Elimination Network who stated "[w]e were quite shocked" and she suggested that "[c]ertainly this study will have ramifications".[62] She further stated '[a]lthough the sample of the recent study was too small for firm conclusions …the toxins weakened the immune system and might theoretically be a factor in the disease that threatens to wipe out the Tasmanian devil.'[63] Despite the concerns raised by Lloyd-Smith no further studies into the dioxins found in devil tissues were undertaken. The only peer-reviewed publication following the toxicology studies was a paper by Vetter at el, which only covered the results of the dioxin studies at the NMI. The authors claimed that 'the contamination status of Tasmanian devils with anthropogenic pollutants was investigated'.[64] However, support for this statement relied on a newspaper article, which does not make reference to pollutants, and the DPIPWE website where the link is broken. Other discrepancies in citation also occurred.

---

[60] Vetter W, Recke R von der, Symons R & Pyecroft S, 2008, Determination of polybrominated biphenyls in Tasmanian devils (Sarcophilus harrisii) by gas chromaography coupled to electron capture negative ion tandem mass spectrometry or electron ionization high-resolution mass spectrometry, *Rapid Communications in Mass Spectrometry*, Vol 22, pp 4165-4170

[61] US Environmental Protection Agency, 2010, *Emerging Contaminants – Polybrominated Diphenyl Ethers (PBDEs) and Polybrominated Biphenyls (PBBs),* Available at: http://www.epa.gov/fedfac/documents/emerging_contaminant_pbde_pbb.pdf last accessed 7 September 2011

[62] *Cosmos Magazine Online,* 2008 Toxic chemicals: no link to devil facial tumours, Cosmos Media Pty Ltd. Available at http://www.cosmosmagazine.com/news/1817/toxic-chemicals-no-link-devil-facial-tumours last accessed 2 February 2010

[63] ibid.

[64] : Vetter W, Recke R von der, Symons R & Pyecroft S, 2008, Determination of polybrominated biphenyls in Tasmanian devils (Sarcophilus harrisii) by gas chromaography coupled to electron capture negative ion tandem mass spectrometry or electron ionization high-resolution mass spectrometry, *Rapid Communications in Mass Spectrometry*, Vol 22, pp 4165-4170, p 4166

McGlashan et al's published paper documenting evidence of the need for an investigation into the possibility of a toxin-related aetiology from human land-use activities in Tasmania was not cited in the Vetter et al paper.[65] Likewise, in 2007 in the journal *EcoHealth* Stephen Pyecroft, a co-author of the Vetter et al paper, in charge of the DPIPWE laboratory in Launceston, and on a NATA committee, also failed to cite the McGlashan et al publication.[66] The reason for the omission of the McGlashan et al article is not known, but it is compatible with a chilling effect as described in Chapter 2.

5.5.2 Alan Fletcher Research Station – Sodium Fluoroacetate (1080) testing

Tasmanian devil liver samples were sent from the DPIPWE laboratory in Launceston to Robert Parker at the Alan Fletcher Research Station (AFRS) in Sherwood, Queensland for 1080 analysis. Parker had requested stomach content, liver and kidney as preferred samples.[67] He specifically asked for the largest samples and specified that 'with an old sample, you have degradation and contamination. These factors will reduce the effectiveness of the test'.[68] Some of the samples sent to NMI had been stored since 2003 and it is probable that some of these same samples were sent to AFRS. In Australia there is no maximum residue limit (MRL) set for 1080 according to the APVMA.[69] The results of the tissue samples indicated 1080 was not detected. This is not unexpected as

---

[65] McGlashan, Neil D, DL Obendorf and JS Harington. 2006. "Aspects of the fatal malignant disease among the Tasmanian devil population (*Sarcophilus laniarius*)." *European Journal of Oncology* 11(2):95-102

[66] Pyecroft SB, Pearse AM, Loh R, Swift K, Belov K, Fox N, Noonan E, Hayes D, Hyatt A, Wang L, Boyle D, Church J, Middleton D & Moore R, 2007, Towards a Case Definition for Devil Facial Tumour Disease: What Is It? *EcoHealth* Vol 4(3), pp 346-351

[67] Email communication from Alan Fletcher Research Station to DPIWE Mt Pleasant Laboratory dated 23 May 2007

[68] ibid.

[69] Australian Government Australian Pesticides and Veterinary Medicines Authority, 2008, Sodium Fluoroacetate Final Review Report and Regulatory Decision. Available at: http://www.apvma.gov.au/products/review/docs/1080_final_review_report.pdf last accessed 6 September 2011

the tendency for 1080 is not to accumulate in tissue post exposure.[70] There is no published report on the analysis undertaken at this laboratory.

5.5.3 Analytical Services Tasmania (AST) – testing of agrichemicals used in Tasmania

The critical analysis on the devil tissues for agrichemicals used in plantation forests was undertaken by the Tasmanian government DPIPWE operated AST laboratory. At the time the DPIPWE was also in charge of:

- monitoring chemicals used in forestry;
- analyses carried out by AST;
- funding the devil research through UTAS; and
- analyses of chemical residue in the devil tissues.

A conflict of interest would seem to be apparent when the body charged with enabling the progress of the forestry industry, DPIPWE, is also charged with monitoring chemicals in the environment and assessing chemical residue in devil tissues used by that industry.

It is likely that samples similar to those sent to the other laboratories were also sent to AST. The AST is an accredited NATA laboratory for the testing of chemicals but only in water and sediment, not in biological samples such as devil tissue.[71] The analyses at AST were for endocrine disrupters, such as atrazine, which are usually detected in urine.[72] It is also known that there are critical times in the development of an organism when these chemicals cause the most damage with effects not manifest until later in life

---

[70] Twigg LE, Lowe TJ, Kirkpatrick WE & Martin GR, 2003, Tissue residue levels in rabbits and rats poisoned with 1080 One-shot bait and the location of poisoned rabbit carcasses, *Wildlife Research* Vol 30, pp 621-631

[71] Personal communication with National Association of Testing Authorities (NATA), Brisbane, Queensland dated 19 May 2009

[72] Zhou Z, Jin M, Ding J, Zhou Y, Zheng J & Chen H., 2007, Rapid detection of atrazine and its metabolite in raw urine by exractive electrospray ionization mass spectrometry, *Biomedical and Life Sciences* 3(2), pp 101-104

and in some instances in the next generation.[73] Therefore, non-detection of endocrine disrupting chemicals is not necessarily an indicator of lack of harm. The results of the tests from AST were that agrichemicals, including the triazines (atrazine/simazine), were undetected. A consequence of the chemicals being undetected is that further scientific experiments on the effects of these chemicals on devils, has been left undone.

It would also appear that through a lack of appropriate studies the AST avoided producing 'negative knowledge', namely scientific results which may have proved harmful to vested interests or those funding the research. Atrazine and its metabolites enter some organs or fat but do not build up or remain in the body, usually leaving through the urine within 24-48 hours.[74] They are absorbed from the gastro-intestinal tract with the highest concentrations usually detected in red blood cells.[75] No testing was done of these chemicals in either blood or urine of the devils.

Further studies into the role of these chemicals and endocrinology studies, identified in the DPIPWE report in 2005, should not be avoided or abandoned simply because these limited tests resulted in non-detection. When tests for chemicals that are known endocrine disrupters, such as the triazines, atrazine and simazine, come up negative scientists then must decide whether further studies are warranted. This raises further questions - are there limits to detection, that is, is the science undoable?

5.6 Scientific opinions on the toxicology results

The details of the chemical testing, carried out on devil tissues at the various laboratories, were not made public in Australia. There were however, two opinions that

[73] Myer, P & Hessler.W, 2007, Does the dose make the poison? *Environmental Health News*. Available at http://www.endocrinedisruption.com/endocrine.introduction.related.php last accessed 16 August 2009
[74] Pathak RK & Dikshit AK, 2011, Atrazine and Human Health, *International Journal of Ecosystem*, 1(1), pp 14-23
[75] ibid.

appeared on 27 February 2008 on the *Save the Tasmanian Devil* website, a joint initiative of the Tasmanian Government and the University of Tasmania (UTAS).[76] Professor Michael Moore from the University of Queensland provided a letter giving his opinion,[77] whilst Dr Tony Ross, a Veterinary Pathologist from Tasmania, provided a report.

Professor Moore's response raised concerns about the levels of concentration of PCDDs and polybrominated diphenyl ethers (PBDEs) in Tasmanian devils.[78] Although Moore admitted that the devil numbers tested were too low to be significant he recommended that they warranted further study. He acknowledged that these chemicals are known for suppression of immune function and perpetuation of cancerous cell lines. In relation to the dioxin studies, undertaken at NMI, he stated,

> The evaluation of the difference that might occur between these various measures have been divided into those animals that have been found to have cancer and those who did not have cancer. I have tried to establish whether there are any reasonable geographic associations but have been limited because of the lack of detailed information on likely environmental exposures of the animals who did and did not have cancer. Again the numbers are too small.[79]

In conclusion he stated,

> [i]t is now 12 years since the disease was first detected in 1996 in north-east Tasmania. There are no specific unusual characteristics in that region which would account for excessive exposure to any specific chemicals.

Moore appears unaware of the *Scammell Report* of 2003, which although it had not mentioned any specific chemicals, had made a correlation in time and space between

---

[76] Links to the Professor Moore and Dr. Ross letters were published on the Save the Tasmanian Devil website. Available at: http://www.tassiedevil.com.au/tasdevil.nsf/TheDisease/01E084030D8DE533CA2576D200176CC3 last accessed 7 September 2011

[77] Letter from Professor Michael Moore to Professor Hamish McCallum, University of Tasmania dated 27 February 2008, Opinion on a chemical aetiology for Facial tumour development in the Tasmanian devil

[78] Moore M, 2008, Letter to Professor Hamish McCallum dated 27 February 2008.

[79] Ibid.

the devil disease and the increase in plantation forests and their use of chemicals as a possible aetiology of the devil disease.

5.7 Practical limitations or political influence?

All three laboratories are federal or state government bodies, operating under government departments, the main role and responsibilities of which are to support industry or agriculture. The NMI is a division within the Australian Government, Department of Industry, Innovation, Climate Change, Science, Research and Tertiary Education, operating under the *National Measurement Act 1960.* The Alan Fletcher Research Station (closed in 2011) operated under the Queensland Government Department of Employment, Economic Development and Innovation and Queensland Department of Agriculture, Fisheries and Forestry.[80] These laboratories carried out the testing on devil samples for dioxins and 1080. Meanwhile AST, the laboratory that undertook the critical studies on pesticides used in forestry and agriculture in Tasmania, is a Tasmanian government laboratory located in the Chemistry Department of UTAS. It has close collaborations with UTAS and DPIPWE and carries out testing for the Government and the forestry industry including the largest plantation forestry operator, Gunns Limited. All the samples were selected and sent by the Tasmanian DPIPWE Mt Pleasant laboratory in Launceston.

UTAS and DPIPWE work in close collaboration on the Tasmanian devil DFTD project, controlling both the funding and scientific research into the Tasmanian devil disease. UTAS as a research and educational institution receives substantial funding from both the Tasmanian government and the forestry industry. DPIPWE controls the use of

[80] Owen C, 2011, What will become of the Alan Fletcher Station? Available at: http://www.thesatellite.com.au/news/what-will-become-of-the-alan-fletcher-station/850385/ last accessed 8 May 2013

chemicals, the monitoring of water and manages the Threatened Species Unit. At the time, the Tasmanian government minister presiding over DPIPWE was also the minister for Department of Industry, Energy and Resources (DIER), which regulates Forestry Tasmania, the Tasmanian Regional Forest Agreement and the Forestry Practices Code.[81] This situation still exists: Bryan Green, the Minister for Primary Industries and Water (under DPIPWE), is also the Minister for Energy and Resources (under DIER) although there are now multiple ministers overseeing other portfolios within these departments. In Tasmania, the DPIPWE, UTAS and the forestry industry form what Hess describes as the 'elites', those with the power and funding to control the research program.

It would appear that practical limitations are not the reason for a lack of further studies. Limits to detection and the lack of statistically significant numbers of samples were acknowledged as not insurmountable barriers. There was also no indication that the studies were undoable. It is therefore likely that political influence or the avoidance of negative knowledge, especially in relation to the tests undertaken at AST, the Tasmanian facility, provides a valid reason for the abandonment of the toxicology studies.

5.8 Conclusion

The Tasmanian economy relies heavily on the forestry industry's ability to continue its operations unimpeded. If toxicology findings revealed that chemicals used in plantation forests were responsible for the devil cancer, this would be devastating not least for the forestry industry, but also the Tasmanian government, which depends on forestry jobs and votes, and also the chemical industry which depends on profits from the sale of chemicals used in plantations. Syngenta, the biggest agrichemical company in the world

[81] DPIPWE. Bryan Green is minister for Primary Industries and Water and Brian Wightman for Environment, Parks and Heritage. Bryan Green is also Minister for Energy and Resources under DIER.

and the manufacturer of both atrazine and paraquat, both at the centre of international controversies and banned in the EU, would risk substantial harm to its image as an environmentally responsible company if these chemicals were linked to the devil cancer. All three powerful elites would consider adverse toxicology results to be 'negative knowledge'. It is in their interests that further toxicological studies to investigate the role of environmental toxins in DFTD remain undone. It is also possible that the political and economic fallout from adverse toxicological studies would not be lost on those involved in the research and it could be expected to have a 'chilling effect' on those making critical research decisions.

The limited scientific research into the Tasmanian devil disease DFTD has followed the research pathway determined by the allograft theory, that the cancer is transmissible. The research into a competing hypothesis, that an environmental toxin might play a role in initiating or progressing the devil cancer, remains under-examined. Initial toxicology study results revealed only PBBs in the devil fat tissues, all other tests proved negative, but no further studies have sought to expand or replicate these tests. There are no practical reasons such as ignorance or non-knowledge that would prevent further studies being undertaken. The necessary studies are routine toxicological analyses that are regularly and easily done in identifying environmental toxins in wildlife.

The DPIPWE commissioned toxicological analyses of the devil tissues were only revealed following a successful FOI application. A key paper linking the use of chemicals with the devil cancer was not cited.[82] The political controversy surrounding the continued contamination of surface and ground water, and the ever increasing

[82] Obendorf DL & McGlashan ND, 2008, Research priorities in the Tasmanian devil facial tumour debate, *European Journal of Oncology*, Vol 13(4), pp 229-238,

plantations and the use of chemicals, could also be contributing to self-censorship.[83] The Tasmanian devil may well become extinct before the aetiology of this cancer is established.

The stated aim of the DPIPWE Strategy had been to undertake a study of a statistically valid number of samples using a range of toxins to be followed up by a study of normal cell cultures to test ten of the most commonly isolated toxins. These studies have not been undertaken. There is sufficient evidence of harmful toxins in devil tissue, including the National Dioxin Study in 2003, which found brominated flame retardants, the NMI study which found PBBs, both known as probable human carcinogens and immune suppressors. Devils in captivity, isolated from wild devils, have on numerous occasions in different locations developed DFTD. Both Moore and Ross have proposed that more studies be undertaken as a result of the pilot study findings. It would appear however, that a conflict of interest exist within the DPIPWE when it is responsible for both the management of the use of chemicals used in plantation forestry and for the Save the Tasmanian devil Program.

The hypothesis that DFTD is a transmissible cancer spread from one devil to another still awaits conclusive studies, as has happened in CTVT, to demonstrate that the cancer is capable of being established in a new host. The studies examined in this chapter have shown that toxins capable of causing cancer and suppressing the immune system have been identified in devil tissue. The scientific research has not settled the question of how the devils became victims of this deadly cancer and the toxicology studies have only added to the uncertainty. In the next chapter I propose that due to this scientific

[83] Personal communication

uncertainty the Precautionary Principle be implemented to mitigate the harm being caused to the devils and that its core impact will be to trigger appropriate studies to further investigate the DFTD problem.

Chapter 6 – The precautionary principle

6.1 Introduction

The precautionary principle is a tool that enables decision makers to act in the face of scientific uncertainty. Given the evidence I have provided in the previous chapters I contend that the cause of the Tasmanian devil cancer is currently uncertain. According to the Tasmanian government and the Devil Facial Tumour Disease (DFTD) scientific research, it is an allograft, a contagious cell line transmitted via biting from devil to devil. In the previous chapters I have shown that this is not the only possible hypothesis to explain the cancer and that an alternative, toxins in the environment, is also a possible cause, albeit neglected. As a consequence of this uncertainty the precautionary principle should apply to enable further scientific research into all avenues of research. I also propose that it be implemented to restrict the use of triazine chemicals, in particular atrazine, in plantation forestry in Tasmania until a probability of no harm can be attained. In the following sections I outline the legal and legislative role of the precautionary principle in addressing scientific uncertainty and in the mitigation of irreversible environmental harm.

6.2 The precautionary principle

The precautionary principle is a legal and moral guideline for how private and public decision-makers should act when confronted with uncertainty, potential danger and the possibility of irreversible harm. It has evolved through environmental law and policy to address the need for better environmental management in the face of increasing

scientific uncertainty.[1] At its most simple it is a mechanism to prompt timely action when dealing with the harmful effects of human activities. However, there is wide debate surrounding how the principle should apply in practice as the following discussion will demonstrate.

The precautionary principle is an essential part of many international treaties and declarations and is becoming an important fundamental feature of international law.[2] Its adoption in 2000 as the core of the *Cartagena Protocol on Biosafety* would appear to be the most advanced expression of the principle so far in any international agreement.[3] In Article 10, paragraph 6 the *Protocol* states that 'lack of scientific certainty…shall not prevent [a] party from taking a decision, as appropriate…' in relation to living modified organisms and their potential risk to biodiversity. It establishes the precautionary principle as a feature of international environmental law and its treatment makes the dispute, that it is not a principle of customary international law, more difficult to maintain.[4]

The precautionary principle is a shift from traditional risk management, where risk assessment depends on the quantification of probabilities of cause and effect scenarios. The precautionary principle is a timely intervention undertaken not only before the effects are known but also seeks to avoid or diminish harmful effects.[5] The

---

[1] Harding R & Fisher E, 1999, 'Introducing the precautionary principle' in R Harding & E Fisher, (eds), *Perspectives on the Precautionary Principle*, The Federation Press, Leichhardt

[2] De Sadeleer N 2002, *Environmental Principles, From Political Slogans to legal Rules,* Oxford University Press, Oxford, p 97

[3] ibid, p 98

[4] Cosbey A & Burgiel S 2000, *The Cartagena Protocol on Biosafety: An analysis of result.* Available at: http://www.iisd.org/pdf/biosafety.pdf last accessed 22 February 2007

[5] United Nations Educational Scientific and Cultural Organization (UNESCO), Precautionary Principle Expert Group, World Commission on the Ethics of Scientific Knowledge and Technology (COMEST), 2005, *The Precautionary Principle*. Available at: http://unesdoc.unesco.org/images/0013/001395/139578e.pdf last accessed 4 November 2013

precautionary principle, as a shift from traditional risk management, is undergoing the difficult process of breaking new ground and as such it is the subject of many interpretations, debates and some controversy. Ronnie Harding and Elizabeth Fisher point out that the precautionary principle is mainly concerned with 'situations where scientific uncertainty is recognised in regard to the environmental outcomes of our activities'.[6]

There is a comprehensive body of knowledge relating to the principle consisting of official statements by authorities declaring operational frameworks, individual interpretations by experts studying the principle, and judicial statements as the result of litigation. The United Nations *Rio Declaration on Environment and Development* is an official statement, which expresses the definition in Principle 15 as:

> In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation.[7]

A group of experts at a conference in the United States developed the interpretation in the *Wingspread Statement on the Precautionary Principle,* which is as follows:

> Where an activity raises threats of harm to the environment or human health, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically.[8]

---

[6] Harding R & Fisher E, 1999, 'Introducing the precautionary principle' in R Harding & E Fisher, (eds), *Perspectives on the Precautionary Principle*, The Federation Press, Leichhardt, p 2

[7] United Nations Environment Program, *Rio Declaration on Environment and Development.* Available at: http://www.unep.org/Documents.multilingual/Default.asp?DocumentID=78&ArticleID=1163 last accessed 4 November 2013

[8] *Wingspread Statement on the Precautionary Principle*, 1998. Available at: http://www.gdrc.org/u-gov/precaution-3.html last accessed 4 November 2013

These two interpretations are a source of controversy. For example toxicologist Bernard Goldstein complains that the different wordings mean the precautionary principle 'lack[s] clarity in definition and consistency in use'.[9] However, in support of its many versions Nicolas de Sadeleer believes that '[a]ny attempt to define a legal principle by overly precise wording would definitively restrict its meaning, thereby rendering it useless'.[10] For de Sadeleer, the precautionary principle texts need to remain flexible and adaptable, amenable to a complex and context specific world. The culmination of these debates and its many interpretations is a definitive working definition of the precautionary principle formulated by the World Commission on the Ethics of Scientific Knowledge and Technology (COMEST), as follows:

> [w]hen human activities may lead to morally unacceptable harm that is scientifically plausible but uncertain, actions shall be taken to avoid or diminish that harm.[11]

In the COMEST definition the wording of the phrase 'actions shall be taken' is an imperative to act implying the urgency of the current environmental situation. This imperative contrasts with the weaker recommendation sketched above in Principle 15 of the *Rio Declaration* which simply says that a precautionary approach shall be widely applied.

The precautionary principle has been developed as an important tool in acting to mitigate harm to both the environment and human health in situations of scientific

---

[9] Goldstein BD, 2005, 'The Precautionary Principle: Is It a Threat to Toxicological Science?' *International Journal of Toxicology*, Vol 25, pp 3-7, p 3

[10] De Sadeleer N 2002, *Environmental Principles, From Political Slogans to legal Rules,* Oxford University Press, Oxford p 174

[11] United Nations Educational Scientific and Cultural Organization (UNESCO), Precautionary Principle Expert Group, World Commission on the Ethics of Scientific Knowledge and Technology (COMEST), 2005, *The Precautionary Principle*. Available at: http://unesdoc.unesco.org/images/0013/001395/139578e.pdf last accessed 4 November 2013, p 14

uncertainty.[12] It is evident in the European Union (EU) acceptance of the Registration, Evaluation and Authorisation of Chemicals (REACH)[13] regulation to assess new and existing chemicals, which shifts the burden of proof to the proponent or manufacturer to demonstrate that certain chemicals are safe. There is uneasiness when the proponent is in control of the science assessing the safety of the chemical, but as James Cameron points out, the precautionary principle 'does have legal effect'[14] and as such the proponent is liable to litigation if false data is produced.

Many authors identify timely action or interventions in the face of scientific uncertainty as a vital component of the precautionary principle. The consequences of not acting to mitigate potential but uncertain/unproven damage are demonstrated in the case studies of Harremoes et al[15] as "late lessons from early warnings". They expose the extent of human suffering and financial costs of delaying action. These authors identified warning signs, such as the potential irreversibility of actions, the novelty of new chemicals and harm to wildlife as triggers for early action.[16]

The case studies in Harremoes et al are all "false negatives", human activities initially thought to be harmless (e.g. asbestos) when in fact history proved them to be extremely harmful. Carolyn Raffensperger and Peter deFur argue that in science the false negative is emphasised because in these cases certainty has been considered necessary before acting to prevent harm. The precautionary principle reverses the preferred error to the

[12] ibid.
[13] European Commission, Enterprise and Industry, REACH - Registration, Evaluation , Authorisation and Restriction of Chemicals. Available at: http://ec.europa.eu/enterprise/reach/index_en.htm last accessed 4 November 2013
[14] Cameron J, 1994, 'The Status of the Precautionary Principle in International Law' in T O'Riordan & J Cameron (eds), *Interpreting the Precautionary Principle*, Earthscan Publications Ltd, London, p 16
[15] Harremoes P, Gee D, MacGarvin M, Stirling A, Keys J, Wynne B & Vaz SG, (eds), 2002, *The Precautionary Principle in the 20th Century, Late Lessons from Early Warnings*, Earthscan Publications Ltd, London, Sterling, VA
[16] ibid.

false positive, taking preventive action before an outcome is known, even though the outcome could result in no harm. This shift in emphasis should in fact generate more scientific research and prevent the possibility of irreversible damage.[17]

6.3 Scientific uncertainty in environmental studies

The precautionary principle applies to specific environmental problems that are of a complex nature, especially with regard to their causal relationships, and which exhibit unquantifiable scientific uncertainty limiting the applicability of traditional risk assessment.[18] This apparent move away from scientific certainty has caused critics to view the precautionary principle as unscientific. However, as stated in the introduction to the COMEST paper '[t]he Precautionary Principle is not unscientific; it acknowledges uncertainty in scientific practice'.[19] Sharon Beder writes, in respect of chemical use in the environment, 'scientists are usually unable to tell policy makers exactly where and how far a pollutant will spread, how it will interact with other pollutants, and how it will affect the health of people and the functioning of ecosystems'.[20] This view is supported by Harremoes et al who state that '[n]o matter how sophisticated knowledge is, it will always be subject to some degree of ignorance'.[21] Kriebel et al state that the 'cumulative and interactive effects of multiple

---

[17] Raffensperger C & deFur PL, 1999, 'Implementing the Precautionary Principle: Rigorous Science and Solid Ethics' *Human and Ecological Risk Assessment*, Vol 5(5), pp 933-941, p 937

[18] United Nations Educational Scientific and Cultural Organization (UNESCO), Precautionary Principle Expert Group, World Commission on the Ethics of Scientific Knowledge and Technology (COMEST), 2005, *The Precautionary Principle*. Available at: http://unesdoc.unesco.org/images/0013/001395/139578e.pdf last accessed 4 November 2013, p 25

[19] United Nations Educational Scientific and Cultural Organization (UNESCO), Precautionary Principle Expert Group, World Commission on the Ethics of Scientific Knowledge and Technology (COMEST), 2005, *The Precautionary Principle*. Available at: http://unesdoc.unesco.org/images/0013/001395/139578e.pdf last accessed 4 November 2013, p 15

[20] Beder S, 2006, *Environmental Principles and Policies, An interdisciplinary approach*, UNSW Press, Sydney, p 56

[21] Harremoes P, Gee D, MacGarvin M, Stirling A, Keys J, Wynne B & Vaz SG, (eds), 2002, *The Precautionary Principle in the 20th Century, Late Lessons from Early Warnings*, Earthscan Publications Ltd, London, Sterling, VA, p 187

insults on an organism or ecosystem are very difficult to study'[22] and they refer to the recent problems with endocrine disruption as an example:

> So shocking was this revelation [about the widespread observation of endocrine disruption in wildlife] that no scientist could have expressed the idea using only the data from his or her discipline alone without losing the respect of his or her peers.[23]

The precautionary principle is dependent on scientific methods to inform precautionary policy. Kriebel et al acknowledge that in environmental sciences observational studies are the rule because often experiments are not feasible or are unethical; hence they explore other types of evidence such as the accumulation of plausible conclusions from various independent lines of study.[24] They suggest some of these study lines into environmental causes of cancer may be provided by 'the geographic distributions of cancers; time trends in cancer frequency; …and experimental knowledge of chemical pathways of cancer induction'.[25] Whilst any one line may prove inadequate, '[i]t is the preponderance of evidence that finally prevails'.[26] However, Breitholtz et al call for more decisive rules, which 'stipulate that when relevant ecotoxicological information, i.e., sufficient test data is lacking this automatically calls for precautionary actions'.[27]

De Sadeleer argues that '[t]his type of complexity is the rule, rather than the exception, in ecosystems', consequently in approaching such complexity scientists 'put forward

---

[22] Kriebel D, Tickner J, Epstein P, Lemons J, Levins R, Loechler EL, Quinn M, Rudel R, Schettler T & Stoto M, 2001, 'The Precautionary Principle in Environmental Science' *Environmental Health Perspectives*, Vol. 109,(9), pp 871-876, p 874
[23] ibid.
[24] ibid.
[25] ibid, p 874
[26] Kriebel D, Tickner J, Epstein P, Lemons J, Levins R, Loechler EL, Quinn M, Rudel R, Schettler T & Stoto M, 2001, 'The Precautionary Principle in Environmental Science' *Environmental Health Perspectives*, Vol. 109,(9), pp 871-876
[27] Breitholtz M, Ruden C, Hansson SO, Bengtsson BE, 2006, 'Ten challenges for improved exotoxicological testing in environmental risk assessment' *Ecotoxicology and Environmental Safety* Vol. 63, pp 324-335, p 332

hypotheses rather than assertions'.[28] Kriebel et al also make the point that these hypotheses, provided by scientists to policy makers, are further 'limited by their tools and their imaginations and to a degree socially determined'.[29] However, regardless of these limitations, scientists still have an obligation to carry out science that protects both human health and the environment. In this situation the precautionary principle provides a 'standard that is to be observed, not because it will advance or secure an economic, political or social situation deemed desirable, but because it is a requirement of justice or fairness or some other dimension of morality'.[30] It is, therefore, a tool to support both scientists and decision-makers in carrying out their obligations to humans and the environment. As Peter Saunders notes '[b]y itself, the precautionary principle does not stop anything. What it does is prevent government and regulators from deliberately ignoring a strong scientific case by using the excuse that there is no proof of danger'.[31]

6.4 The precautionary principle and undone science

In 2013 the European Environment Agency's report *Late lessons from early warnings* found that governance of scientific ignorance and unknown unknowns has been neglected.[32] It acknowledged a need to identify uncertainties and ignorance and reveal why they exist. Although the authors acknowledged a recent increase in the

---

[28] De Sadeleer N 2002, *Environmental Principles, From Political Slogans to legal Rules,* Oxford University Press, Oxford, p 153

[29] Kriebel D, Tickner J, Epstein P, Lemons J, Levins R, Loechler EL, Quinn M, Rudel R, Schettler T & Stoto M, 2001, 'The Precautionary Principle in Environmental Science' *Environmental Health Perspectives*, Vol. 109,(9), pp 871-876, p 875

[30] Dworkin R quoted in S Marr, 2003, *The Precautionary Principle in the Law of the Sea, Modern Decision Making in International Law,* Martinus Nijhoff Publishers, The Hague, London, New York, p 12

[31] Saunders P, 2010, The Precautionary Principle, *Policy Responses to Societal concerns in food and agriculture: Proceedings of an OECD Workshop*, OECD. Available at: http://www.oecd.org/tad/agricultural-policies/46838007.pdf last accessed 23 September 2013

[32] European Environment Agency, 2013, *Late lessons from early warnings: science, precaution, innovation* EEA Report 1/2013. Available at: http://www.eea.europa.eu/publications/late-lessons-2 last accessed 29 October 2013

communication of scientific uncertainty, especially in the field of climate change, in other areas progress has been hampered by the existence of silos of knowledge created by bureaucratic structures. However, they found that even if a greater understanding of the complexity of the environment and awareness of scientific ignorance and uncertainties are gaining acceptance, serious impediments to action still exist. Philippe Grandjean points out two impediments, the first being a lack of institutional response to early warnings. But more importantly for my research he found, in agreement with David Hess, that the second was key decisions on research pathways are made and funded by those with vested interests.[33] Two measures the Report identified to overcome these limitations were ensuring independence from undue influence through using appropriate funding sources and applying robust policies on conflicts of interest.

Another basic problem, according to Grandjean, is 'that prevention has too often been deferred due in part to the alleged absence of convincing scientific evidence'.[34] This problem is further confounded when the absence is due to undone science as is the case in the Tasmanian devil disease. Grandjean also found that toxicologists continued to research known toxins. For instance, in his analysis of journals relating to environmental toxicology he found that the most frequent studies are still undertaken on lead and mercury.[35] These studies verify what is already known, whilst chemicals that act as endocrine disrupters where the boundaries of knowledge are limited, considerably fewer studies are undertaken. He further claims research should expand on current knowledge, not just be repetitive solely for the purposes of verifying the risks of chemicals already known to be hazardous. Studies should also deliver findings that

[33] ibid.
[34] Grandjean P, 2013, Science for precautionary decision-making in European Environment Agency Report, EEA Report 1/2013, *Late lessons from early warnings: science, precaution, innovation*, p 624. Available at: http://www.eea.europa.eu/publications/late-lessons-2 last accessed 11 September 2013
[35] ibid.

further support the magnitude of the suspected hazards thus facilitating precautionary and timely decision-making.

In the interim decisions need to be made. Inaction cannot be justified if plausible scientific evidence of serious harm exists on the basis of a lack of 'perfect' knowledge. This is especially relevant when studies have been abandoned or left undone for political reasons. It is often vested interests calling for more evidence of harm and insisting on a high level of causation or 'sound science' that lack credible scientific evidence in support of their argument of safety of their products or practices.[36] The probability of producing this level of confidence between cause and effect from a particular chemical, diffused within an environment, is low. As described in the preceding chapters the effort is often hampered by the same vested interests not funding studies that may prove harmful to their interests.

6.5 Precautionary principle status in Australia

In Australia the precautionary principle was adopted in February 1992 through the non-binding *Intergovernmental Agreement on the Environment*, whereby the Commonwealth, States, Territories, and Local Governments agreed to follow the precautionary principle as part of a commitment to ecologically sustainable development.[37] The parties agreed that:

> Where there are threats of serious or irreversible environmental damage, lack of full scientific certainty should not be used as a reason for postponing measures to prevent environmental degradation. In the application of the precautionary principle, public and private decisions should be guided by:

[36] European Environment Agency Report, EEA Report 1/2013, *Late lessons from early warnings: science, precaution, innovation*. Available at: http://www.eea.europa.eu/publications/late-lessons-2 last accessed 4 November 2013, p 646

[37] De Sadeleer N 2002, *Environmental Principles, From Political Slogans to legal Rules,* Oxford University Press, Oxford, p 147

(i) careful evaluation to avoid, wherever practicable, serious or irreversible damage to the environment; and
(ii) an assessment of risk-weighted consequences of various options.[38]

Subsequently, in Australia specific reference has been made to the precautionary principle in several Australian court considerations and more than twenty statutes and policy documents.[39]

The precautionary principle has been adopted at both the international and the national level in the protection of biodiversity. It is a key guiding principle in Australia's protection of biodiversity. At the international level biodiversity is protected under the United Nations *Convention on Biological Diversity* (*CBD*), which Australia ratified in 1993. It offers decision-makers guidance based on the precautionary principle and states:

> Concerned that biological diversity is being significantly reduced by certain human activities
>
> Noting that it is vital to anticipate, prevent and attack the causes of significant reduction or loss of biological diversity at source
>
> Noting also that where there is a threat of significant reduction or loss of biological diversity, lack of full scientific certainty should not be used as a reason for postponing measures to avoid or minimize such a threat[40]

---

[38] ibid, pp 147-148
[39] 'Of the Commonwealth's legislation, the *Environment Protection and Biodiversity Conservation Act 1999* provides the most detailed legislative exercise in its reference to the precautionary principle. Section 391 requires that the Minister take account of the precautionary principle in making decisions or granting permits. The New South Wales Parliament has been particularly active in promoting the principle. The *Protection of the Environment Administration Act 1991* adopted the precautionary principle as an objective…..' in N De Sadeleer, 2002, *Environmental Principles, From Political Slogans to legal Rules,* Oxford University Press, Oxford, p 148
[40] United Nations, 1993, Treaty Series, No. 30619, *Convention on Biological Diversity.* Available at: http://www.cbd.int/convention/text/ last accessed 4 November 2013

The precautionary principle is not directly named in the above declarations. However, it is implicit in the wording - 'lack of full scientific certainty should not be used as a reason for postponing measures to avoid or minimize such a threat' - as a directive to govern decision making under conditions of uncertainty at the international level.[41] Supporting the *CBD* at the international level is *Agenda 21*, a series of action plans, developed through the United Nations Division for Sustainable Development, which recognise the impact human activities have on the environment.[42] *Agenda 21* is a guide for 21st century decision-makers in their efforts to halt the degradation of ecosystems that sustain life. The action plans include guidelines to conserve biological diversity, to combat deforestation and to manage toxic chemicals in the environment.

The Australian government has implemented both the *CBD* and *Agenda 21* under the *Environment Protection and Biodiversity Conservation Act 1999* (*EPBC*). The object of the *Act*, through the promotion of ecologically sustainable development, is the protection of biodiversity. The *Act* commits Australia to the precautionary principle with the direction that '[t]he Minister must consider the precautionary principle in making decisions'[43] and its objective of ecologically sustainable development[44] which includes the precautionary principle.[45] The *EPBC Act* and its promotion of ecologically sustainable development are implemented through the *National Strategy for Ecologically Sustainable Development*. The *Strategy* is primarily a framework for government to implement measures for the protection of biodiversity and is linked to

[41] De Sadeleer N 2002, *Environmental Principles, From Political Slogans to legal Rules,* Oxford University Press, Oxford
[42] United Nations, Sustainable Development, United Nations Conference on Environment & Development, Rio de Janerio, Brazil, 3 to 14 June 1992, *Agenda 21*. Available at: http://sustainabledevelopment.un.org/content/documents/Agenda21.pdf last accessed 4 November 2013
[43] Australian Government, ComLaw, *Environment Protection and Biodiversity Conservation Act 1999,* Part 16, 391. Available at: http://www.comlaw.gov.au/Details/C2013C00539 last accessed 4 November 2013
[44] ibid, Section 3
[45] ibid, Section 3A

Agenda 21 action plans. Although the *Strategy* does not directly refer to the precautionary principle, it states that two of its key goals are 'providing equity within and between generations' and 'dealing cautiously with risk and irreversibility'. Both of these are fundamental to the precautionary principle.

Although the implementation of the *CBD* into Australian legislation under the *EPBC Act* fails to identify explicitly the role of the precautionary principle, it is implicit in the wording of the *EPBC Act* and its directive through the *Strategy* and *Agenda 21* for decision-makers to act to protect biodiversity from human activities under environmentally sustainable development. The implicit understanding that the precautionary principle informs the protection of biodiversity is evidenced more emphatically in litigation, in both the international forum and in Australia.

Within the legal framework in Australia, even in the absence of an express legislative mandate to apply the precautionary principle, the judiciary in New South Wales (and elsewhere in Australia), has sought to apply the precautionary principle. In *Leatch v National Parks and Wildlife Service,* Justice Stein found:

> [w]hile there is no express provision requiring consideration of the 'precautionary principle', consideration of the state of knowledge or uncertainty regarding a species, the potential for serious or irreversible harm to an endangered fauna and the adoption of a cautious approach in protection of endangered fauna is clearly consistent with the subject maker, scope and purpose of the Act.[46]

[46] Stein, Hon. Justice PL, 1999, 'Are Decision Makers too cautious with the Precautionary Principle'. Available at: http://www.lawlink.nsw.gov.au/lawlink/supreme_court/ll_sc.nsf/pages/SCO_speech_stein_141099 last accessed 4 November 2013

More recently, in the Federal Court of Australia, Justice Marshall on 19 December 2006, in *Brown –v- Forestry Tasmania*, found in favour of the applicant who 'submits that the interpretation of the *EPBC Act* and the *Regional Forest Agreement (RFA)* are informed by the precautionary principle'.[47] The finding also states that '[T]he view I have taken about the construction of the *EPBC Act* is informed by the following matters:

> The *EPCB Act* was enacted to implement the provisions of the *Convention on Biological Diversity 1992*, and other international environmental agreements into Australian law.[48]

In summary Justice Marshall expanded on the level of protection by giving the following definition of protection:

> [p]rotection is not delivered if one merely assists a species to survive. Protection is only effective if it not only helps a species to survive, but aids in its recovery to a level at which it may no longer be considered to be threatened.[49]

6.6 Precautionary principle in relation to Australian fisheries and forestry

A comparison between two areas of biodiversity use, fisheries and forestry, provides an illustration of the level of protection provided to both marine and terrestrial biodiversity. Marine biodiversity is impacted by the activities of the numerous recreational and commercial fishers. It is, however, legislatively assured a level of protection, with an explicit reference to the precautionary principle. As a result of a 1997 Amendment, the *Fisheries Management Act 1991* now includes the precautionary principle. It states 'activities are conducted in a manner consistent with the principles of ecologically sustainable development and the exercise of the precautionary principle', including 'the

[47] *Brown v Forestry Tasmania* (no. 4) (2006) FCA 1729. Available at: http://www.austlii.edu.au/au/cases/cth/federal_ct/2006/1729.html last accessed 4 November 2013
[48] ibid.
[49] ibid.

need to have regard to the impact of fishing activities on non-target species and the long term sustainability of the marine environment'.[50] This reference to 'non-target species' provides a measure of protection to biodiversity for its own value, as opposed to its use value for goods and services.

In contrast, there is no equivalent explicit reference to the precautionary principle in the protection of terrestrial biodiversity in the management of forests. In 1992 under a framework for the sustainable development of Australian forests the *National Forestry Policy Statement* (*NFPS*) was introduced. Within this framework Regional Forests Agreements (RFAs) were adopted between the Commonwealth and state governments. *RFAs* are 20-year plans agreed between state and federal governments for the conservation and sustainable management of Australia's native forests. The role of the RFAs was to ensure a balance between conservation and economic development of Australia's native forests and the introduction of forest plantations. Further, in order to protect Australia's forest environment, the *RFA* implements the *Comprehensive, Adequate and Representative Reserve Systems for Forests (CAR)* in Australia.[51] The *CAR* system is specifically designed to 'safeguard biodiversity, old-growth forests, wilderness, and other natural and cultural values' but it does not explicitly include the precautionary principle. The CAR reserve system allows for RFAs to be exempt from the *Environmental Protection and Biodiversity Conservation Act 1999 (EPBC Act)*

[50] Commonwealth Numbered Acts, Fisheries Legislation Amendment Act 1997, No 120 of 1997 – Schedule 1. Available at: http://www.austlii.edu.au/au/legis/cth/num_act/flaa1997302/sch1.html last accessed 4 November 2013

[51] Joint ANZECC/MCFFA National Forest Policy Implementation Sub-Committee, 1997, *Comprehensive, Adequate and Representative Reserve Systems for Forests in Australia*. Available at: http://www.daffa.gov.au/__data/assets/pdf_file/49493/nat_nac.pdf last accessed 22 February 2007

under Section 38, which states 'approval not needed for forestry operations permitted by regional forest agreements'.[52]

The CAR assessments, however, rather than providing forestry operations with exemptions, are understood to constitute a form of assessment and approval for the purposes of the *EPBC Act*.[53] This arrangement circumvents the need for the Commonwealth to be involved in every assessment of logging practices on a coupe by coupe[54] basis which was deemed administratively impracticable. Responsibility for monitoring and assessment therefore devolves to the Forestry Practices Authority (FPA) through Forestry Practices Plans (FPPs), which must be submitted before logging commences. The forestry industry however operates under a self-regulatory regime and issues of non-compliance and lack of accountability are a cause for ongoing concern in Tasmania.

Meanwhile, the precautionary principle is referred to in the *RFA* but in relation to 'Information Collection and Assessment' which states '[i]f key information is lacking, the precautionary principle may need to be applied to avoid unacceptable environmental degradation'.[55] In Australia's *State of the Forest Report* 2008, the precautionary principle is recognised in the development and implementation of indicators used to

---

[52] Australian Government, *Environment Protection and Biodiversity Conservation Act 1999*, Section 38. Available at: http://www.austlii.edu.au/au/legis/cth/consol_act/epabca1999588/s38.html last accessed 2 December 2013

[53] Australian Government, The Australian Environment Act: Report of the Independent review of the Environment Protection and Biodiversity Conservation Act 1999, Final Review, Chapter 10, Regional Forest Agreements. Available at: http://www.environment.gov.au/epbc/review/publications/final-report.html last accessed 8 August 2012

[54] Coupe is an area of forest with established boundaries which has been set aside for commercial forestry activities. Available at: http://www.daff.gov.au/rfa/publications/deferred/kit/glossary last accessed 9 August 2012

[55] Australian Government, Department of Agriculture, Fisheries and Forests, *Regional Forest Agreement*. Available at: http://www.daffa.gov.au/rfa/about/process/introduction last accessed 22 February 2007

characterise the essential components of sustainable forest management including the conservation of biological diversity.[56]

6.7 Precautionary principle in relation to biodiversity in Tasmania

The *EPBC Act*, administered at the federal level through the Department of Sustainability, Environment, Water, Population and Communities, provides a legal framework to protect and manage important biodiversity.[57] As such the *Act* is the primary Australian legislation for the protection of threatened species. Biodiversity protection is activated through the identification of threatened species and ecological communities for which Recovery Plans are developed. Recovery Plans are a Federal government legislative requirement but are not a requirement under the threatened species legislation in Tasmania. The management of threatened species in Tasmania is the responsibility of the Biodiversity Conservation Branch of the Department of Primary Industries, Parks, Water, and the Environment (DPIPWE). This management is implemented through *Tasmania's Nature Conservation Strategy 2002-2006.* [58]

The *Nature Conservation Strategy* has explicit references to the precautionary principle. Firstly, the *Strategy* defines the precautionary principle as 'when threats or potential threats may cause serious environmental or species damage, a lack of scientific certainty should not be used as a reason for postponing or preventing protective action'.[59] Also, as one of its guiding principles states: '[p]rotecting natural diversity requires identifying,

[56] Montreal Process Implementation Group for Australia, 2008, *Australia's State of the Forests Report, 2008,* Bureau of Rural Sciences, Canberra
[57] Australian Government, Department of the Environment, *Environmental Protection and Biodiversity Conservation Act.* Available at: http://www.environment.gov.au/epbc/ last accessed 22 October 2013
[58] Tasmanian Government Department of Primary Industry and Water, *Tasmanian's Nature Conservation Strategy 2002-2006.* Available athttp://www.dpiw.tas.gov.au/inter.nsf/WebPages/JCOK-5KZTT4?open last accessed 10 February 2007
[59] Tasmanian Government Department of Primary Industry and Water, *Tasmania's Nature Conservation Strategy 2002-2006.* Available at: http://www.dpiw.tas.gov.au/inter.nsf/Attachments/JCOK-5L2664/$FILE/NCS%20Final%20Report%202003.pdf last accessed 15 February 2007, p 55

preventing and reducing threats and, where necessary, acting cautiously (i.e. applying the precautionary principle)'.[60] However, in contrast to this *Strategy* which calls for the implementation of the precautionary principle to conserve species, DPIPWE has also developed a *Threatened Species Strategy for Tasmania*[61] which does not refer specifically to the precautionary principle. It does however require care of the land in an ecologically sustainable manner, which implies the precautionary principle since it is integral to ecologically sustainable development in Australia. It is therefore evident that forestry operations in Tasmanian have a commitment to the precautionary principle in the protection of biodiversity, especially the protection of endangered or threatened species.

6.8 The *EPBC Act* and the Tasmanian devil

In 2006 the Tasmanian devil was listed as a vulnerable species under the *EPBC Act,* which meant the issue of a Policy Statement 3.6 giving the reasons for the listing.[62] The Policy provides guidelines for persons undertaking actions that might impact on the Tasmanian devil and require referral under the *Act* to the Federal Minister. Activities that might require referral include:

- actions that may assist or accelerate the spread of DFTD,
- the construction of new roads or substantial upgrades to existing roads in sensitive locations, and
- any actions that involve the loss or intensified use of large or important areas of Tasmanian devil habitat such as clearing for urban development, flooding associated with dam building, or the intensifying or changing of agricultural land use.

---

[60] ibid, p 4
[61] Tasmanian Government Department of Primary Industry and Water, *Threatened Species Strategy for Tasmania*. Available at: http://www.dpiw.tas.gov.au/inter.nsf/Attachments/RLIG-542642/$FILE/threatspstrat.pdf last accessed 15 February 2007
[62] Australian Government, Department of the Environment and Heritage, 2006, *EPBC Act Policy Statement 3.6 Tasmanian Devil (Sarcophilus harrisii),* Department of Environment and Heritage, Canberra

It was noted that listing of the Tasmanian devil as threatened under the *EPBC Act* complemented the commitments of both the Australian and Tasmanian governments in finding a cure for DFTD. These included:

- diagnostic research to understand the cause of the disease
- laboratory work to pinpoint the origin of the disease, to find a test to diagnose individuals before cancers appear and hopefully to find a cure
- field monitoring to map the spread of the disease
- management strategies to combat the impact of the disease – trialing disease suppression and establishing disease-free captive populations.

In 2009 the Tasmanian devil listing under the *EPBC Act 1999* was upgraded to endangered. Under the *Act* provision is made for the adoption or implementation of recovery plans to identify the research and management actions necessary to maximize the survival of the species in the wild. Destruction of habitat is considered the most threatening human activity to the survival of biodiversity worldwide. It is therefore the protection of devil habitat in Tasmania that should be of the highest priority. The DPIPWE has drafted, but not implemented a Recovery Plan, which addresses the measures it proposes including a disease-free insurance population, maintaining genetic diversity within this population, and managing and protecting devils and their habitats in the wild.

6.9 The Draft Recovery Plan

The *Draft Recovery Plan for the Tasmanian devil* as proposed by the DPIPWE is in accordance with the *EPBC Act* therefore it is a requirement that the Plan adhere to the principles therein. Given this context it is imperative that due consideration is given to all possible threats to the survival of the endangered Tasmanian devil. This includes not only the implementation of measures to conserve the remaining population in the hope of reintroducing the devil to Tasmania but also mitigating the current threats from

human activities in Tasmania. This protection should include not only securing suitable habitat and limiting loss, degradation and fragmentation from agricultural and forestry practices, but within this framework it should also, given the evidence provided in the previous chapters, specify minimizing impacts from the use of pesticides.

According to the Draft Recovery Plan the strategies for recovery given the highest priorities are 'applied research' including:

1. develop a diagnostic technique;
2. determine latency periods;
3. investigate the nature of transmission;
4. identify resistant genotypes; and
5. develop a vaccine capable of being delivered in the wild.

Attempts have been made to develop a diagnostic tool but as yet no tool exists. It was initially thought a blood test might be possible but efforts to do field testing were hampered by a lack of funds to buy portable equipment.[63] More recently a biomarker for a pre-clinical diagnosis was the subject of the PhD thesis submitted by Jessica Gathercole at the University of Tasmania. She found encouraging results from her research but acknowledged validation of the methods would need to be undertaken.[64]

Determining the latency period of the disease has also not been resolved. McCallum et al in a paper published *EcoHealth* in 2007 suggested it might be six months.[65] In 2012 Hamede et al published a paper that claimed whilst the latent period of the disease was

[63] Duffy C, 2009, Time running out for Tasmanian devil, The 7.30 Report, Australian Broadcasting Commission. Available at: http://www.abc.net.au/7.30/content/2009/s2548975.htm last accessed 22 October 2013
[64] Gathercole JL, 2012, *Biomarker discovery for pre-clinical diagnosis of Tasmanian Devil Facial Tumour Disease,* Doctor of Philosophy, University of Tasmania, Hobart.
[65] McCallum H, Tompkins DM, Jones M, Lachish S, Marvanek S, Lazenby B, Hocking G, Wiersma J & Hawkins CE, 20007, Distribution and Impacts of Tasmanian Devil Facial Tumour Disease, *EcoHealth*, Vol 4(3), pp 318-325

unknown it probably varied from between three to twelve months.[66] Meanwhile, there is no evidence that a resistant genotype exists and to date no vaccine is available. The possibility that pesticides might play a role in the disease has been ignored, consequently this scientific research remains undone.

The *Draft Recovery Plan* notes that Forestry Tasmania, Private Forests Tasmania, Gunns Limited and Tasmanian Farmers and Graziers Association are stakeholders in the recovery program. Once again a conflict of interest exists when those with the most to gain from plantation forestry are not at arms length from the strategies to protect endangered species.

6.10 Conclusion

The precautionary principle is a tool to enable policy makers to act in the face of scientific uncertainty as to the cause of harm, in this case the Tasmanian devil cancer. The situation for the Tasmanian devil is mired in scientific uncertainty. Little is known about the devil in the wild including identification and mapping of its habitat, location of maternal den sites, population numbers, impacts of logging, plantation forestry or the use of pesticides and poisons on devils or their native prey. Much of the scientific research into the devil cancer DFTD as demonstrated in the previous chapters is under-researched, abandoned or simply undone. The uncertainty I have raised is in relation to the cause; is it a contagious cancer (perhaps the result of an original environmental toxin) or is it a cancer initiated by current environmental toxins? The evidence of harm, although not linked to the use of pesticides, is confirmed in the findings of scientific

[66] Hamede R, Bashford J, Jones M & McCallum H, 2012, Simulating devil facial tumour disease outbreaks across empirically derived contact networks, *Journal of Applied Ecology*, Vol 49, pp 447-456

studies on devils and other wildlife species in Tasmania.[67] Further evidence of the harmful effects of pesticides and poisons, the same as those used in plantation forestry in Tasmania, are evident in peer-reviewed overseas studies. Considering the magnitude of the harm and the possible irreversible consequences it is appropriate that the precautionary principle under the *EPBC Act* be implemented to mitigate the harm to the Tasmanian devil until relevant research into the cancer can be undertaken.

Impediments to action however exist in the form of undue influence and conflicts of interest and in the final chapters I explore these factors. In chapter 8 I establish that undue influence exerted on the US regulator by Syngenta, the manufacturer of atrazine, continues to delay action to further restrict or ban this chemical. The Australian regulator, the APVMA, has followed the US for reasons that are not obvious. In chapter 9 I argue that in Tasmania the government department, the DPIPWE, is in a conflict of interest because of its links to the forestry industry. In chapter 10 I also question the political will of the Tasmanian government to act according to the *EPBC Act* and the precautionary principle to protect the Tasmanian devil and its habitat. I suggest therefore that public participation and lay knowledge need to be incorporated into the governance of contentious issues that impact on human and environmental

[67] A fungal infection in platypus populations in Tasmania cited in Connolly JH, Obendorf DL, Whittington RJ & Muir DB, 1997, Causes of Morbidity and Mortality in Platypus (Ornithorhynchus Anatinus) from Tasmania, with particular reference to Mucor Amphibiorum infection, *Australian Mammology*, Vol 20, pp 177-187. A chytrid fungal infection in frogs in Tasmanian populations cited in Pauza M & Driessen M, 2008, *Distribution and Potential Spread of Amphibian Chytrid Fungus, Batrachochytrium dendrobatidis in the Tasmanian Wilderness World Heritage Area*, Biodiversity Conservation Branch, Department of Primary Industries and Water, Hobart, Tasmania. Wobbly possum disease in Tasmania cited in Hufschmid J & Holz P, 2011, Dasyurids, Numbats, Possums and Gliders, Viral Diseases, Australian Registry of Wildlife Health. Available at: http://arwh.org//sites/default/files/files-uploads/15%20DASYURIDS.pdf last accessed 23 October 2013. Abnormalities in commercial oysters in Tasmania cited in Scammell, M, 2004, *Environmental Problems Georges Bay, Tasmania*, Tasmanian Seafood Industry Council, Hobart, Tasmania.

health in order to overcome some of the shortcomings I have mentioned in the previous chapters.

In this chapter I have argued that the precautionary principle under the *EPBC Act* be implemented to mitigate further harm to the Tasmanian devil. In the next chapter I will argue that the precautionary principle should be implemented to further restrict or ban the use of atrazine, a carcinogen that the devils may be exposed to, as has occurred in Europe under their REACH program. In support of this argument I analyse four wildlife cancers, including the Tasmanian devil, to show that studies are indeed focused on current knowledge and that relevant toxicology studies are avoided.

Chapter 7 – The need for the precautionary principle – Atrazine and four wildlife cancer case studies

7.1 Introduction

In this chapter I analyse and compare four wildlife cancer clusters[1] including the Tasmanian devil and advocate the need to implement the precautionary principle in restricting the use of atrazine. In 2009 Denise McAloose and Alisa Newton published an article in *Nature Reviews Cancer* titled 'Wildlife cancer: a conservation perspective'.[2] They noted that cancer is much more widespread in wildlife than is generally realised. The Wildlife Conservation Society (WCS), where McAloose is the chief pathologist, found a common cause: pollution created by humans.[3] McAloose and Newton also note that cancer in wildlife is reduced when environmental contaminants are removed from the environment.[4]

In these four major wildlife cancers I have made a comparison between the different research programs to show that in all cases few or no toxicology studies have been undertaken. In all cases various toxins have been found in the tissue or fat of the species but further studies to determine the possible role of these contaminants in

[1] Cancer clusters are identified by certain circumstances including: a large number of cases of a specific type of cancer, rather than several different types; a rare type of cancer, rather than common types; or an increased number of cases of a certain type of cancer in an age group this is not usually affected by that type of cancer. Available at: http://imsdd.meb.uni-bonn.de/cancernet/600358.html last accessed 27 January 2013

[2] McAloose D & Newton AL, 2009, *Nature Reviews: Cancer*, Vol 9, pp 517-526

[3] Rogers S, 2009, Our pollution is giving animals cancer, too. Available at http://www.mnn.com/earth-matters/wilderness-resources/stories/our-pollution-is-giving-animals-cancer-too last accessed 29 July 2009

[4] McAloose D & Newton AL, 2009, *Nature Reviews: Cancer*, Vol 9, pp 517-526, p 523

initiating or progressing the cancers have not been done. Considerable funding has, however, progressed the research into other areas, as observed by Philippe Grandjean in the previous chapter, namely into known causes of cancer. Meanwhile, the lack of full toxicology studies means the cause of the cancer is uncertain. I will show that evidence exists that the habitats of each population are contaminated by agricultural chemicals including atrazine.

7.2 Chemicals in the environment

The sheer volume of chemicals entering the environment, not only as individual active ingredients but also in mixtures, means that regulation must aim to adequately provide a measure of safety for the environment and human populations. Global chemical pollution is a serious problem. It is estimated that ninety per cent of water and fish samples are contaminated by pesticides and an estimated three per cent of agricultural workers suffer from acute exposure.[5] In 2011 it was estimated that more than 248,000 chemical products were commercially available and subject to regulatory systems.[6] In Australia the National Industrial Chemicals Notification and Assessment Scheme (NICNAS), lists approximately 250 chemicals introduced at more than 1000 tonnes per year, with another 450 chemicals introduced at more than 100 tonnes per year.[7] The Hazardous Substances Information System (HSIS) complied by the Office of the

[5] United Nations Environment Programme, 2012, Global Environmental Outlook 5, Chapter 6, Chemicals and Waste. Available at: http://www.unep.org/geo/pdfs/geo5/GEO5_report_C6.pdf last accessed 25 July 2013

[6] Barra R, Portas P & Watkinson RV, 2011, Chemicals and Waste, in United Nations Environment Program, *Global Environmental Outlook 5*. Available at: http://www.unep.org/geo/pdfs/geo5/GEO5_report_C6.pdf last accessed 5 November 2013

[7] Australian Government, Department of Health and Ageing, NICNAS [National Industrial Chemicals Notification and Assessment Scheme], 2006 'Promoting safer chemical use: towards better regulation of chemicals in Australia' Final Report and Recommendations. Available at: http://www.chemicalspolicy.org/downloads/EC_Review_FINAL_REPORT.pdf last accessed 5 November 2013

Australian Safety and Compensation Council lists about 3,000 chemicals classified as hazardous.[8]

While some modern synthetic chemicals are known or suspected of being carcinogens (having the ability to cause cancer) many more are yet to be tested for their long-term effects.[9] Of the approximately 80,000 chemicals on the market, many ubiquitous in the environment, few have been tested for their ability to induce cancer either singularly or in combination with other chemicals or factors.[10] As Samuel Epstein revealed in his classic book *The Politics of Cancer,* manufacturers often fail to undertake relevant studies or produce findings biased in their favour in order to avoid linking their product or products to cancer causation.[11] Independent studies, on the other hand, are often dismissed as irrelevant or inadequate or are not funded and hence remain undone. In this chapter I will outline why a closer examination of the role of environmental toxins in the initiation and progression of wildlife cancers, and in particular the devil cancer, is needed if progress is to be made in understanding these cancers.[12]

It has been proposed that environmental factors play a more important role than has previously been acknowledged.[13] It is not new knowledge that environmental factors cause cancer; Percival Potts described scrotum cancer in young London chimney

---

[8] ibid.
[9] Aronson K, 2010, *Environmental chemicals and cancer*, David Suzuki Foundation. Available at: http://www.davidsuzuki.org/blogs/docs-talk/2010/04/environmental-chemicals-and-cancer/ last accessed 7 January 2013
[10] Reuben SH, 2010, President's Cancer Panel, 2008-2009 Annual Report, *Reducing Environmental Cancer Risk*, National Cancer Institute. Available at: http://deainfo.nci.nih.gov/advisory/pcp/annualReports/index.htm last accessed 7 January 2013
[11] Epstein SS, 1978, *The Politics of Cancer,* Sierra Club Books, San Francisco
[12] Servan-Schreiber D, 2008, We can stop the cancer epidemic, *The New York Times*. Available at: http://www.nytimes.com/2008/09/19/opinion/19iht-edservan.1.16308287.html?_r=0 last accessed 7 January 2013
[13] Irigaray P, Newby JA, Clapp R, Hardell L, Howard V, Montagnier Epstein S & Belpomme D, 2007, Lifestyle-related factors and environmental agents causing cancer: An overview, *Biomedicine and Pharmacotherapy*, Vol 61(10), pp 640-658

sweeps in the 18th century. This claim was more recently supported by the 2010 *US President's Cancer Panel Report*, which also found environmental factors have contributed to the increase in cancer.[14] It recommended stronger policies to reduce exposure. The *Report* was however not without its critics.[15]

Given the acknowledged awareness of the role of environmental toxins in cancer and the more recent research developments, which highlight the role of epigenetics and endocrine disrupters in cancer causation, it seems appropriate to incorporate this knowledge into wildlife cancer studies. The roles of epigenetics (changes in gene expression) and endocrine disrupters (synthetic chemicals that mimic hormones) need to be incorporated into the research programs to better understand the complexities of cancer. The uncertainties that exist as to the role of epigenetics and endocrine disrupters, as Kriebel et al found in the previous chapter, in the evolution of cancer are further compelling reasons for the implementation of the precautionary principle to mitigate harm.

7.3 Epigenetic factors

John Peterson Myers, Chief Scientist of Environmental Health Services and co-author of *Our Stolen Future*, states that epigenetic mechanisms affecting development have profound importance. He claims 'contaminants altering the epigenetic control of gene expression are key to understanding fetal origins of adult disease'.[16] Although epigenetics is a new field of research there is already compelling evidence for the role

[14] Reuben SH, 2010, The President's Cancer Panel, 2008-2009 Annual Report, *Reducing Environmental Cancer Risk, What We Can Do Now?* US Department of Health and Human Services, National Institutes of Health, National Cancer Institute, Bethesda, MD
[15] Grady D, 2010, US Panel Criticized as Overstating Cancer Risks, *The New York Times*. Available at: http://www.nytimes.com/2010/05/07/health/research/07cancer.html?_r=0 last accessed 8 January 2013
[16] Email from John Peterson Myers 20 January 2010 at 6:48 pm.

of epigenetics in cancer.[17] Randle Jirtle of Duke University Medical Center in Durham in the United States (US) in an interview stated that epigenetic changes 'may be thought of as chemical switches that can turn on and off the expression of genes in response to environmental factors'.[18]

Scientific evidence exists of key epigenetic processes with links to both the initiation and the progression of cancer.[19] Epigenetics, unlike the oncogene theory of cancer causation, is the study of changes in gene activity. According to Andy Bannister, Senior Research Associate at the Cambridge Cancer Centre in the United Kingdom, 'epigenetics refers to heritable changes in gene expression that occur without alteration in DNA sequence'.[20] This research linking environmental pollutants with epigenetic variation is a rapidly growing area.[21] In a review Lifang Hou and colleagues found some diseases have been linked to environmental chemical-related epigenetic changes.[22] These chemicals included: heavy metals such as arsenic and cadmium; pesticides such as vinclozolin and methoxyclor, dioxins, Bisphenol A (industrial plasticiser); and RDX[23] (Hexahydro-1,3,5-trinitro-1,3,5-triazine).[24]

The US National Institute of Environmental Health Services (NIEHS) in January 2012 held a minisymposium to communicate information about the emerging science and

---

[17] Kuehn BM, 2008, Epigenetics a Window on Gene Dysregulation, Disease, *Journal of American Medical Association*, 29(11), pp 1249-1250
[18] ibid.
[19] Bannister A, nd, *The role of epigenetics in cancer*. Available at: http://www.abcam.com/index.html?pageconfig=resource&rid=10755&pid=10628 last accessed 9 May 2012
[20] ibid.
[21] Hou L, Zhang X, Wang D & Baccarelli A, 2012, Environmental chemical exposures and human epigenetics, *International Journal of Epidemiology,* Vol 41, pp 79-105
[22] ibid, p 79
[23] RDX - British code name for Research Department Explosive or Royal Demolition Explosive. Available at: http://www.epa.gov/IRIS/subst/0313.htm#oralrfd last accessed 30 April 2009
[24] Hou L, Zhang X, Wang D & Baccarelli A, 2012, Environmental chemical exposures and human epigenetics, *International Journal of Epidemiology,* Vol 41, pp 79-105, p 91

technologies being developed to explore the epigenetic mechanisms underlying the developmental basis for disease. In his opening address Deputy Director Rick Woychik was reported as stating 'environmental exposure is increasingly linked to changes in epigenetic profiles and subsequently with disease'.[25]

The role of epigenetics in cancer initiation and progression is still little understood but environmental factors including synthetic chemicals appear to play an important part. Further research will need to be funded and studies undertaken for this important aspect of cancer development to be better understood and preventative measure adopted. In the case of the Tasmanian devil cancer, epigenetic research has commenced. In 2013 the DFTD researchers published the first results of a study into epigenetics with the ambiguous findings 'that DFTD should not be treated as a static entity, but rather as an evolving parasite with epigenetic plasticity'.[26] It is, however, generally considered more appropriate to use epigenetic knowledge to prevent disease rather than cure it.[27] Research associates at the University of Florida's Department of Zoology in the US also suggest epigenetic research could in the future be adopted for better risk assessment of chemical agents.[28]

Environmental chemicals appear to influence not only epigenetic processes but also endocrine, neural and immune systems leading to developmental and reproductive

---

[25] Godfrey A, 2012, *Minisymposium brings epigenetic experts to NIEHS*. Available at: http://www.niehs.nih.gov/news/newsletter/2012/2/science-epigenetic/index.htm last accessed 7 May 2012

[26] Ujvari B, Pearse AM, Peck S, Harmsen C, Taylor R, Pyecroft S, Madsen T, Papenfuss AT & Belov K, 2013, Evolution of a contagious cancer: epigenetic variation in Devil Facial Tumour Disease, *Proceedings of the Royal Society B*, Vol 280(1750), 20121720

[27] Johnston J, 2010, Lamarck lives! The epigenetic revolution in environmental health, *Health & Environment,* Issue 22. Available at: http://healthandenvironmentonline.com/issue-archive/epigenetics/ last accessed 11 May 2012

[28] ibid.

diseases and cancer. The role of endocrine disrupters in these processes is the topic of the next section.

7.4 Endocrine disrupters

Mounting evidence is identifying endocrine disrupters in the aetiology of diseases such as cancer. Endocrine disrupters are chemicals that 'interfere with gene-controlled signaling systems' in the control of 'prenatal and postnatal development and function through life'.[29] This is also a new and rapidly developing research area that 'has evolved from many disciplines and encompasses molecular and cellular *in vitro* studies, whole animal studies and human epidemiology'.[30] Linda Birnbaum, Director of the National Institute of Environmental Health Sciences, National Institutes of Health and Director of the National Toxicology Program of the US Department of Health and Human Services, in listing the following four important aspects of endocrine disrupters stated -

- First, the effect of low doses. Normal endocrine signaling involves very small changes in hormone levels, yet these changes can have significant biological effects. That means subtle disruptions of endocrine signaling is a plausible mechanism by which chemical exposures at low doses can have effects on the body.
- Second, the wide range of effects. Endocrine signals govern virtually every organ and process in the body. That means that when outside chemicals interfere with those systems, the effects can be seen in many different diseases and conditions – some of which we are just learning to recognize as the result of endocrine disruption.
- Third, the persistence of effects. We are finding that the effects of exposure to endocrine disruptors can be observed long after the actual exposure has ceased. This is especially true for growth and development, processes that are very sensitive to endocrine regulation. The question of how these kinds of latent effects occur is an active area of investigation.
- Fourth, the ubiquity of exposure. Both naturally occurring and manmade substances can be endocrine disruptors. Some, e.g., arsenic and agricultural chemicals, are ubiquitous in the environment. In addition to the growing use of

[29] Chapin et al, 1996 cited in T Colborn and LE Carroll, 2007, Pesticides, Sexual Development, Reproduction and Fertility: Current Perspective and Future Direction, *Human and Ecological Risk Assessment,* Vol 13(1078-1110), p 1078
[30] ibid.

> hormonally-active pharmaceuticals that pass through the bodies of those taking them and end up in water treatment systems and surface waters, many of the chemicals that are being found to have endocrine effects are components of a wide range of consumer products, including some water bottles, cosmetics, sunscreens, and other personal care products. Substances applied to the skin can be directly absorbed but also end up getting washed off our bodies and into our water systems. As a result, chemicals with endocrine disrupting activity are widely dispersed in our environment, often at levels plausibly associated with biological effects; exposure to humans is widespread.[31]

There have been a number of studies in different areas indicating changes in developmental and reproductive systems. One important area is the early onset of puberty. In *The Copenhagen Puberty Study* the results concluded significant earlier breast development among girls born more recently.[32] This phenomenon has been documented in the Tasmanian devil population, with females producing young at an earlier age, although it has been explained as a natural response to the dramatic decline in devil numbers.[33]

As early as 1982 *The Erice Statement* of the World Federation of Scientists declared the need for new scientific processes to protect the planet.[34] It subsequently identified planetary emergencies and established permanent monitoring panels and working groups. One such group, the Permanent Monitoring Panel – Pollution, identified the problem of endocrine-disruptor chemicals in oceans, surface water, groundwater and drinking water supplies.[35] In a consensus statement from a work session on

---

[31] Birnbaum L, 2010, *Biology's Clock Interrupted: Endocrine Disrupting Chemicals in Drinking Water*, Testimony Before the Subcommittee on Energy and Environment, Committee on Energy and Commerce, United States House of Representatives, Department of Health and Human Services, Washington

[32] Aksglaede L, Sorensen K, Petersen JH, Skakkebaek NE & Juul A, 2009, Recent decline in age at breast development: the Copenhagen Puberty Study, *Pediatrics*, Vol 123(5), pp e932-939

[33] Jones ME, Cockburn A, Hamede R, Hawkins C, Hesterman H, Lachish S, Mann D, McCallum H & Pemberton D, 2008, Life-history change in disease-ravaged Tasmanian devil populations, *PNAS*, Vol 105(29) pp 10023-10027

[34] Dirac PAM, Kapitza P & Zichichi A, 1982, *The Erice Statement*. Available at: http://www.federationofscientists.org/WfsErice.asp last accessed 13 May 1012

[35] World Federation of Scientists, Permanent Monitoring Panel – Pollution. Available at: http://www.federationofscientists.org/PMPanels/Pollution/Pollution.asp last accessed 13 May 2012

environmental endocrine-disrupting chemicals the following was reached:

> 1. *We are certain of the following:*
>
> Wildlife, laboratory animals, and humans exhibit adverse health effects at contemporary environmental concentrations of man-made chemicals that act as endocrine disruptors. New technology has revealed that some man-made chemicals are present in tissue at concentrations previously not possible to measure with conventional analytical methods, but at concentrations which are biologically active.[36]

The consensus was more circumspect however in relation to uncertainties and understanding. They concluded:

> Relatively few of the man-made chemicals found in human tissue have been identified. Lack of funding has seriously constrained testing these chemicals for their potential to disrupt natural systems.
>
> Trade secret laws afford industry confidentiality depriving the consumers and public health authorities of the right to know the components of commercial products so they can be tested.[37]

In 1991 at the Wingspread Conference a framework for the concept of "endocrine disruption" was formulated. In 1996 Theo Colborn and colleagues published *Our Stolen Future* linking endocrine-disrupting chemicals to human and wildlife abnormalities.[38] It noted a warning from Noboru Takasugi and Howard Bern who reported findings that signaled links between early estrogen exposure and later cancers. They warned "[w]e feel that abnormal hormonal environments during early postnatal (and antenatal) life should not be underestimated as to their possible contribution to abnormal changes of neoplastic [cancerous] significance later in life".[39] Also in 1996 the European Environment Agency published a similar report on a major conference on

---

[36] Statement from the Work Session on Environmental Endocrine-Disrupting Chemicals: Neural, Endocrine, and Behavioral Effects, 1998, *Toxicology and Industrial Health*, Vol 14(1/2), pp 1-8, p 2
[37] ibid, pp 4-5
[38] Colborn T, Dumanoski, D & Myers JP, 1996, *Our Stolen Future, Are We Threatening Our Fertility, Intelligence, and Survival? – A Scientific Detective Story,* Little, Brown and Company, London
[39] ibid, pp 57-58

endocrine disrupting chemicals held in Weybridge England. The Report referred to as the *Weybridge Report* drew similar conclusions. Specifically in relation to cancer the report stated:

> It is evident that there are adverse health trends affecting the reproductive organs of both men and women. Thus, the incidence of testicular cancer has increased quite dramatically in countries with cancer registries[,] including Scandinavia, the countries around the Baltic Sea, Germany, UK [England], USA and New Zealand. Similarly there has been an increase in the incidence of breast cancer in many countries and the incidence of prostate cancer also appears to have risen. While changes in the incidence of prostate cancer may have been influenced by better reporting and better diagnostics, this can not explain the bulk of the increase in testis cancer. Similarly, the reported increase in breast cancer incidence seems real.[40]

Whilst in relation to wildlife, the following kinds of effects noted are quoted as:

- Female molluscs (e.g., snails, mussels) have turned into males as a result of exposure to endocrine-disrupting chemicals (a condition called imposex),
- In fish, males have been observed producing vitellogenin (a protein that gives rise to the yolk of eggs, and which is ordinarily only found in females). Furthermore, hermaphroditism has been observed in fish (a single fish having both male and female sex organs),
- Some reptiles (turtles and alligators), have reduced fertility due to undeveloped male sex organs (small penises),
- In birds, abnormal nesting behavior has been observed, namely female-female pairing,
- In mammals: disturbed fertility has been observed in common seals, grey seals, and Florida panthers.[41]

Similarly, in 1997 the US EPA recognized the growing evidence that a number of chemicals in the environment may disrupt endocrine systems of aquatic life and wildlife.[42] The Report concluded that evidence existed of disruption of endocrine systems in fish and feral species from synthetic chemicals, such as alkylphenols, bisphenol-A, 2,3,7,8-TCDD, 2,3,7,8-tetrachlorodibenzo-furan (TCDF), PCBs and some pesticides, such as alachlor, DDT, dicofol, methoxychlor, chlordane and many others.

---

[40] European Workshop on the Impact of Endocrine Disrupters on Human Health and Wildlife, 2-4 December 1996, Weybridge, UK, Report of Proceedings, p 13

[41] ibid, p 14

[42] Special Report on Environmental Endocrine Disruption: An Effects Assessment and Analysis, 1997, US Environmental Protection Agency, Washington, DC, p 54

The possible disruption of endocrine systems in a wide range of organisms by chemicals had become an important global issue as early as 1998. The Scientific Committee on Problems of the Environment (SCOPE) and the International Council for Science (ICSU) identified the need to deal with this issue and launched a joint Scientific Committee on Problems of the Environment/International Union of Pure and Applied Chemistry (SCOPE/IUPAC) project in 2000.[43] The project intended to prioritize future research needs, facilitate effective risk assessment and to deal with the problems on an international basis something that was stated as being quite unique. A SCOPE/IUPAC International Symposium on Endocrine Active Substances was held in Japan in 2002.[44]

Meanwhile, in 2001 John Peterson Myers, Sheldon Krimsky and R. Thomas Zoeller had identified progress in eroding the boundaries of ignorance surrounding endocrine disruptors whilst summarizing research that needed to be undertaken and are quoted as follows:

1. Further studies designed to test whether environmental chemicals can cause specific developmental defects by interacting with endogenous endocrine mechanisms.
2. Additional laboratory studies to characterize the low-dose, non-linear, and non-monotonic dose-response characteristics of endocrine disruptors
3. Development of strategies to characterize the effects of chemical mixtures on endocrine-guided developmental events in both wildlife and humans.
4. Additional studies to define the mechanisms by which hormones can influence early events in the developing brain and in the developing immune system.
5. Continued laboratory research to determine the mechanisms by which classes of environmental chemicals can interfere with hormone action in the adult and during development.
6. Studies aimed at identifying markers of exposure to chemicals and markers of endocrine disruption capable of detection at the time of health impacts which may be decades after exposure.

[43] IUPAC is the International Union of Pure and Applied Chemistry
[44] SCOPE/IUPAC International Symposium on Endocrine Active Substances, 17-21 November 2002, Yokohama, Japan. Available at: http://endo.endojournals.org/content/143/7/2774.short last accessed 13 May 2012

7. Systematic characterization of human [and wildlife] exposure patterns to hormonally-active compounds: what are the exposure pathways and what levels of contamination do they produce?
8. Mechanistic and epidemiological exploration of other systems vulnerable to hormonal disruption, with priority given to those potentially linked to important public health problems. Three examples are: learning disability and behavioral disorders; the hormonal control of body weight regulation; and immune system dysfunction.[45]

However, despite early recognition of endocrine disrupting chemicals, major problems exist, with regulatory systems and traditional toxicological and medical science being slow to incorporate this knowledge. This is in part because this class of chemical operates at extremely low levels and through very complex mechanisms. (The need for new testing regimes in regulatory practice is discussed further in Chapter 8.) In 2009 the American Medical Association (AMA) House of Delegates adopted a resolution calling on the AMA to support the US federal government to enact new policies to decrease exposure to endocrine disrupting chemicals.[46] Consistent with this view the Center for Biological Diversity petitioned the US EPA to establish water quality criteria for numerous endocrine-disrupting chemicals under the *Clean Water Act* as a first step in regulating and eliminating persistent and widespread chemicals that damage reproductive functions in wildlife and humans.[47]

[45] Myers JP, Krimsky S & Zoeller RT, 2001, Endocrine Disruptors – A controversy in Science and Policy: Session III Summary and Research Needs, *NeuroToxiciology*, Vol 22, pp 557-558
[46] Endocrine Society, Press Release, 2009, *AMA Adopts Endocrine Society Resolution Calling for New Policies to Decrease Public Exposure to Endocrine-Disrupting Chemicals*. Available at: http://www.sehn.org/rpr188.html#t2 last accessed 13 May 2012
[47] Center for Biological Diversity, Press Release, 2010, *EPA petitioned to regulate chemicals that pose widespread risk to human and animal reproduction.* Available at: http://www.biologicaldiversity.org/news/press_releases/2010/endocrine-disruptors-01-11-2010.html last accessed 13 May 2010

Also in 2009 the Endocrine Society issued a scientific statement on endocrine-disrupting chemicals.[48] It identified important issues in endocrine disruption including age at exposure, latency from exposure, importance of mixtures, nontraditional dose-response dynamics as well as transgenerational, epigenetic effects. According to the scientific statement '[t]here is no endocrine system that is immune to these substances, because of the shared properties of the chemicals and the similarities of the receptors and enzymes involved in the synthesis, release and degradation of hormones'.[49]

In 2010 Ana Soto and Carlos Sonnenschein published a review highlighting the carcinogenic properties of endocrine disrupting chemicals focusing on bisphenol A.[50] Three of the key points quoted from the review were:

- Hormones act as morphogens: extemporaneous exposure to even low doses of hormonally active chemicals increases the susceptibility to various diseases, including cancer
- Neoplasia is a tissue-based disease caused by various deleterious exposures that interfere with the reciprocal communication between cells and between cells and their surrounding extracellular matrix
- Sufficient supporting data have been gathered on the deleterious effects of endocrine disrupting chemicals to warrant immediate action to decrease human and wildlife exposure to these agents.[51]

The possible harmful effects of chemicals acting as endocrine disrupters has been known at least since Rachel Carson published her book *Silent Spring* in 1962.[52] Carson raised the issue of the harmful effects from chemicals that act to interfere with hormones in living organisms. By 1996 substantial evidence to support Carson's claim was published by Theo Colburn and colleagues in *Our Stolen Future* shifting the

[48] Diamanti-Kandarakis E, Bourguignon JP, Giudice LC, Hauser R, Prins GS, Soto AM, Zoeller T & Gore AC, 2009, Endocrine-Disrupting Chemicals: An Endocrine Society Scientific Statement, *Endocrine Reviews*, 30(4), pp 293-342
[49] ibid, p 296
[50] Soto AM & Sonnenschein C, 2010, Environmental causes of cancer: endocrine disruptors as carcinogens, *Nature Reviews Endocrinology*, Issue 60, pp 363-370
[51] ibid, p 364
[52] Carson R, with an introduction by Al Gore, 1994, *Silent Spring*, Houghton Mifflin, Boston

previous emphasis on chemicals as carcinogens and mutagens to the effects of chemicals on reproductive, neurophysical and developmental functions.[53] The scientific evidence has continued to mount over the years with a corresponding increase in the incidence of cancer, diabetes, autism and other related diseases.

In the following section I focus on atrazine, a known endocrine disrupter (although this is disputed by the manufacturer), providing evidence to support the existence of hazards from this chemical.

7.5 Atrazine its chemistry and production

In 1955 scientists at JR Geigy SA in Switzerland first synthesized atrazine and early tests indicated that it would be as effective on weeds as DDT had been on insects.[54] Atrazine is manufactured by Syngenta, an international corporation formed in 1999 with the merger of agrochemical and seed division of Novartis (formed by the merger of two Swiss giant chemical/pharmaceutical companies Ciba-Geigy and Sandos) and the agrochemical and biotechnology research division of AstraZeneca (part of which was formerly the British company Imperial Chemical Industries). Syngenta has grown to become a giant in the crop protection business, the largest agribusiness company in the world and the largest manufacturer of agrochemicals.[55] In 2011 Syngenta reported sales of $11.6 billion, an increase of 6 per cent over 2010, of which they state crop protection amounted to $8.9 billion, an increase of 3 per cent as a result of a 9 percent increase in

[53] Colborn T, Dumanoski D & Myers JP, 1996, *Our Stolen Future, Are We Threatening Our Fertility, Intelligence, and Survival? A Scientific Detective Story*, Little, Brown and Company, London.
[54] Fagin D, Lavelle M & the Center for Public Integrity, 1996, *Toxic Deception, How the Chemical Industry Manipulates, Science, Bends the Law and Endangers Your Health*, Carole Publishing Group, Secaucus, N.J.
[55] Corporate Watch UK, 2002, Syngenta, A company profile. Available at: http://www.corporatewatch.org.uk/?lid=212 last accessed 12 August 2007

volume.[56] Syngenta also has major interests in the biotechnology industry and in the production of genetically modified crops.[57]

Atrazine is an herbicide that kills weeds by acting to block photosynthesis, the process by which plants convert carbon dioxide and light into food.[58] It is widely used in the US to control weeds in corn, which is naturally resistant because it contains an enzyme that detoxifies atrazine. This ability of corn to neutralize atrazine has led to claims that atrazine is safe because it is metabolized into harmless products by atrazine tolerant plants. It is however far from safe. It is a persistent toxic chemical, which making it extremely dangerous in the environment. Its persistence is the reason why it is found in surface and ground water and even in rain, demonstrating its potential for transport to non-target sites.[59]

The process for producing atrazine involves the combination of toxic chemicals, and in its degradation process it produces equally toxic metabolites. The molecular structure of atrazine means it does not readily break down. The chemical structures of the triazines including atrazine are shown in Figure 7:1 below. According to the US EPA atrazine, simazine, propazine and their metabolites desethyl-s-atrazine (DEA), desisopropyl-s-atrazine (DIA) and diaminochlorotriazine (DACT) should be considered as a common

---

[56] Syngenta, 2011, Media Release 2010 Full Year Results. Available at: http://www.syngenta.com/global/corporate/SiteCollectionDocuments/pdf/media-releases/en/20110209-en-fullversion-full-year-results-2010.pdf last accessed 18 June 2012

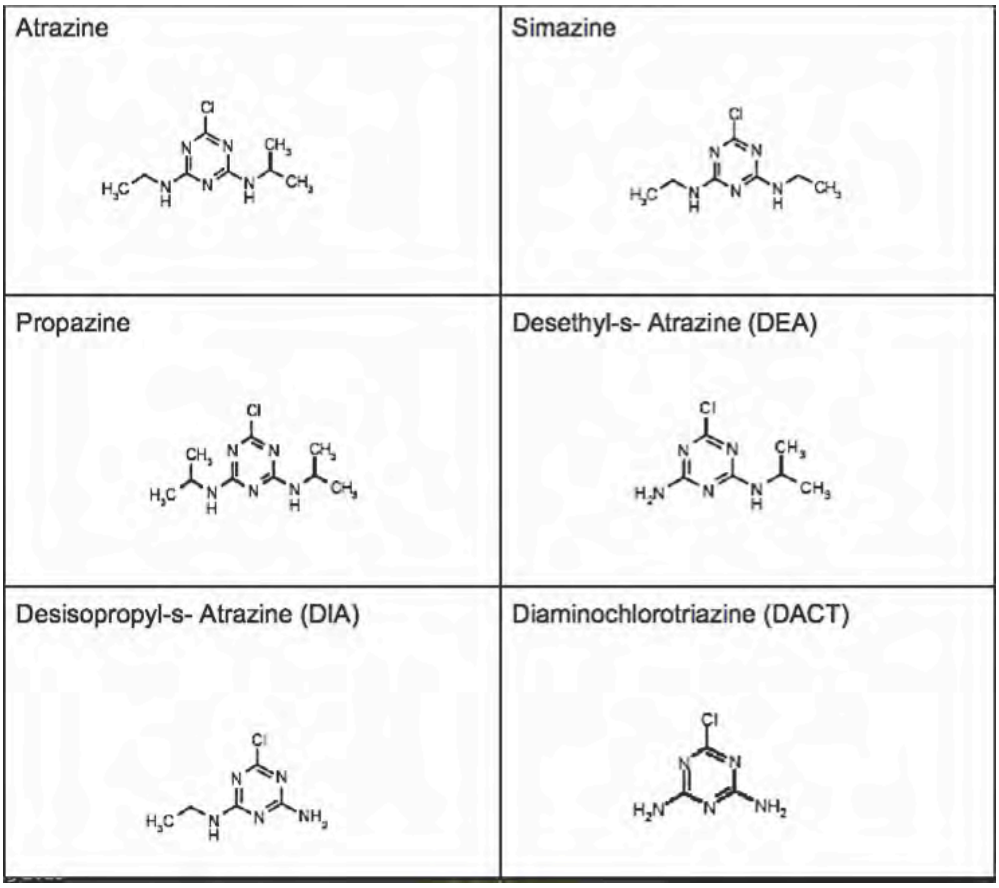
[57] Engdahl FW, 2007, Doomsday Seed Vault in the Arctic, *Global Research*. Available at: http://www.globalresearch.ca/doomsday-seed-vault-in-the-artic/23503 last accessed 16 January 2013

[58] Fagin D, Lavelle M & the Center for Public Integrity, 1996, *Toxic Deception, How the Chemical Industry Manipulates, Science, Bends the Law and Endangers Your Health*, Carole Publishing Group, Secaucus, NJ

[59] J Rohr, T Sage, TM Sesterham & BD Palmer, 2006, 'Exposure, Postexposure and Density-Mediated Effects of Atrazine on Amphibians: Breaking Down Net Effects into Their Parts' *Environmental Health Perspectives*, Vol 114(1), pp 46-50

mechanism group (CMG) due to their ability to cause neuroendocrine and endocrine-related developmental, reproductive and carcinogenic effects.[60]

Figure 7:1 Chemical structures of the triazines[61]



Atrazine is a persistent environmental pollutant. In soils, thirty percent of the original atrazine may exist after three years, whilst in water its relative stability leads to surface and ground contamination.[62] In acidic waters its half-life is measured in days but in groundwater it could be in the order of years due to an exceedingly slow rate of

[60] US, EPA, Office of Pesticide Programs, Health Effects Division, 2006, *Triazine Cumulative Risk Assessment*. Available at: http://epa.gov/oppsrrd1/REDs/triazine_cumulative_risk.pdf last accessed 19 July 2013

[61] US, EPA, Pesticides: Health and Safety, Triazine Cumulative Risk Assessment and Atrazine, Simazine, and Propazine Decisions; June 22, 2006. Available at: http://www.epa.gov/oppsrrd1/cumulative/triazine_fs.htm last accessed 9 September 2013

[62] Boey A & Cooper B, 1996, *Atrazine and its ecological significance*, TS 96.084, Central & North West Regions Water Quality Program, Centre for Natural Resources, Department of Land and Water Conservation, Parramatta

breakdown in water.[63] Atrazine breaks down more rapidly in warm conditions; for example in soils at 25 degrees Celsius it will break down three to four times faster than in cold or dry conditions or temperatures at 10 degrees Celsius.[64] Under ideal conditions atrazine will degrade to its three main chloro-metabolites - desethylatrazine, desisopropylatrazine and diaminochlorotriazin.[65] These metabolites are often more persistent than their corresponding parent compounds.[66] The US Geological Survey researchers estimated that the atrazine metabolite desethylatrazine persisted in groundwater for twenty-five years.[67] Compounding the problem, when metabolite residues are combined with parent residues, estimates of water contamination have the potential to be substantially higher.[68] Furthermore, studies of degradation routes are complex and costly and it is often very difficult to identify the minor degradates (breakdown properties) of a parent compound in a system.[69] Exposure to atrazine in the environment occurs through drinking water, inhaling air or dust or by accidental spills.[70]

7.5.1 Atrazine – a chemical of concern?

In the wildlife cancer case studies, which are covered from section 7.7 in this chapter, detection of atrazine occurs in the environment of all four species, although no studies have been undertaken to investigate a causal relationship between the chemical and the

---

[63] Radcliffe JC, 2002, *Pesticide Use in Australia,* Australian Academy of Technological Sciences and Engineering, Parkville, Victoria
[64] Qiao X, Ma L & Hummel HE, 1996, 'Persistence of Atrazine and Occurrence of Its Primary Metabolites in Three Soils' *Journal of Agricultural Food Chemicals*, 1996, Vol 44, pp 2846-2848
[65] National Registration Authority for Agricultural and Veterinary Chemicals, *The NRA Review of Atrazine*, 1997. Available at: http://www.apvma.gov.au/chemrev/downloads/atrazine_prs.pdf last accessed 18 September 2007, p 18
[66] Boxall ABA, Sinclair CJ, Fenner K, Dolpin D & Maund SJ, 2004, 'When Synthetic Chemicals Degrade in the Environment' *Environmental Science and Technology*, Vol 38(19), pp 369-375
[67] Cox C, 2001, 'Atrazine: Environmental Contamination and Ecological Effects' *Journal of Pesticide Reform*, Vol 21(3), pp 12-20
[68] Fan AM & Alexeeff GV & Office of Environmental Health Hazard Assessment, California Environmental Protection Aency, 1999, 'Public Health Goal for Atrazine in Drinking Water'. Available at: http://oehha.ca.gov/water/phg/pdf/atraz_f.pdf last accessed 5 November 2013
[69] Boxall ABA, Sinclair CJ, Fenner K, Dolpin D & Maund SJ, 2004, 'When Synthetic Chemicals Degrade in the Environment' *Environmental Science and Technology*, Vol 38(19), pp 369-375
[70] Alavanja MCR, Hoppin JA & Kamel F, 2004 'Health Effects of Chronic Pesticide Exposure: Cancer and Neurotoxicity' *Annual Review of Public Health*, Vol 25, pp 155-97

cancers. There are however, numerous studies into the harmful effects of atrazine including a recent study on zebrafish, which identifies changes to genes associated with neuroendocrine and reproductive function, cell cycle regulation and cancer in response to developmental exposure to atrazine.[71] Many other studies show the harmful effects of atrazine as an endocrine (hormone) disrupting chemical,[72] and a carcinogen in laboratory[73] and epidemiological studies.[74] In the US the *National Toxicology Report 2011* listed hexachlorobenzene, a by-product of the production of atrazine, as a

---

[71] US, EPA, Atrazine. Chemical Summary, 2007. Available at: http://www.epa.gov/teach/chem_summ/Atrazine_summary.pdf last accessed 19 July 2013

[72] Kniewald Z, Simic B, and Kniewald J, 2009, Atrazine inhibits reproductive processes in rats. *Biology of Reproduction*, Vol 78, pp 115-116; Kniewald J, Jakominic M, Tomijenovic A, Simic B, Romac P, Vranesic D & Kniewald Z, 2000, Disorders of male rat reproductive tract under the influence of atrazine, *Journal of Applied Toxicology*, Vol 20(1), pp 61-68; Šimic B, Kniewald J & Kniewald Z, 1994, Effect of atrazine on reproductive performance in the rat, *Journal of Applied Toxicology,* Vol 14(6), pp 401-404; Kniewald J, Osredecki V, Gojmerac T, Zechner V & Kniewald Z, 1995, Effect of s-triazine compounds on testosterone metabolism in the rat prostate, *Journal of Applied Toxicology*, 15(3), pp 215-218; Kniewald J, Mildner P & Kniewald Z, Effects of s-triazine herbicides on 5 -dihydrotestosterone receptor complex formation in the hypothalamus and ventral prostate in *Pharmacological Modulation of Steroid Action*, E. Genazzani, F. DiCarlo, and W.I.P. Mainwaring, (eds.), 1980, Raven Press: NY, pp. 159-169; Kniewald J, Mildner P & Kniewald Z, 1979, Effects of s-triazine herbicides on 5α -dihydrotestosterone receptor complex formation, 5α -reductase and 3β -hydroxysteroid dehydrogenase activity at the anterior pituitary level, *Journal of Steroid Biochemistry*, Vol 11(1C), pp 833-838; Šimic B, Kniewald Z, Davies JE & Kniewald J, 1991, Reversibility of inhibitory effect of atrazine and lindane on 5 -dihydrotestosterone receptor complex formation in rat prostate, *Bulletin of Environmental Contamination and Toxicology*, Vol 46(1), pp 92-100; Babic-Gojmerac T, Kniewald Z & Kniewald J, 1989, Testosterone metabolism in neuroendocrine organs in male rats under atrazine and deethylatrazine influence, *Journal of Steroid Biochemistry* Vol 33(1), p 141-146; Fan WQ, Yanase T, Morinaga H, Gondo S, Okabe T, Nomura M, KomatsuT, Morohashi K, Hayes TB, Takayanagi R & Nawata H, 2007, Atrazine-InducedAromatase Expression Is SF-1 Dependent: Implications for Endocrine Disruption in Wildlife and Reproductive Cancers in Humans, *Environmental Health Perspectives*, Vol 115(5), pp 720-727; Weber GJ, Sepulveda MS, Peterson SM, Lewis SS & Freeman JL, 2013, Transcriptome Alterations Following Developmental Atrazine Exposure in Zabrafish are associated with Disruption of Neuroendocrine and Reproductive System Function, Cell Cycle, and Carcinogenesis, *Toxicological Sciences,* 132(2), pp 458-466; McMullin TS, Andersen ME, Nagahara A, Lund TD, Pak T, Handa RJ & Hanneman WH, 2004, Evidence That Atrazine and Diaminochlorotriazine Inhibit the Estrogen/Progesterone Induced Surge of Luteinizing Hormone in Female Sprague-Dawley Rats Without Changing Estrogen Receptor Action, *Toxicological Sciences*, No. 79, pp 278-286

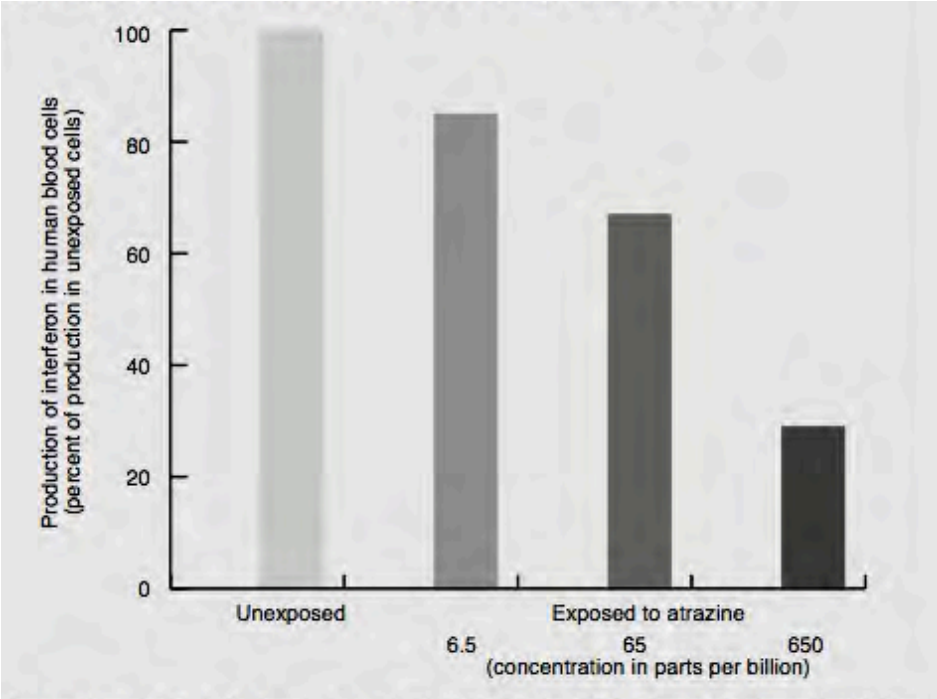
[73] Pintér A, Torok G, Borzsonyi M, Surjan A, Csik M, Kelecsenyi A & Kocsis Z, 1990, Long-term carcinogenicity bioassay of the herbicide atrazine in F344 rats, *Neoplasma*, Vol 37, pp 533-544; Wetzel LT, Luempert LG, Breckenridge CB, Tisdel MO, Stevens JT, Thakur AK, Extrom P & Eldridge C, 1994, Chronic Effects of atrazine on estrus and mammary tumor formation in female Sprague-Dawley and Fisher 344 rats, *Journal of Toxicology and Environmental Health*, Vol 43(2) pp 169-82;

[74] MacLennan PA, Delzell E, Sathiakumar N, Myers SL, Cheng H, Grizzle W, Chen VW & Wu XC, 2002, Cancer Incidence Among Triazine Herbicide Manufacturing Workers, *Journal of Occupational and Environmental Medicine,* Vol 44(11), pp 1048-1058; Mills PK, 1998, Correlation Analysis of Pesticide Use Data and Cancer Incidence Rates in California Counties, *Archives of Environmental Health*, Vol 53(6), pp 410-413; Rusiecki JA, De Roos A, Won JL, Dosemeci M, Lubin JH, Hoppin JA, Blair A, & Alavanja MCR, 2004, Cancer Incidence Among Pesticide Applicators Exposed to Atrazine in the Agricultural Health Study, *Journal of National Cancer Institute*, Vol 96(18), pp 1375-1382

carcinogen and N-Nitrosodiethanolamine, contained in atrazine, is reported to produce tumours in two rodent species.[75]

Meanwhile, a credible body of evidence exists linking atrazine to endocrine disruption. Atrazine has been identified as an endocrine disrupting chemical in more than two dozen human and animal disorders, including reproductive and developmental abnormalities, immune dysfunction, cognitive and behavioural pathologies and cancer.[76] Reduction in immune function, due to exposure to atrazine at concentrations of parts per billion, is shown in Figure 7:2 below.

Figure 7:2 Atrazine reduces the activity of the immune system[77]



[75] According to the US NIH hexachlorobenzene is listed as a carcinogen and is a by-product of the production of the chlorinated pesticides atrazine, propazine and simazine. NIH also note that N-Nitrosodiethanolamine which causes tumours in two rodent species is contained in atrazine pesticide formulation emulsified with triethanolamine at a concentration of 0.5mg/kg. National Toxocolgy Program, 2011, *Report on Carcinogens Twelfth Edition.* US Department of Health and Human Services. Available at*:* *http://ntp.niehs.nih.gov/?objectid=03C9AF75-E1BF-FF40-DBA9EC0928DF8B15* last accessed 24 May 2012

[76] Krimsky S, 2000, *Hormonal Chaos, The Scientific and Social Origins of the Environmental Endocrine Hypothesis*, The John Hopkins University Press, Baltimore and London

[77] Source: Hooghe RJ, Devos S & Hooghe-Peters EL, 2000, Effects of selected herbicides on cytokine production in vitro, *Life Science,* Vol 66, pp 2519-2525 in C Cox, 2001, Atrazine: Toxicology, Journal of Pesticide Reform, Vol 21(2) pp 12-20

Laboratory experiments have corroborated the association between atrazine exposure and increased infection and limb deformities in frogs shown here in Figure 7:3 below.[78]

Figure 7:3 Deformities in frogs[79]

The Center for Biological Diversity, in the US, has linked atrazine to declines of endangered amphibians and fish in California such as the California red-legged frog, California tiger salamander, Delta smelt, Coho and Chinook salmon, and steelhead trout.[80] Atrazine also harms other endangered species including sea turtles in Chesapeake Bay, Barton Springs salamanders in Texas, endangered mussels in Alabama, shortnose sturgeon in Midwest waters, the Wyoming toad and the Illinois cave amphipod.[81]

---

[78] Kiesecker JM, 2002, Synergism between trematode infection and pesticide exposure: A link to amphibian limb deformities in nature? *Proceedings of the National Academy of Sciences*, Vol 99(15), pp 9900-9904

[79] Source: Pieter Johnson, Amphibiaweb, 2004, Amphibian Deformities. Available at: http://amphibiaweb.org/declines/deformities.html last accessed 13 July 2013

[80] Center for Biological Diversity, 2009, New Research: Herbicide Atrazine Linked to Cancer, Birth Defects, Endocrine Disruption, and Endangered Species Impacts. Available at: http://www.biologicaldiversity.org/news/press_releases/2009/atrazine-08-27-2009.html last accessed 28 June 2012

[81] ibid.

Atrazine is also known to act in synergy with other chemicals.[82] A substantial body of peer-reviewed scientific studies exist on the triazines, the group of chemicals of which atrazine is a member. In France, Sandrine Roulland and colleagues linked high pesticide use with increasing incidence of non-Hodgkin's lymphoma (NHL). In their study they also noted that research has found associations between risk of NHL and exposure to phenoxyacetic acid herbicides, triazine herbicides, carbamates or organophosphate insecticides.[83] In a study in the US, Kettles and colleagues linked the triazine herbicides with a statistically significant increase in breast cancer risk with medium and high levels of exposure; although due to the limitations inherent in the ecologic study design, causality could not be drawn.[84] A study in Ontario, Canada found an association between atrazine and nitrate in drinking water with stomach cancer.[85] A study by Jane Schroeder and colleagues found that a causal relationship between agricultural exposures to dieldrin, toxaphene, lindane, atrazine and fungicides and a certain type of NHL are plausible, they cautioned however, associations should be confirmed in a larger study.[86] These are studies indicating the potential for harm from the use of the triazines including atrazine, in combination with other chemicals.[87]

---

[82] Cox C, 2001, Atrazine: Environmental Contamination and Ecological Effects, *Journal of Pesticide Reform,* Vol 21(3), pp 12-20

[83] Roulland S, Lebailly P, Lecluse Y, Briand M, Pottier D & Gauduchon P, 2004, Characterization of the t(14;18)BCL2-IGH Translocation in Farmers Occupationaly Exposed to Pesticides, *Cancer Research*, Vol 64, pp 2264-2269

[84] Kettles MA, Browning SR, Prince TS & Horstman SW, 1997, Triazine Herbicide Exposure and Breast Cancer Incidence in Ecologic Study of Kentucky Counties, *Environmental Health Perspectives*, Vol 105(11), pp 105-111

[85] Van Leeuwen JA, Waltner-Toews D, Abernathy T, Smit B & Shoukri M, 1999, Associations between stomach cancer incidence and drinking water contamination with atrazine and nitrate in Ontario (Canada) agroecosystems, 1987-1991, *International Journal of Epidemology*, Vol 28 pp 836-840

[86] Schroeder JC, Olshan AF, Baric R, Dent GA, Weinberg CR, Yount B, Cerhan JR, Lynch CF, Schuman LM, Tolbert PE, Rothman N, Cantor KP & Blair A, 2001, Agricultural risk factors for t(14;18) subtypes of non-Hodgkin's lymphoma, Epidemiology, Vol 12(6), pp 701-709

[87] Rohr JR & McCoy KA, 2010, A Qualitative meta-analysis reveals consistent effects of atrazine on freshwater fish and amphibians, *Environmental Health Perspectives*, 118(11) pp 20-32

Despite the mounting evidence that atrazine is a persistent environmental contaminant and is hazardous to wildlife and humans, Syngenta, the manufacturer, has continued to deny this is the case and insists that atrazine is safe. Jennifer Sass, senior scientist with the US Natural Resources Defense Council's (NRDC) health and environment program in an interview with Azadeh Ansari of *Cable News Network* (CNN), says Syngenta's 'tactic is to flood the scientific literature with negative data to negate other studies' and that it is only Syngenta's studies that 'show atrazine is not an endocrine disrupter'.[88] Syngenta has played a major role in keeping the controversial herbicide on the market and limiting restrictions on its registration and use. This is further discussed in the next chapter.

In the next section I outline four wildlife cancers, the type of cancer in each species, the contamination of their habitat, an overview of the scientific pathways and finally undertake comparisons between all four case studies.

7.6 Scientific studies into wildlife cancers

The most prominent cancers in animals have occurred in either captive or domesticated species because of their longer lifespan and the greater chance of contact with suspected carcinogenic chemicals. However, it is an increase in clusters of wildlife cancer that is now raising concern. In each case of wildlife cancers examined the aetiology of the disease is uncertain but the scientific communities are working on various hypotheses as to the possible causes. The pathways however as observed by Grandjean, follow established possible causes. These include a virus in both the Green sea turtles and the California sea lions and polycyclic aromatic hydrocarbons (PAHs), a ubiquitous

[88] Ansari A, 2010, Weed killer 'castrates' male frogs, study says. CNN. Available at: http://edition.cnn.com/2010/TECH/science/03/01/pesticide.study.frogs/index.html last accessed 16 January 2013

pollutant, in the St Lawrence River Estuary Beluga whales. On the other hand, in the Tasmanian devil cancer the research pathway follows a transmissible cancer, the cause of which is uncertain.

In analysing the various approaches used in the four wildlife cancers I have again used the concept of undone science as described in Chapter 2. For each species I describe the type of cancer that occurs and look at the distribution and the habitat of each affected population. From this analysis I draw conclusions, based on comparisons between the different scientific approaches, as to whether research has been left undone due to various forms of ignorance or for practical or political reasons.

7.7 Green sea turtle cancer in Florida, Hawaii, the Caribbean and Australia

7.7.1 Type of cancer

Green sea turtles (*Chelonia mydas*) are found in warm and temperate seas throughout the world. Populations inhabiting Florida, Hawaii, the Caribbean and Moreton Bay, Australia are known to have the same cancer; it has been described as an epithelial fibropapilloma.[89] The tumours grow primarily on the skin and most often around the neck and shoulders as shown in Figure 7:4 below. In Hawaii researchers have found that the disease is more prevalent in juvenile Green sea turtles than in adults.[90]

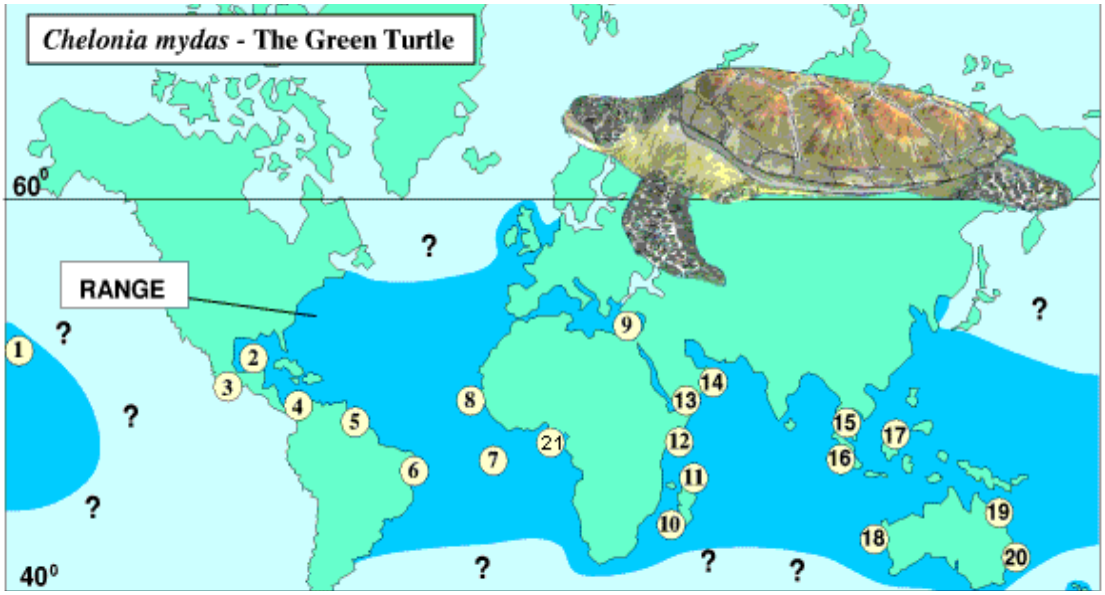
[89] Greenblatt RJ, Quackenbush SL, Casey RN, Rovnak J, Balazs GH, Work TM, Casey JW & Sutton CA, 2005, Variation of the Fibropapilloma-Associated Marine Turtle Herpesvirus across Seven Geographic Areas and Three Host Species, *Journal of Virology,* Vol 79(2), pp 1125-1132

[90] Work TM, 2005, Cancer in Sea turtles, *Hawaii Medical Journal*, Vol 64, pp 23-24

Figure 7: 4 Green sea turtle tumours[91]

7.7.2 Habitat

Figure 7:5 Green sea turtle world distribution[92]



[91] Tumours on eyes. Available at: http://www.turtlehospital.org/fibropapilloma.htm last accessed 1 December 2010. Dean Bagley, UCF. Available at: http://people.wcsu.edu/pinout/herpetology/cmydas/Body.html last accessed 4 October 2010

[92] Map available at http://www.euroturtle.org/outline/dgreen.htm last accessed 1 December 2010

The world distribution of Green sea turtles is shown in Figure 7:5 above. A feature of the habitats where Green sea turtles manifest the cancer is contamination by industrial, agricultural and urban pollution. In Florida there are 63 National Priorities List Sites of Hazardous Waste and according to the US Geological Survey the public water-supply wells in the northern Tampa Bay region are contaminated.[93] The most common pesticides detected were atrazine and its breakdown products, simazine and prometon.[94] In Hawaii, in the National Water Summary 1986 – Ground-Water Quality, atrazine along with other chemicals were detected in various wells and aquifers.[95] In the Caribbean, the Sea Turtle Conservancy, formerly the Caribbean Conservation Corporation, noted pollution in ocean and near shore waters is linked to the sea turtle cancer.[96] According to an article in *Scientific American* in 2013 the situation is unchanged, 'sea turtles are highly contaminated with industrial chemicals and pesticides'.[97]

In Moreton Bay, located in the southeast of Queensland, Australia, significant contamination of waterways draining into the Bay has also been reported. In particular, seagrass[98] declines in the Bay have been attributed to the worsening of water quality

[93] Metz PA, Delzer GC, Berndt MP, Crandall CA & Toccalino PL, 2006, *Anthropogenic Organic Compounds in Ground Water and Finished Community Water Systems in the Northern Tampa Bay Area, Florida,* Scientific Investigations Report, US Geographical Survey. Available at: http://pubs.usgs.gov/sir/2006/5267/ last accessed 20 May 2013
[94] ibid.
[95] US Environmental Protection Agency, *National Water Summary 1986: Hydrologic Events and Ground-Water Supply.* Accessed at: http://yosemite1.epa.gov/ee/epalib/eelib.nsf/85289d60ed01f8f985256a290076d16c/6e07f7eb7235a2d3852564c00001a241!OpenDocument on 13 August 2011
[96] Sea Turtle Conservancy, Sea Turtle Threats: Marine Pollution. Available at: http://www.conserveturtles.org/seaturtleinformation.php?page=pollution last accessed 20 May 2013
[97] Israel B, 2013, Long-lasting Chemicals May Harm Sea Turtles, Scientific American. Available at: http://www.scientificamerican.com/article.cfm?id=long-lasting-chemicals-may-harm-sea-turtles last accessed 20 May 2013
[98] Seagrass is a major part of the diet of Green Sea Turtles from ages five to ten years. Arthur KE, McMahon KM, Limpus CJ & Dennison WC, 2009, Ecology of Green Turtles (Chelonia mydas) from Shoalwater Bay, Australia, *Marine Turtle Newsletter*, No. 123, pp 6-12

due to an increase in contaminants and toxins.[99] The combined Moreton Bay catchment includes 14 major river catchments and 6 drainage basins. Contamination occurs from both point pollution, for example sewerage treatment and industrial waste, and from diffuse (or non-point) pollution. Chemicals detected in the Noosa River, which flows into Moreton Bay, include atrazine, endosulfan sulphate, trichlorfon, carbendazim and the wetting agent nonylphenol.[100] All are endocrine disrupters which interfere at critical times in reproductive and developmental processes in living organisms at extremely low levels (parts per billion).[101] There is a higher incidence of the disease in green turtles from the inshore soft-bottomed seagrass habitats in contrast to the coral reef habitats.[102] There is also a higher incidence of fibropapilloma in Green sea turtles than loggerhead turtles living in the same habitat.

7.7.3 Scientific pathway

Various scientific pathways have been adopted for studying the Green sea turtle cancer. To date the scientific evidence suggests a fibropapilloma tumour, which in other animals is spread by a virus.[103] It is hypothesized that a herpesvirus is an important aetiological factor.[104] A viral aetiology is suspected as DNA from an alphaherpes virus has been associated with tumoured tissue, but according to Herbst and Klein whether it

---

[99] Marine Species Section Approvals and Wildlife Division, 2003, *Recovery Plan for Marine Turtles in Australia,* Environment Australia, Canberra, p 27. Haynes D, Muller J & Carter S, 2000, Pesticide and Herbicide Residues in Sediments and Seagrasses from the Great Barrier Reef World Heritage Area and Queensland Coast, *Marine Pollution Bulletin*, Vol 41(7-12), pp 279-287

[100] Gardiner P, 2010, Sex change chemicals in river, *Sunshine Coast Daily*. Available at: http://www.sunshinecoastdaily.com.au/story/2010/03/05/scientists-find-sex-change-chemicals-in-noosa-rive/ last accessed 30 March 2011

[101] Hamlin HJ & Guillette LJ, 2010, Birth Defects in Wildlife: The Role of Environmental Contaminants as Inducers of Reproductive and Developmental Dysfunction, *Systems Biology in Reproductive Medicine*, Vol 56, pp 113-121

[102] Limpus CJ & Miller JD, 1994, The occurrence of cutaneous fibropapillomas in marine turtles in Queensland, *Proceedings of the Australian marine turtle conservation workshop*, Ed. R James, Queensland Department of Environment and Heritage and Australian Nature Conservation Agency, Canberra, Australia

[103] Flint M, Limpus CJ, Patterson-Kane JC, Murray PJ & Mills PC, 2010, Corneal Fibropapillomatosis in Green Sea turtles (*Chelonia mydas*) in Australia, *Journal of Comparative Pathology*, Vol 142, pp 341-346

[104] ibid.

is the cause, or just happens to be found in association with the tumoured tissue, is unknown.[105] Herbst and Klein also claim recent transmission studies point to an infectious aetiology, but it is not known how this might occur.[106] To date no studies indicate genetics is involved but studies to sequence the genome of the green turtle herpesvirus have begun.[107] Studies indicate that Green sea turtles in Moreton Bay have a suppressed immune system associated with chemical pollution.[108] But Herbst and Klein suggest that co-carcinogenesis and contaminant-induced immune suppression could be involved.[109] Greenblatt et al support this hypothesis, that environmental factors particularly water pollutants likely play a role in the cancer pathogenesis.[110] Studies have also been undertaken to assess the role of compounds produced by cyanobacterium in the development of the Green sea turtle disease.[111] Although the findings showed no conclusive relationship between cause and effect, the authors suggest these naturally produced compounds should be considered in the aetiology of the disease.

---

[105] Work T.M, 2005, Cancer in Sea Turtles. *Hawaii Medical Journal*, Vol 64, pp 23-24

[106] Herbst LH & Klein PA, 1995, Green Turtle Fibropapillomatosis: Challenges to Assessing the Role of Environmental Cofactors, *Environmental Health Perspectives*, Vol 103, Supplement 4, pp 27-30

[107] Greenblatt RJ, Quackenbush SL, Casey RN, Rovnak J, Balazs GH, Work TM, Casey JW & Sutton CA, 2005, Genomic Variation of the Fibropapilloma-Associated Marine Turtle Herpesvirus across Seven Geographic Areas and Three Host Species, *Journal of Virology*, Vol 79(2), pp 112-1132

[108] Van de Merwe JP, 2008, *Persistent organic pollutants and heavy metals in the green sea turtle, Chelonia mydas,* PhD, Griffith School of Environment and Australian Rivers Institute, Griffith University, Queensland, Australia

[109] Herbst LH & Klein PA, 1995, Green Turtle Fibropapillomatosis: Challenges to Assessing the Role of Environmental Cofactors, *Environmental Health Perspectives*, Vol 103, Supplement 4, pp 27-30

[110] Greenblatt RJ, Quackenbush SL, Casey RN, Rovnak J, Balazs GH, Work TM, Casey JW & Sutton CA, 2005, Genomic Variation of the Fibropapilloma-Associated Marine Turtle Herpesvirus across Seven Geographic Areas and Three Host Species, *Journal of Virology*, Vol 79(2), pp 112-1132

[111] Arthur K, Limpus C, Balazs G, Caper A, Udy J, Shaw G, Keuper-Bennett U & Bennett P, 2008, The exposure of green turtles (*Chelonia mydas*) to tumour promoting compounds produced by the cyanobacterium *Lyngbya majuscule* and their potential role in the aetiology of fibropapillomatosis, *Harmful Algae*, Vol 7, pp 114-125

7.8 St Lawrence Estuary (SLE) Beluga whales, Canada

7.8.1 Type of cancer

In 2002 Daniel Martineau and colleagues carried out a study of Beluga whale carcasses reported stranded in the St Lawrence Estuary between 1983 and 1999.[112] They found the main cause of death was cancer (27% incidence).[113] It is higher than the death rate from cancer of any other wild mammal species. Cancers detected included mammary gland cancer – a first for marine mammals. A proximal intestine cancer was identified, as shown in Figure 7:6 below, in 30% of the stranded SLE Beluga whales.[114] This type of cancer is rare but it is etiologically associated with the ingestion of herbicides such as 2,4-D (2,4-dichlorophenoxyacetic acid). [115]

[112] Martineau D, Lemberger K, Dalaire A, Labelle P, Lipscombe TP, Pascal M & Mikaelian I, 2002, Cancer in Wildlife a case study: Beluga from the St Lawrence Estuary, Quebec, Canada, *Environmental Health Perspectives*, Vol 110(3), pp 285-292
[113] Discover science and conservation, Whales. Available at: www.whales-online.net/eng/FSC.html?sct=2&page=2.1.27.html last accessed 31 December 2008
[114] Martineau D, Lemberger K, Dallaire A, Labelle P, Lipscomb TP, Michel P & Mikealian I, 2002, Cancer in Wildlife, a Case Study: Beluga from the St. Lawrence Estuary, Quebec, Canada, *Environmental Health Perspectives*, Vol 110(3), pp 285-292
[115] ibid.

Figure 7.6 Intestinal cancer – Beluga whale

7.8.2 Habitat

Concerned about the level of contamination in the St Lawrence River, the St Lawrence Centre[116] established a research programme to quantify contaminants in the drainage basin.[117] The results revealed that, of the twenty-two pesticides monitored, atrazine and metolachlor were the most frequently detected and at the highest concentrations.[118] Claire Lemieus and Ken Lum found the Great Lakes contributed 68% of the chemical loading of atrazine while Quebec tributaries accounted for only 8% but there was an unmeasured source of 24%.[119] It was noted that other researchers had linked the deposition of atrazine to its concentrations in the rain and air.[120] Hence because of its

[116] The St Lawrence Centre is a research centre, it operates under the Government of Canada body Environment Canada in Montreal Quebec. Available at: http://www.universadecouvrir.gc.ca/page/index.php?l=e&p=86 last accessed 15 April 2011
[117] Pesticides are Entering the St Lawrence River through its Tributaries, St Lawrence Centre, Canada. Available at: http://www.qc.ec.gc.ca/csl/inf/inf044e.html, last accessed 6 February 2008
[118] Environment Canada, 2013, Pesticides are Entering the St. Lawrence River through it Tributaries. Available at: http://www.ec.gc.ca/stl/default.asp?lang=En&n=45B1191F-1 last accessed 2 November 2013
[119] Lemieux C & Lum KR, 1996, Sources, Distribution and Transport of Atrazine in the St. Lawrence River (Canada), *Water, Air, and Soil Pollution*, Vol 90, pp 355-374
[120] Eisenreich SJ & Strachan WMJ, 1992, Estimating Atmospheric Deposition of Toxic Substances to the Great Lakes – An Updata, Report on the workshop held at the Canada Centre for Inland Waters,

prevalence, its persistence and its potential toxicity, atrazine was identified as a contaminant of concern.[121] Atrazine is thought to inhibit photosynthesis and hence has the potential to affect the growth of phytoplankton and the dynamics of the aquatic food chain.[122] Beluga whales in the St Lawrence estuary are known to have the cancer whereas Beluga whales in the much less contaminated Arctic appear not to have the cancer.[123]

7.8.3 Scientific pathways

The various pathways used by the scientific research community enquiring into the SLE Beluga whales include a suspected viral aetiology, possibly infectious. Toxicology testing has so far detected high concentrations of organochlorines, as well as benzo[a]pyrene (BaP) exposure, polychlorinated biphenyls (PCBs), dichlorophenyl trichloroethane (DDT) and polycyclic aromatic hydrocarbons (PAHs) in the tissue of stranded SLE Beluga whales but these were not in Arctic beluga whale tissue.[124]

PBDEs have also been found in the blubber of Beluga whales from the St Lawrence estuary as well as those from the western Hudson Bay in the Canadian Arctic.[125] The detection of elevated levels of CYP enzymes suggests a possible exposure to high levels of polychlorinated biphenyls (PCBs). Lungworms have targeted the St Lawrence

---

Burlington, Ontario, January 21-February 2, 1992, p 59 and Hall JC, Van Deynze TD, Struger J & Chan CH, 1993, Enzyme Immunoassay Based Survey of Precipitation and Surface Water for the Presence of Atrazine, Metolachlor and 2,4-D, *Journal of Environmental Science and Health*, Vol B28(5), pp 577-598

[121] Lemieux C & Lum KR, 1996, Sources, Distribution and Transport of Atrazine in the St Lawrence River (Canada), *Water, Air and Soil Pollution*, Vol 90, pp 355-374

[122] ibid

[123] De Guise S, Martineau D, Beland P & Fournier M, 1995, Possible Mechanisms of Action of Environmental Contaminants on St. Lawrence Beluga Whales (*Delphinapterus leucas*), *Environmental Health Perspectives,* Vol 103(4), pp 73-77

[124] Martineau D, Lemberger K, Dalaire A, Labelle P, Lipscombe TP, Pascal M & Mikaelian I, 2002, Cancer in Wildlife a case study: Beluga from the St Lawrence Estuary, Quebec, Canada, *Environmental Health Perspectives*, Vol 110(3), pp 285-292

[125] McKinney MA, De Guise S, Martineau D, Beland P, Lebeuf M & Letcher RJ, 2006, Organohalogen contaminants and metabolites in beluga whale (Delphinapterus leucas) liver from two Canadian populations, *Environmental Toxicology and Chemistry*, Vol 25(5), pp 1246-57

Beluga whales and it is proposed this may be linked to the PCBs, which are immunosuppressive compounds.[126] According to Sylvain De Guise and colleagues, the lesions in most of the SLE Beluga whale target organs have been identified in toxicological studies of other species and they propose that these long-lived (30 years) whales 'reflect particularly well the risks associated with life in a polluted ecosystem'.[127] Other research has found that SLE Beluga whales have a reduced level of genetic variation, which was not found in Beaufort Sea Beluga whales suggesting these individuals may be closely related.[128] No connection has been made between the reduced genetic variation in the whales and the incidence of cancer.

7.9. California sea lions, United States

7.9.1 Type of cancer

The predominant cancer in California sea lions, in both sexes, is a urogenital (urinary tract) carcinoma, which is epithelial (skin).[129] The cancer was first discovered in a group of sea lions on Pier 33 near Fisherman's Wharf in the San Francisco Bay. A California sea lion undergoing a post mortem is shown in Figure 7:7 below.

---

[126] Measures LN, Beland P, Martineau D & De Guise S, 1995, Helminths of an endangered population of belugas, Delphinapterus leucas, in the St Lawrence estuary, Canada, *Canadian Journal of Zoology*, Vol 73, pp 1402-1409

[127] De Guise S, Martineau D, Beland P & Fournier M, 1995, Possible Mechanisms of Action of Environmental Contaminants on St. Lawrence Beluga Whales (Delphinapterus leucas), *Environmental Health Perspectives*, Vol. 103, Supplement 4, pp 73-77

[128] Patenaude NJ, Quinn JS, Beland P, Kingsley M & White BN, 1994, Genetic variation of the St Lawrence beluga whale population assessed by DNA fingerprinting, *Molecular Ecology*, Vol 3(4), pp 375-381

[129] Ylitalo GM, Stein JE, Hom T, Johnson LL, Tilbury KL, Hall AJ, Rowles T, Greig D, Lowenstine LJ & Gulland FMD, 2005, The role of organochlorines in cancer-associated mortality in California sea lions (Zalophus californianus), *Marine Pollution Bulletin*, Vol 50, pp 30-39

Figure 7:7 Post mortem examination on a California sea lion with cancer[130]

7.9.2 Habitat

The Gulf of California is heavily polluted and from the late 1940s until the early 1970s millions of pounds of DDTs and polychlorinated biphenyls (PCBs) were discharged into the sea.[131] Herbicides most commonly detected in urban streams in a 2006 report were simazine, prometon, tebuthiuron, 2,4-D and diuron, and insecticides, diazinon, chlorpyrifos and carbaryl.[132] Waters from the surrounding urbanized and agricultural areas drain into the Bay making it especially vulnerable to pollution.[133]

---

[130] Sea Lion Cancer Consortium, 2012, Workshop, The Marine Mammal Center, Sausalito, California. Available at: http://www.smru.st-andrews.ac.uk/slicc/workshop.html last accessed 9 July 2013

[131] Clean Estuary Partners, nd. Legacy pollution What does it mean for the health of the Bay? Available at: http://www.waterboards.ca.gov/sanfranciscobay/water_issues/programs/TMDLs/sfbaymercury/final_legacy_pollution.pdf last accessed 20 May 2013

[132] TDC Environmental, 2006, *Pesticides in Urban Surface Water*, San Francisco Estuary Project, San Mateo California, p 15

[133] Clean Estuary Partners, nd. Legacy pollution What does it mean for the health of the Bay? Available at: http://www.waterboards.ca.gov/sanfranciscobay/water_issues/programs/TMDLs/sfbaymercury/final_legacy_pollution.pdf last accessed 20 May 2013

7.9.3 Scientific pathways

The scientific research has included a hypothesis that a virus could be involved, possibly a gamma herpes virus. However, Drs Gulland and Lowenstine questioned if discharges of DDTs and PCBs into the sea are solely to blame asking 'why is the cancer originating mainly in the uro-genital tract and not in the kidney or liver where it would be expected?' Scientists speculated that the virus and environmental chemicals could be interacting to trigger the cancers.[134] Gulland and Lowenstine recently published research exploring the possibility that the contaminants (PCBs found in blubber[135]) interact with hormone receptors in the reproductive tract of sea lions to help promote cancer.[136] Meanwhile, according to Lowenstine, PCBs can suppress the immune system, which may increase the sea lions' vulnerability to the virus infection. Inbreeding has also been proposed as a contributing factor. In 2003 in Brief Communications in *Nature* Karina Acevedo-Whitehouse and colleagues proposed that inbreeding could have a significant impact on wildlife as inbred individuals could act as reservoirs of infectious agents; in the case of California sea lions with herpes virus infection.[137] In 2005 Lizabeth Bowen and colleagues added support to this hypothesis by proposing CSL class II MHC genes may confer susceptibility to the cancer, although they noted further studies are needed.[138]

---

[134] Buckles El, Lowenstine LJ, DeLong RL, Melin SR, Vittore RK, Wong HN, Ross GL, St Leger JA, Greig DJ, Duerr RS, Gulland FMD & Stott JL, 2007, Age-prevalence of Otarine Herpesvirus-1, a tumor-associated virus, and possibility of its sexual transmission in California sea lions, *Veterinary Microbiology*, Vol 120, pp 1-8

[135] Ylitalo GM, Stein JE, Hom T, Johnson LL, Tilbury KL, Hall AJ, Rowles T, Greig D, Lowenstine LJ & Gulland FMD, 2005, The role of organochlorines in cancer-associated mortality in California sea lions (Zalophus californianus), *Marine Pollution Bulletin*, Vol 50, pp 30-39

[136] Chen I, 2010, Cancer Kills Many Sea Lions, and Its Cause Remains a Mystery, *The New York Times.* Available at http://www.nytimes.com/2010/03/05/science/05sfsealion.html?_r=1&pagewanted=1 last accessed 4 October 2010

[137] Acevedo-Whitehouse K, Gulland F, Greig D & Amos William, 2003, Disease susceptibility in California sea lions, *Nature,* Vol 422, p 35

[138] Bowen L, Aldridge BM, DeLong R, Melin S, Buckles EL, Gulland F, Lowenstine LJ, Stott JL & Johnson ML, 2005, An immunogenetic basis for the high prevalence of urogenital cancer in a free-ranging population of California sea lions (Zalophus californianus), *Immunogenetics*, Vol 56, pp 846-848

7.10 Tasmanian devil cancer, Tasmania, Australia

7.10.1 Type of cancer

The Tasmanian devil cancer is hypothesized to be a neuro-endocrine tumour of possibly Schwann cell origin.[139] It is also hypothesized to be a contagious cancer, an allograft spread via biting. Richmond Loh and colleagues state that '[h]istological examination of the tumours found the neoplastic cells were located predominantly within the sub-epithelial connective tissue of the skin or oral cavity'.[140] As the tumours appear mainly on the face and neck as shown in Figure 7:8 below, it has been termed Devil Facial Tumour Disease (DFTD).

Figure 7:8 Devil Facial Tumour Disease (DFTD)[141]

7.10.2 Habitat

The habitat of the Tasmanian devil is heavily polluted with chemicals used in both agriculture and plantation forestry.[142] There has been an ongoing controversy over the

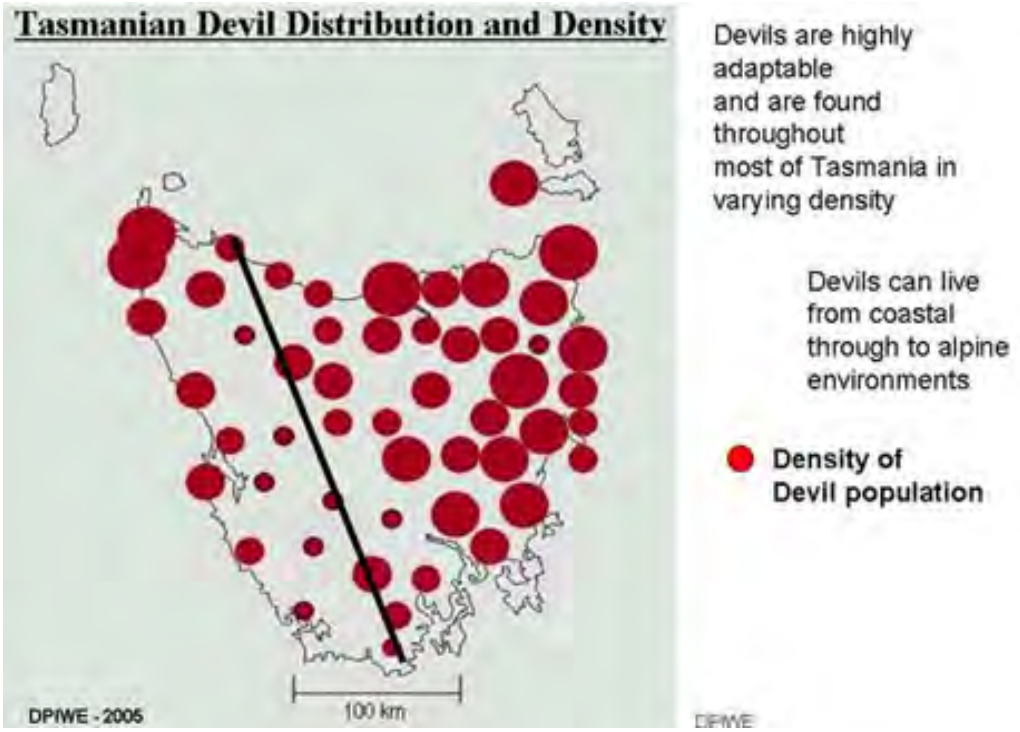
[139] Murchison EP, Tovar C, Hsu A, Bender HS, Kheradpour P, Rebbeck CA, Obendorf D, Conlan C, Bahlo M, Blizzard CA, Pyecroft S, Kreiss A, Kellis M, Stark A, Harkins TT, Graves JAM, Woods GM, Hannon GJ, Papenfuss AT, 2010, The Tasmanian Devil Transcriptome Reveals Schwann Cell Origins of a Clonally Transmissible Cancer, *Science*, Vol 327, pp 84-87

[140] Loh R, O'Hara M, Raidal S, Pyecroft S & Sharpe R, nd. Devil Facial Tumour Disease, What is happening to our devils? Tasmanian Department of Primary Industries, Water and Environment, Hobart, Tasmania and Murdoch University, Western Australia

[141] Source: Christo Baars. Available at: http://tasmaniantimes.com/index.php/article/the-devil-paradox last accessed 13 July 2013

widespread contamination of surface and now ground water in Tasmania.[143] Triazine chemicals including atrazine and simazine have been and continue to be detected in water monitoring in Tasmania. The issue of water contamination from plantation forestry practices is the topic of chapter 9. In Figure 7.9 below the size of the red dots indicate the density of Tasmanian devils across Tasmania. The line drawn through the state indicates the edge of the spread of DFTD. West of the line is DFTD free whilst the area east of the line, where DFTD has been most prevalent, is highly utilized for agriculture but mainly plantation forestry.

Figure 7:9 Map of Tasmanian devil distribution and density[144]



[142] Vetter W, Recke R von der, Symons R & Pyecroft S, 2008, Determination of polybrominated biphenyls in Tasmanian devils (Sarcophilus harrisii) by gas chromatography coupled to electron capture negative ion tandem mass spectrometry or electron ionization high-resolution mass spectrometry, *Rapid Communications in Mass Spectrometry*, Vol 22, pp 4165-4170

[143] Pollution Information Tasmania: Telling the Truth About Toxics in Tasmania. Available at: http://www.sourcewatch.org/index.php/Pollution_Information_Tasmania last accessed 31 March 2014

[144] Department of Primary Industries, Parks, Water and Environment, 2005. devils@cradle, Devil Facts, Tasmanian Devil Sanctuary, Cradle Mountain, Tasmania. Available at: http://devilsatcradle.com/content.php?id=devil-facts last accessed 13 July 2013

7.10.3 Scientific pathways

The scientific research has determined that a virus is not involved, as no virus particles have been found. The research pathway has been directed to support the allograft hypothesis as shown in previous chapters. A toxicology pilot program was conducted on a limited number of chemicals, as discussed previously in Chaper 5, which found PBBs in the devil fat. Their source is mainly eucalypt plantation forestry practices of aerial spraying of pesticides to be discussed in full in Chapter 9. No further studies have been undertaken. Genetics was hypothesized to be involved because the devils lacked MHC diversity as a result of inbreeding[145]. The immune system of the devils has been found to be structurally competent although important studies, as discussed previously in Chapter 4, on the immune system remain undone.

7.11 Comparison of the four case studies and their scientific pathways

A comparison of scientific pathways is shown in Table 7:1 below, which indicates the commonalities and differences between the four case studies of wildlife cancers. Although the cancer types are different in each case study they are all epithelial or skin related. In three of the cancers a virus is suspected. Also in three cases, except for the Beluga whales, the cancers are thought to be infectious. In all cases toxins in the environment have been identified in the animal bodies or are suspected. In two case studies inbreeding or genetics are implicated in the cancer. In all but one case, the Tasmanian devil, the immune system is suppressed. Although chemicals have been detected in some studies, no toxicological experiments have been undertaken to assess the effects of these chemicals on the target species. In all four case studies, atrazine,[146] a

[145] This hypothesis is still to be confirmed.
[146] The triazine, simazine with the same properties as atrazine but subject to fewer studies, has also been detected.

known endocrine disrupter, has been detected in water monitoring and identified as a chemical of concern but no studies have been undertaken in relation to this chemical.

Table 7:1 Comparison of scientific pathways in wildlife case studies

	Beluga whale	**Tasmanian devil**	**California sea lions**	**Green sea turtle**
Cancer type	Intestinal/epithelial Origin – transitional cell	Neuro-endocrine/sub-epithelial Origin – Schwann cell	Urogenital/epithelial Origin – transitional cell	Papilloma Origin – skin
Viral	Suspected	No	Gamma Herpes-virus association	Herpes-virus association
Infectious	No	Transmitted via biting	Transmitted sexually	Yes
Suspected Carcinogen	PAHs and PCBs found in blubber	PBBs found in fat	Organochlorines – PCBs	Suspected not established
Genetic		Inbreeding – lack MHC diversity	Inbreeding MHC linked to tumours	
Immune System	Suppressed	Competent	Suppressed	Suppressed
Toxicity Studies[147]	Nil	Nil	Nil	Nil

7.12 Comparison of other factors in the four case studies

Other relevant factors in the four case studies shown in a comparison in Table 7:2 below, indicate that all wildlife cancers occur within sub-populations of larger, cancer-free populations. The habitats of the four species are contaminated with industrial, agricultural and municipal pollution but the commonality is that all are contaminated with agricultural pollution, in particular atrazine. There are no similarities in their feeding. All are threatened to some degree under the IUCN Red List for endangered species. The threat of habitat contamination is only recognized in two of the cases of wildlife cancer. Whilst other threats include motor vehicles in Tasmania for Tasmanian

[147] No studies have been undertaken to test the effects of chemicals found in the animals or in their environments on the affected populations.

devils and incidental fishing, harmful algal blooms and stranding for the California Sea lions.

Table 7:2 Comparison of other relevant factors in wildlife case studies

	Beluga whales	**Tasmanian devil**	**Californian sea lions**	**Green sea turtle**
Sub-population affected	St Lawrence Estuary, Canada	East coast devils	San Francisco Bay, Gulf of California	Moreton Bay Hawaii, Florida, Bermuda
Habitat Contamination	Industrial, agricultural & municipal	Agricultural, possible mining	Industrial, agricultural & municipal	Industrial, agricultural and municipal
Feeding	Partially sediment feeding, fish and shellfish	Scavengers	Opportunistic predators; all food from the sea	Sea grass
Status	IUCN Red List – near threatened	IUCN Red List – Endangered EBPC Act - Endangered	IUCN – Red List Low Risk Least Concern	IUCN – Red List Endangered EPBC Act – Vulnerable
Recognised Threats	Habitat contamination	Road kill	Incidental fishing, harmful algal blooms & stranding	Habitat contamination

Although carcinogens are suspected in the Moreton Bay Green sea turtles, to date this has not been established. No studies to determine whether or not contaminants found in the Green sea turtle habitats are involved in the initiation or promotion of the cancer have been undertaken. Finding mutations in genes p53 and *ras* in Beluga whales would strongly support an aetiologic role of contaminants in carcinogenesis.[148] However, relevant studies have not been undertaken and it is suggested that a large study would be required to confirm the role of contaminants.

[148] Martineau D, Lemberger K, Dallaire A, Labelle P, Lipscomb TP, Michel P & Mikealian I, 2002, Cancer in Wildlife, a Case Study: Beluga from the St. Lawrence Estuary, Quebec, Canada, *Environmental Health Perspectives*, Vol 110(3), pp 285-292, p 290-291

In the case of the Tasmanian devil disease, following an initial pilot study of devil tissue and fat for toxins, which revealed levels of PBBs and PBCDs, no further toxin investigations have been undertaken.

Uncertainty exists as to the cause of the cancers in each case study. Research has been undertaken following established pathways into the known causes of cancer but no studies have been undertaken to date that attempt to discover the role of chemicals such as atrazine or the possibility of epigenetic effects in these wildlife cancers.

7.13 Conclusion

Recent developments in the relatively new scientific field of epigenetics and the role of endocrine disrupters in the initiation and progression of cancer are challenging the orthodox theory of cancer, thus increasing the knowledge of the complexity of cancer. More research needs to be undertaken to extend this knowledge and the wildlife cancers mentioned in this chapter could be the focus of those studies. The cancers in these four wildlife species occur in environments where detectable levels of pollution occur. The manufacturers of chemicals may not wish to fund research that might prove contrary to their interests. Therefore, governments and their regulators who are charged with acting in the public interest need to ensure that more research is undertaken.

The precautionary principle should be implemented, given the uncertainty surrounding the cause of the cancer in each of these cases, particularly in relation to the lack of toxicology studies. As identified in the European Environment Agency's Report, discussed in the previous chapter, a lack of action on the part of decision makers is often the result of undue influence and conflict of interest. This appears to be the situation in

relation to the Tasmanian devil cancer and in the next two chapters I discuss these impediments. In chapter 8 I discuss how undue influence by the chemical industry on the regulators in both the United States and Australia has hampered the restricting or banning of atrazine registration and use in those countries. In chapter 9 I establish that a conflict of interest exists in Tasmania both within the government and between the government and the forestry industry.

Chapter 8 – Impediments to the regulation of atrazine

8.1 Introduction

In 2003 Canada acted to restrict the use of atrazine, following Syngenta's withdrawal of support for its use, with the exception of corn.[1] In 2009 following a petition to the Canadian government, in relation to the findings of scientific studies confirming atrazine's adverse effects on amphibian populations, a joint response of federal departments and agencies supported the earlier restrictions.[2] Health Canada concluded that 'the use of atrazine on corn for weed control does not entail unacceptable risk to the environment'.[3]

In 2004 the European Union under their Registration, Evaluation, Authorisation and Restriction of Chemical substances (REACH) program implemented the precautionary principle to restrict the continued registration of atrazine because of its potential to contaminate groundwater.[4] The restrictions are based on findings of ground water contamination in European countries, such as France.[5] Ground water contamination is

[1] Proposed Acceptability for Continuing Registration, Re-evaluation of Atrazine, 2003, Pest Management Regulatory Agency, Canada. Available at: http://www.hc-sc.gc.ca/cpsspc/pest/part/consultations/_pacr2003-13/index-eng.php last accessed 16 January 2013
[2] Response of the Federal Departments and Agencies to Environmental Petition 283 Filed by Frank Woodcock under the *Auditor General Act,* 2009, Concerns regarding the pesticide Atrazine. Available at http://www.oag-bvg.gc.ca/internet/English/pet_283_e_32986.html last access 19 July 2013
[3] ibid.
[4] Commission Decision, 2004, Concerning the non-inclusion of atrazine in Annex I to Council Directive 91/414/EEC and the withdrawal of authorisations for plant protection products containing this active substance, *Official Journal of the European Union*, L78 pp 53-55
[5] Mirgain I, Schenck C & Monteil H, 1993, Atrazine contamination of ground waters in eastern France in relation to the hydrogeological properties of the agricultural land, *Environmental Technology*, Vol 14, pp 741-769

viewed as a major problem because it is extremely difficult and expensive to remove chemicals from groundwater.[6]

Whilst there have been impediments to a complete ban on the registration and use of atrazine in both Canada and the EU the challenge to impose tighter restrictions on the use of atrazine in the US and Australia has been a different story. The role of regulatory capture and undue influence in these regulatory regimes is the focus of this chapter.

8.2 Regulatory Capture

The idea that private interests may capture a government in order to foster its own interest is not new - it has its origins in Marx's view that "big business controls institutions'.[7] More recently, George J. Stigler introduced the concept of 'regulatory capture' into modern economic analysis.[8] The concept of regulatory capture describes a practice whereby those responsible for regulation shift from protecting the public interest to serving the interests of the industry.[9] Greg McMahon broadens this understanding of "capture" to include measures taken by responsible authorities that act to protect illegal or undemocratic practices, which the same authorities are legislated to

[6] US EPA, Getting Up to Speed, Ground Water Contamination. Available at: http://www.epa.gov/region1/students/pdfs/gwc1.pdf last accessed 16 January 2013
[7] Laffont J & Tirole J, 1991, 'The Politics of Government Decision-making: A theory of Regulatory Capture' *The Quarterly Journal of Economics*, Nov. 1991, pp 1090-1127, p 1089
[8] Boehm F, 2007, Regulatory Capture Revisited – Lessons from Economics of Corruption, PhD Economics, Anti-Corruption Training & Consulting and Research Center in Political Economy, Universidad Externado de Colombia. Available at: http://xa.yimg.com/kq/groups/22107528/670468172/name/Boehm+-+Regulatory+Capture+Revisited.pdf last accessed 13 July 2013
[9] Briody M & Prenzler T, 1998, The Enforcement of Environmental Protection Laws in Queensland: A Case of Regulatory Capture? *Environmental and Planning Law Journal*, Vol 15(1), pp 54-72

control.[10] According to McMahon, the "capture" is completed when the industry assists the regulator to defeat the regulatory regime and thereby gain exemptions for the industry. In the US Syngenta has used its considerable influence to gain continued registration of atrazine, despite it not being in the public interest.

Undue influence is another method of capture, described as 'a bag of dirty tricks' including the "revolving door"[11], direct personal enticement or 'outright corruption – the crudest form of capture'. [12] It also includes 'disinformation campaigns, compromising regulators through receipt of gifts and favours, discrediting determined regulators, non-disclosure of evidence, implied threats such as disinvestment and political patronage through party political donations'.[13] Laffont and Tirole support these findings, whilst adding techniques such as feasibility of monetary bribes, the expectation of future employment, (a form of "revolving door"), personal relationships and lobbying.[14] There is also threatening behaviour, which includes a resort to legal processes, such as defamation suits to silence opponents.[15] Some of these practices

[10] McMahon G, 2002, 'Regulatory Capture: Causes and Effects' in *Proceedings of the International Institute for Public Ethics Biennial Conference on Restructuring 'The Public Interest'*, Globalising World: Business, The Professions and the Public Sector, Brisbane, Australia, 4-7 October 2002. Available at: http://www.iipe.org/conference2002/papers/McMahon.pdf last accessed 1 May 2007

[11] A revolving door describes the movement of personnel between high-level government appointments and lucrative corporate positions supportive of business aims. Available at: http://www.herinst.org/BusinessManagedDemocracy/government/trade/bureaucrats.html last accessed 2 December 2013

[12] Cites Freitag, 1983 in M Briody & T Prenzler, 1998, The Enforcement of Environmental Protection Laws in Queensland: A Case of Regulatory Capture? *Environmental and Planning Law Journal*, Vol 15(1), pp 54-72, p 55

[13] Briody M & Prenzler T, 1998, The Enforcement of Environmental Protection Laws in Queensland: A Case of Regulatory Capture? *Environmental and Planning Law Journal*, Vol 15(1), pp 54-72 pp 55-56

[14] Laffont JJ & Tirole J, 1991, The Politics of Government Decision-Making: A Theory of Regulatory Capture, *The Quarterly Journal of Economics*, Vol 106(4), pp 1089-1127

[15] Briody M & Prenzler T, 1998, 'The Enforcement of Environmental Protection Laws in Queensland: A Case of Regulatory Capture? *Environmental and Planning Law Journal*, Vol 15(1), pp 54-72

have occurred in the cultural and political milieu that surrounds the regulatory process in relation to atrazine.

8.3 US EPA and the regulation of atrazine

In the US the registration of atrazine, its use and continued contamination of surface and drinking water, have been the site of controversy with a regulator clearly compromised. In the US atrazine contaminates 93.9% of drinking water samples tested by the USDA[16] and it is the second most used pesticide at 76.4 million pounds applied every year.[17] The US EPA has a mandate to prevent and reduce pesticide and industrial chemical risk to humans, communities and ecosystems.[18] According to the US Office of Management and Budget the EPA's 2012 budget includes $9 billion to continue to deliver on its mission – to protect human health and the environment.[19]

In a review of atrazine under the then Bush administration the US EPA rejected all data except that produced by Syngenta.[20] In taking this approach the US EPA not only ignored the evidence that atrazine acted as an endocrine disrupter it also sought to assist Syngenta to evade further restrictions. In 2003 EPA officials and Syngenta representatives in closed meetings devised a plan to avoid tighter restrictions, the details of which the EPA declined to release.[21] The plan called for Syngenta to monitor

[16] Tupper K, 2009, "Team Atrazine" tries out new talking points, *Ground Truth*, Pesticide Action Network. Available at: http://www.panna.org/blog/"team-atrazine"-tries-out-new-talking-points last accessed 17 September 2013
[17] US EPA, nd. Atrazine Background. Available at: http://www.epa.gov/pesticides/factsheets/atrazine_background.htm last accessed 17 September 2013
[18] US EPA, Science and Technology: Pesticides. Available at: http://www.epa.gov/gateway/science/pesticides.html last accessed 4 July 2012
[19] US Office of Management and Budget, Environmental Protection Agency, The Federal Budget Fiscal Years, 2012, Factsheet. Available at: http://www.whitehouse.gov/omb/factsheet_department_epa last accessed 4 July 2012
[20] Weiss R, 2004, 'Data Quality' Law Is Nemesis Of Regulation, *The Washington Post*. Available at: http://www.washingtonpost.com/wp-dyn/articles/A3733-2004Aug15.html last accessed 16 September 2013
[21] ibid.

atrazine use and contamination over three years, as a condition of its re-registration in forty US watersheds, and to monitor farmers' efforts to minimize contamination.[22] If concentrations rose above a level that the company agreed was "of concern", then the company was required to work with the farmer to try to reduce the levels.[23]

One consequence of this self-regulatory approach surfaced in August 2009 when Charles Duhigg reported, in a series of articles entitled "Toxic Waters" in the *New York Times,* how huge spikes in atrazine concentration in drinking water were hidden from the American public.[24] The US NRDC also issued a major report in 2009 titled *Atrazine: Poisoning the Well* in which Jennifer Sass, senior scientist with the NRDC's health and environment program, raised the alarm about atrazine.[25] Her statement said 'there is strong evidence that atrazine is an endocrine disrupting chemical interfering with critical reproductive hormones even at extremely low levels. This is because hormones in our bodies are active at very low levels, parts-per-billion or lower'.[26] The NRDC had recommended that atrazine use be phased out and that it be filtered out of US drinking water.

In 2009 under the Obama administration, Lisa P. Jackson took office as Administrator of the EPA. In October 2009, Jackson decided to re-review the growing body of

[22] US EPA, 2012, Pesticide Programs' Monitoring in Community Water Systems. Available at: http://www.epa.gov/oppsrrd1/reregistration/atrazine/atrazine_update.htm last accessed 16 January 2013
[23] Weiss R, 2004, 'Data Quality' Law Is Nemesis Of Regulation, *The Washington Post*. Available at: http://www.washingtonpost.com/wp-dyn/articles/A3733-2004Aug15.html last accessed 16 September 2013
[24] Duhigg C, 2009, Debating How Much Weed Killer Is Safe in Your Water Glass, *The New York Times*. Available at: http://www.nytimes.com/2009/08/23/us/23water.html?ref=us last accessed 25 June 2012
[25] Natural Resources Defense Council, 2010, *Atrazine: Poisoning the Well*. Available at: http://www.nrdc.org/health/atrazine/ last accessed 25 June 2012
[26] Hodai B & Graves L, 2012, Syngenta PR's Weed-Killer Spin Machine: Investigating the Press and Shaping the "News" about Atrazine, The Center for Media and Democracy's PR Water. Available at: http://www.prwatch.org/news/2012/02/11277/syngenta-prs-weed-killer-spin-machine-investigating-press-and-shaping-news-about?page=1 last accessed 31 January 2013

scientific evidence indicating atrazine is harmful at levels below existing toxicity standards and scheduled four Scientific Advisory Panels (SAPs) reviews.[27] The scheduled US EPA review on atrazine commenced in mid-2013.[28]

Meanwhile, in 2011 the American Council on Science and Health (ACSH) published *Scared to Death, How Chemophobia Threatens Public Health*.[29] John Entine, a science reporter from the American Enterprise Institute (AEI), was the author.[30] It contained a chapter in defence of atrazine. The AEI is a right-wing think tank which had also published on its website an article titled "Over-regulation fever at the White House" claiming the Australian APVMA had found, in a review of a recent study of atrazine,[31] that the laboratory work was flawed.[32] The APVMA's only reference to the study linking atrazine to feminization of frogs states that there is no evidence to warrant a reconsideration of the APVMA's regulatory settings.[33] The APVMA has also decided to re-examine the more recent studies on atrazine, which is discussed in the next section.

---

[27] US EPA, 2013, Atrazine Updates, Atrazine Evaluation Process, Atrazine SAP meetings. Available at: http://www.epa.gov/oppsrrd1/reregistration/atrazine/atrazine_update.htm last accessed 16 September 2013

[28] US EPA, 2013, Pesticides: Reregistration, Atrazine Updates. Available at: http://www.epa.gov/oppsrrd1/reregistration/atrazine/atrazine_update.htm#atrazine last accessed 6 November 2013

[29] Entine J, 2011, *Scared to Death, How Chemophobia Threatens Public Health*, The American Council on Science and Health, New York

[30] Hodai B & Graves L, 2012, Syngenta PR's Weed-Killer Spin Machine: Investigating the Press and Shaping the "News" about Atrazine, The Center for Media and Democracy's PR Water. Available at: http://www.prwatch.org/news/2012/02/11277/syngenta-prs-weed-killer-spin-machine-investigating-press-and-shaping-news-about?page=1 last accessed 31 January 2013

[31] Hayes TB, Collins A, Lee M, Mendoza M, Noriega N, Stuart AA & Vonk A, 2002, Hermaphroditic, demasculinized frogs after exposure to herbicide atrazine at low ecologically relevant doses, *Proceedings of the National Academy of Sciences of the United States of America,* Vol 99(8), pp 5476-5480

[32] Entine J, 2012, Over-regulation fever at the White House. Available at: http://www.aei.org/article/over-regulation-fever-at-the-white-house/ last accessed 26 June 2012

[33] Australian Government, Australian Pesticides and Veterinary Medicines Authority, 2011, Chemicals in the News: Atrazine. Available at: http://www.apvma.gov.au/news_media/chemicals/atrazine.php last accessed 28 July 2013

8.4 The Australian APVMA and the regulation of atrazine

Whilst the EU banned the use of atrazine and Canada restricted its use, the Australian regulator, the APVMA, has followed the US and re-registered atrazine with some restrictions.[34] The APVMA is the centralized regulatory authority for the assessment and registration of all agricultural and veterinary chemicals, more than 8,000 AgVet chemicals, prior to sale. The registration process determines whether the proposed product works as intended and if used according to label directions will have no harmful or unintended effects on people, animals, the environment or international trade.

In 1997 the then National Registration Authority for Agricultural and Veterinary Chemicals (NRA) released its report on the Review of Atrazine.[35] It found that increased restrictions on the use of atrazine introduced in 1995 had not been fully implemented by users. The then registrant, Ciba-Geigy, informed the NRA that atrazine use was critical to the success of plantation forestry. Regulatory actions taken following this Review included cancellation of industrial and non-agricultural uses of atrazine and the introduction of a range of label instructions to reduce the risk of atrazine entering waterways. In 2008 atrazine was re-registered with label instructions to further reduce the risk of atrazine entering waterways, information on withholding periods and additional information on reporting weed resistance.[36]

---

[34] Australian Government, Australian Pesticides and Veterinary Medicines Authority, Atrazine. Available at: http://www.apvma.gov.au/products/review/completed/atrazine.php last accessed 21 July 2013

[35] National Registration Authority, 1997, The NRA Review of Atrazine, APVMA. Available at: http://www.apvma.gov.au/products/review/completed/atrazine_summary.php last accessed 29 June 2012

[36] Australian Government, Australian Pesticides and Veterinary Medicines Authority, Atrazine. Available at: http://www.apvma.gov.au/products/review/completed/atrazine.php last accessed 21 July 2013

Similarly, in line with the EPA's practice of allowing Syngenta to self-regulate by monitoring atrazine contamination of water and soil, in Australia this self-monitoring was undertaken by the Forest Herbicide Research Management Group (FHRMG); a partnership between the APVMA and Syngenta.[37] In Tasmania Gunns Limited undertook monitoring in forestry plantations. More recently, monitoring has been conducted by the Department of Primary Industries, Parks, Water and Environment (DPIPWE) which has continued to find contamination levels of both atrazine and simazine in surface water and atrazine in ground water in Tasmania.[38]

In Australia the APVMA has a close relationship with the chemical and agricultural peak industry organization, CropLife Australia. APVMA Advisory Board members as at 10 January 2012 include the following affiliations:

- Mark Allison (chairman) is a life member and former Chairman of CropLife Australia and former president of Farmoz Pty Ltd;
- Claude Gauchat is a life member of CropLife Australia and former Executive director of Avcare Ltd. He was formerly a Division Manager from 1986-1992 at Ciba-Geigy (now Syngenta), the manufacturer of atrazine;
- Dr Simon Robinson is from the CSIRO Plant Industry Division;
- Roger Toffolon is Manager of the Biological and Chemical Risk Management Unit of the New South Wales state Department of Primary Industries;
- Wayne Cornish is Chairman of the South Australian Farmers Federation;
- Dr Richard Russell is the Managing Director of RAR Investments Pty Ltd whose investment partners include Prosafe Binatama[39] with clients including oil, gas and petro-chemical and manufacturing industry sectors.[40]

---

[37] Australian Pesticides and Veterinary Medicines Authority, 2004, *Second Draft Final Review Report*, Australian Pesticides and Veterinary Medicines Authority, Canberra, Australia.
[38] Tasmanian Government, Department of Primary Industries, Parks, Water and Environment, Pesticide monitoring in water catchments. Available at: http://www.dpipwe.tas.gov.au/inter.nsf/WebPages/CART-69STWK?open last accessed 28 July 2013
[39] Prosafe Binatama, nd. Our clients. Available at: http://prosafe-binatama.com/clients.php last accessed 5 August 2013
[40] CropLife Australia, Annual Review 2005-06. Available at: http://www.croplifeaustralia.org.au/files/aboutcroplife/annualreview/2005-06%20Annual%20Review%20-%20Website%20Version.pdf last accessed 13 April 2011

CropLife Australia has sought to directly influence the APVMA in regard to its Community Consultative Committee (CCC). In response to a newspaper article regarding the hazards of simazine (a triazine with the same properties as atrazine) the then Chief Executive Officer of CropLife, Paula Matthewson sent a letter (Appendix E) to the APVMA expressing concern that individual members of the CCC were using their membership to pursue their own agendas. It stated '[t]he CCC is no more than a convenient vehicle for activists to legitimize their outlandish and misleading campaigns'. Matthewson made the point that the Committee members 'receive sitting fees generously furnished by CropLife members through the APVMA's full cost recovery processes'. The letter commended the APVMA for its 'patient and ongoing rebuttals of the alarmist accusations being generated on a non-stop basis by the activists', declaring '[t]hey continue to achieve hyperbolic headlines with absolutely no scientific evidence to back it up'.

Further, in response to the suggestion that there is a large body of international studies into the harm caused by chemicals acting as endocrine disrupters, CropLife Australia issued a press release. It insisted that the 'APVMA must be allowed to independently assess chemicals for their true effects on the Australian environment without ideological pressure being placed upon them by ill-informed activists'.[41] To date no scientific studies into the effects of atrazine on Australia's unique fauna have been undertaken, consequently there is no Australian data. In fact little is known about the effects of many industrial and agricultural chemicals on Australia's marsupials.[42]

---

[41] Matthewson P, 2009, Pesticides regulator must be allowed to regulate pesticides, Media Release, CropLife Australia. Available at: http://www.croplifeaustralia.org.au/default.asp?V_DOC_ID=2238 last accessed 29 June 2012

[42] ASTEC, 1990, Australian Science and Technology Council: *Environmental Research in Australia. The Issues*. Australian Government Publishing Service, Canberra, Australia

According to Bolton and Ahokas the reproductive system in marsupials potentially makes them more vulnerable to effects of environmental chemicals.[43] In 2011 the results of a study on the toxicity of an organophosphorus insecticide, fenitrothion, to fat-tailed and stripe-faced dunnarts found an unexpectedly high sensitivity of these Australian marsupials to this chemical.[44] The authors also noted the scarcity of information on the effects of pesticides on native Australian vertebrates and the implications for biologically relevant risk assessment for the registration of pesticides. When studies into the effects of hazardous environmental toxins on native species are missing then it is prudent for the APVMA to act on the basis of the results of studies undertaken overseas that indicate harm from the use of endocrine disrupters such as atrazine.

Close connections also have existed between those serving on the APVMA and some figures involved with the Tasmanian forestry industry and the Tasmanian devil research. Dr Simon Cubit the manager of the APVMA's Regulatory Strategy and Compliance Program, was formerly a senior manager with Forestry Tasmania.[45] He is also listed as a team member on the Save the Tasmanian Devil Program.[46] Professor Michael Moore whilst on the board of the APVMA undertook to peer-review Gunns Limited's Integrated Impact Statement for the proposed pulp mill on behalf of

---

[43] Bolton RM & Ahokas JT, 1995, Review: Detoxication in Australian Marsupials – Ecotoxicological implications, *Australasian Journal of Ecotoxicology*, Vol 1, pp 85-98, p 95

[44] Story P, Hooper MJ, Astheimer Lb & Buttemer WA, 2011, Acute oral toxicity of the organophosphorus pesticide fenitrothion to Fat-tailed and Stripe-faced Dunnarts and its relevance for pesticide risk assessments in Australia, *Environmental Toxicology and Chemistry*, Vol 30(5), pp 1163-1169

[45] Dr Simon Cubit . Available at: http://www.zoominfo.com/#!search/profile/person?personId=843268213&targetid=profile last accessed 15 June 2012

[46] Save the Tasmanian Devil Program Team Contact Numbers. Available at www.tassiedevil.com.au last accessed 8 January 2013

UniQuest Pty Limited, giving approval for the pulp mill, albeit with restrictions. UniQuest Pty Limited is the main commercialization company of the University of Queensland and in 2009 it brokered a deal with Syngenta for exclusive licensing rights with CSR to develop sugar for ethanol biofuel.[47] Moore also evaluated the toxicology results of chemicals found in Tasmanian devils on behalf of Hamish McCallum and the DPIPWE.

8.4.1 Assessment by APVMA of recent studies on atrazine

In 2008 Matthew Denholm from *The Australian* newspaper published an article calling on the APVMA to act on the basis of a new study by Professor Ingraham from the University of California, San Francisco that demonstrated that atrazine had significant effects on human placental cells when exposed to as little as 20 parts per billion.[48] This study published by Miyuki Suzawa and Holly Ingraham in *PLoS One* in 2008 was the first to identify atrazine's full effect on human cells, including altering hormonal signaling.[4950] Simon Cubit, spokesperson for the APVMA, informed Denholm that the regulatory decision not to tighten atrazine restrictions was based on "weight-of-evidence" from many studies.[51]

---

[47] *NewsMaker*, 2009, UQ innovation boosts sugar company's global deal. Available at: http://www.newsmaker.com.au/news/2117 last accessed 14 April 2011

[48] Denholm M, 2008, Alarm at weed-kill chemical in water, *The Australian*. Available at: http://www.theaustralian.com.au/news/health-science/alarm-at-weed-kill-chemical-in-water/story-e6frg8gf-1111116343141 last accessed 2 July 2012

[49] Suzawa M & Ingraham HA, 2008, The Herbicide Atrazine Activates Endocrine Gene Networks via Non-Steroidal NR5A Nuclear Receptors in Fish and Mammalian Cells, *PLoS, One*, Vol 3(5), pp e2117 1-11

[50] Ravven W, 2008, Common herbicide disrupts human hormone activity in cell studies, University of California San Francisco. Available at: http://www.ucsf.edu/news/2008/05/5687/common-herbicide-disrupts-human-hormone-activity-cell-studies last accessed 18 September 2013

[51] ibid.

In March 2010 the APVMA requested two federal government departments - the Department of Sustainability, Environment, Water, Population and Communities and the Department of Health and Ageing - to assess the following studies:[52]

- Research by Prof Tyrone Hayes that atrazine turns male frogs into females[53]
- Research by Dr Sarah Waller and her team at the University of Washington in Seattle that links atrazine to the birth defect gastroschisis, looking at agricultural-related chemical exposures, season of conception, and risk of gastroschisis in Washington State[54]
- The effects of atrazine on freshwater fish and amphibians [Rohr and McCoy][55]

In June 2010 the APVMA published the results of the analysis, however only the following two studies were assessed:

- Agricultural-related chemical exposures, season of conception, and risk of gastroschisis in Washington State; [Waller et al] and
- Hermaphroditic, demasculinized frogs after exposure to the herbicide atrazine at low ecologically relevant doses. [Hayes et al][56]

The study by Waller et al published in 2010 in the *American Journal of Obstetrics and Gynecology* found that atrazine might be associated with the birth defect gastroschisis affecting abdominal wall.[57] The baby's intestines, and sometimes other organs such as the stomach and liver, extend outside the body through a hole in the abdomen. Waller

---

[52] Australian Government, Australian Pesticides and Veterinary Medicines Authority, 2010, APVMA to have atrazine studies formally assessed. Available at: http://www.apvma.gov.au/news_media/our_view/2010/2010-03-05_atrazine_studies.php last accessed 17 September 2013

[53] Hayes TB, Collins A, Lee M, Mendoza M, Noriega N, Stuart AA & Vonk A, 2002, Hermaphroditic, demasculinized frogs after exposure to the herbicide atrazine at low ecologically relevant doses, *Proceedings of the National Academy of Sciences of the United States of America*, Vol 99(8), pp 5476-5480

[54] Waller SA, Paul K, Peterson SE & Hitti JE, 2010, Agricultural-related chemical exposures, season of conception, and risk of gastroschisis in Washington State, *American Journal of Obstetrics & Gynecology,* Vol 202(3), pp 241.e1-241.e6

[55] Rohr JR & McCoy KA, 2010, A Qualitative Meta-analysis Reveals Consistent Effects of Atrazine on Freshwater Fish and Amphibians, *Environmental Health Perspectives*, Vol 118(1), pp 20-32

[56] Australian Government, Australian Pesticides and Veterinary Medicines Authority, 2011, Chemicals in the News: Atrazine. Available at: http://www.apvma.gov.au/news_media/chemicals/atrazine.php last accessed 17 September 2013

[57] Waller SA, Paul K, Peterson SE & Hitti, JE, 2010, Agricultural-related chemical exposures, season of conception, and risk of gastroschisis in Washington State, *American Journal of Obstetrics & Gynecology*, Vol 202, Issue 3, pp 241.e1-241.e6

et al undertook a case-controlled study of 805 cases and 3616 control subjects. They found that gastroschisis occurred more frequently among those who resided <25 km from a site of high atrazine concentration. They concluded '[m]aternal exposure to surface water atrazine is associated with fetal gastroschisis, particularly in spring conceptions'.[58]

The study by Hayes et al on the feminization of frogs had been published in 2002 in *PNAS*.[59] In this study the researchers found that atrazine, at low ecologically relevant doses, leads to hermaphroditic and demasculinized frogs. The APVMA concluded that there was no evidence to warrant reconsideration of the registration of atrazine and that there was no need to amend the existing human health risk assessment.[60]

No explanation is given as to why the Suzawa and Ingraham study and the Rohr and McCoy study were not included in the review. There was another study not included in the assessment that provides evidence that atrazine acts as an endocrine disrupter.

This study by Fan et al was published in 2007 in *Environmental Health Perspective*s.[61] It was a collaborative study by a group of scientists in Japan and the US who concluded that 'current findings are consistent with atrazine's endocrine-disrupting effects in fish,

---

[58] Waller SA, Paul K, Peterson SE & Hitti, JE, 2010, Agricultural-related chemical exposures, season of conception, and risk of gastroschisis in Washington State, *American Journal of Obstetrics & Gynecology*, Vol 202, Issue 3, pp 241.e1-241.e6, p241.e1

[59] Hayes TB, Collins A, Lee M, Mendoza M, Noriega N, Stuart AA & Vonk A, 2002, Hermaphroditic, demasculinized frogs after exposure to the herbicide atrazine at low ecologically relevant doses, *PNAS,* Vol. 99(8), pp 5476-5480

[60] Australian Government, Australian Pesticides and Veterinary Medicines Authority, 2010, *Atrazine Toxicity: Analysis of Potential Modes of Action*, Australian Pesticides and Veterinary Medicines Authority, Canberra

[61] Fan WQ, Yanase T, Morinaga H, Gondo S, Okabe T, Nomura M, Komatsu T, Morohashi KI, Hayes TB, Takayanagi R & Nawata H, 2007, Atrazine-Induced Aromatase Expression Is SF-1 Dependent: Implications for Endocrine Disruption in Wildlife and Reproductive Cancers in Humans, *Environmental Health Perspectives*, Vol 115(5), pp 720-727

amphibians, and reptiles; the induction of mammary and prostate cancer in laboratory rodents; and correlations between atrazine and similar reproductive cancers in humans'.[62]

These latest findings were not without challenge. In 2008 Solomon et al published a paper claiming that based on a 'weight of evidence' analysis of atrazine, a definitive conclusion against atrazine could not be made.[63] In a further response to this counter-claim, in 2010 Rohr and McCoy analysed the review by Solomon et al on the basis of 'conflict of interest'.[64] They found that the review, which had been industry-funded by Syngenta Crop Protection, Inc., the manufacturers of atrazine, had misrepresented over 50 studies while there were 122 inaccurate and 22 misleading statements. They stated, of the '144 seemingly inaccurate or misleading statements, 96.5% appeared to be beneficial for Syngenta Crop Protection, Inc., in that they supported the safety of the chemical, whereas only 3.5% appeared to be neutral or detrimental to the company'.[65] The APVMA did not include this study by Rohr and McCoy in their review of atrazine.

The Australian government and the APVMA are aware of the dangers from exposure to chemicals that act as endocrine disrupters. In 2004 the CSIRO Land and Water and the Australasian Society of Ecotoxicology's Special Interest Group on Endocrine Disrupting Chemicals held a conference at the CSIRO's Discovery Centre, Black Mountain in Canberra as part of the series 'What's in Our Water'.[66] The outcome of

---

[62] ibid. p 720

[63] Solomon KR, Carr, JA, Du Preez LH, Giesy JP, Kendall RJ, Smith EE, & Van Der Kraak, GJ, 2008, Effects of Atrazine on Fish, Amphibians, and Aquatic Reptiles: A Critical Review, *Critical Reviews in Toxicology,* Vol 38, pp 721-772

[64] Rohr JR & McCoy, KA, 2010, Preserving environmental health and scientific credibility: a practical guide to reducing conflicts of interest, *Conservation Letters* 3, pp 143-150

[65] ibid, p 146

[66] CSIRO, Environmental Side Effects (2004) Australian Government, Land and Water. Available at: http://www.clw.csiro.au/conferences/ourwater/2004/ last accessed 28 July 2013

the Symposium was a paper called "The Black Mountain Declaration on Endocrine Disrupting Chemicals" outlining a precautionary approach to the possibility that endocrine disrupters may contaminate drinking water in Australia.[67] One of the aims of this Symposium series is to bring together key stakeholders to discuss and exchange current information and knowledge.[68] However, neither members of the APVMA's Community Consultative Committee nor the Tasmanian Water Quality Initiative (TWQI) (who were funded to monitor drinking water in Tasmania especially for endocrine disrupters such as atrazine) were informed of the Symposium. Syngenta, on the other hand, appeared as an interested party on the CSIRO's website. Following enquiries from the TWQI regarding the Symposium and why Syngenta had been informed but not them or the CCC, Syngenta's name no longer appeared as an interested party.

Substantial evidence exists that atrazine causes harm in the environment and probably to human health, which should trigger further restrictions on its use. But the industry's argument based on obfuscation is employed to delay actions by decision makers in their regulatory role to protect the public and the environment from hazardous chemicals that act as endocrine disrupters, such as atrazine.

Precautionary measures should be implemented to protect human health, wildlife and the environment when the scientific evidence for harm is strong but uncertain. Unlike the EU's introduction of the REACH program, the Australian APVMA has not implemented the precautionary principle. The REACH program is moving to require

[67] The Black Mountain Declaration on Endocrine Disrupting Chemicals in Australian Waters, 2007. Available at: http://www.clw.csiro.au/conferences/ourwater/EDC-conference-declaration.pdf last accessed 28 July 2013

[68] CSIRO, What's in Our Water Symposium Series. Available at: http://www.csiro.au/science/whats-in-our-water last accessed 28 July 2013

evidence of safety of chemicals from the chemical registrants, whereas in Australia the onus for proving that a chemical is hazardous continues to fall to the victim.

8.5 The need to implement the precautionary principle

Recent developments and a better understanding of the mode of action of endocrine disrupting chemicals in the environment have prompted the need for a review of regulations.[69] To assess the harm caused by chemicals in the environment it is no longer sufficient to measure the level of toxicity caused by a single chemical. There is new research, which shows chemicals have synergistic effects when mix with other chemicals in the environment. Chemicals that act as endocrine disrupters, either singularly or as mixtures, in the environment can interfere with hormone action at specific times in the growth of the organism. Importantly there are also currently no safe exposure levels for vulnerable members of society such as pregnant women and children. Meanwhile, new studies indicate that some chemicals have the capacity to subtly alter gene signals (epigenetics) resulting in a variety of diseases and disorders, including cancer.[70]

The Endocrine Society recently released new protocols for identifying endocrine disrupting chemicals, which they hope will strengthen the ability of current screening programs to identify endocrine disrupting chemicals (EDCs).[71] In a scientific position statement published in 2009 the Society provided a comprehensive summary of the

---

[69] Vandenberg LN, Colborn T, Hayes TB, Heindel JJ, Jacobs DR Jr, Lee DH, Shioda T, Soto AM, vom Saal FS, Welshons WV, Zoeller RT & Myers JP, 2012, Hormones and endocrine-disrupting chemicals: low-dose effects and nonmonotonic dose responses, *Endocrine Reviews*, Vol 33(3), pp 378-455

[70] Hileman B, 2009, Chemicals can turn genes on and off; new tests needed, scientists say, *Environmental Health News*. Available at: http://www.environmentalhealthnews.org/ehs/news/epigenetics-workshop last accessed 8 October 2010

[71] Endocrine Society, 2012, Experts Say Protocols for Identifying Endocrine-Disrupting Chemicals Inadequate. Available at: http://www.endo-society.org/media/press/2012/Experts-Say-Protocols-for-Identifying-Endocrine-Disrupting-Chemicals-Inadequate.cfm last accessed 28 June 2012

scientific background that justifies concern for the effects of EDC exposure to humans and wildlife.[72]

Francisco Sanchez-Bayo says current ecotoxicology approaches are based on the dose-response relationship and consider toxic effects at fixed exposure times and therefore cannot make predictions for a wide range of exposures in the environment, making them of little relevance in risk assessment.[73] Tjalling Jager, however, states the problem is the use of outmoded and inadequate risk assessment methods not only by regulators but also by scientists.[74]

The APVMA currently adopts a risk-based assessment of chemicals based on known scientific knowledge. Given the growing new evidence and awareness of the complexities of chemicals that act as endocrine disrupters or have epigenetic effects on living organisms in the environment, the APVMA should now adopt the precautionary principle. The APVMA currently acknowledges that it 'exercises caution where scientific opinion is divided or scientific information is incomplete'.[75] But as I have shown above there is no provision for assessing all scientific studies. This potential for wilful ignorance can only add to the uncertainty surrounding endocrine disrupters such as atrazine. The precautionary principle is a legal instrument that requires timely action or interventions in the face of scientific uncertainty. Its emphasis on the preferred error

---

[72] Endocrine Society, 2009, Endocrine-Disrupting Chemicals, June 2009. Available at: http://www.endo-society.org/advocacy/policy/upload/Endocrine-Disrupting-Chemicals-Position-Statement.pdf last accessed 28 June 2012

[73] Sanchez-Bayo F, 2009, From simple toxicological models to prediction of toxic effects in time, *Ecotoxicology*, Vol 18, pp 343-354

[74] Jager T, 2012, Bad habits die hard: the NOEC's persistence reflects poorly on ecotoxicology, *Environmental Toxicology and Chemistry*, Vol 31(2), pp 228-229

[75] Australian Government, Australian Pesticides and Veterinary Medicines Authority, 2010, APVMA response to the discussion paper "*A National Scheme for Assessment, Registration and Control of Use of Agricultural and Veterinary Chemicals",* Australian Pesticides and Veterinary Medicines Authority, Canberra, Australia, p 9

of false positive should also generate the need to incorporate and seek more scientific evidence to validate the action by decision makers. The APVMA does not intend to express a view as to whether it should or should not adopt the precautionary principle but admits it currently exercises caution. In exercising caution the APVMA refers to the statutory test in the general principles laid down in the judicial review of the matter *Friends of Hinchinbrook Society Inc v Minister for the Environment (1997)*. The principle in relation to caution states:

> …to proceed with caution when reviewing an administrative decision on the ground that it does not give proper weight to relevant factors, lest it exceed its supervisory role by reviewing the decision on its merits.[76]

This test, according to the APVMA, would apply by exercising caution where scientific opinion is divided or scientific information is incomplete.[77] The outcome in the Hinchinbrook case was in favour of the commercial developer.[78]

8.6 Conclusion

Many new chemicals have been manufactured and used for industrial purposes, including agriculture, since the beginning of the 20th century. Few of these chemicals have been adequately tested for long-term harmful effects on the environment or human populations. The result is an unquantifiable experiment, the unintended consequences of which are only now beginning to surface. Regulatory agencies established by governments, both overseas and in Australia, to protect the environment and human health from the excesses of industrial chemicals continue to delay

---

[76] Friends of Hinchinbrook Society Inc v Minister for Environment & Ors [1997] FCA 55 (14 February 1997). Available at: http://www.austlii.edu.au/au/cases/cth/federal_ct/1997/55.html last accessed 3 November 2013

[77] Australian Government, Australian Pesticides and Veterinary Medicines Authority, 2010, APVMA response to the discussion paper "*A National Scheme for Assessment, Registration and Control of Use of Agricultural and Veterinary Chemicals",* Australian Pesticides and Veterinary Medicines Authority, Canberra, Australia,

[78] Mercer D, 2000, *A Question of Balance, Natural Resources Conflict issues in Australia*, (3rd Ed), The Federation Press, Sydney, p 44

implementing action to mitigate potential harmful effects. These agencies have close relationships with the chemical industry and rely on the chemical manufacturer to provide evidence of safety of the chemical and monitor its use in the environment.

The US EPA and the APVMA are faced with conflicting scientific research as to the harm caused by atrazine; the toxicology studies are using outmoded testing methods; and undue influence and pressure is being exerted by industry on the regulators. The result is regulatory bodies have been slow to adopt more refined toxicology testing for chemicals that pose hazards and risks at the sub-toxic level even though strategies for implementing these new tests have been discussed since 2007.[79] There appear to be no practical reasons why the agencies have been slow to shift to the new paradigm in toxicology testing. The precautionary approach is not in the interests of the industries being regulated and therefore regulatory capture ensures it is unlikely to be adopted by regulatory agencies.

The further problem of lack of regulation for chemicals where the sub-toxic end-point is just as hazardous as toxicity also needs to be addressed. These chemicals include endocrine disrupters that cause developmental and reproductive disorders and cancer at extremely low levels e.g. parts per billion. This further raises the problem of complexity in the environment where timing, mixtures and accumulation of chemicals are sources of non-knowledge or ignorance.

The regulators could do more to effectively monitor the use of chemicals in the environment. They could engage more actively with the current scientific knowledge

[79] Committee on Toxicity Testing and Assessment of Environmental Agents, National Research Council, 2007, *Toxicity Testing in the 21st Century: A vision and Strategy*. Available at: http://www.nap.edu/catalog/11970.html last accessed 23 April 2014

about the harms caused by chemicals such as endocrine disrupters and abide by the legislation that governs their operations. The Tasmanian devil is only one species threatened with extinction from the lack of protection of its environment. The precautionary principle as a legal tool to enable decision makers to act in the face of scientifically plausible but uncertain evidence should be implemented to further restrict or ban the use of atrazine. The evidence of harm in the Tasmanian devil population is strong but the link between atrazine, as initiating or progressing the devil cancer, is weak. This has been confounded by the lack of toxicology studies. But overseas evidence and the fact that atrazine is found in the environment of the three other wildlife cancer studies, discussed in the previous chapter, suggests that it may play a role in cancer. The precautionary principle's role in acting on the probability of a false positive also means it seeks more scientific research. In the meantime a ban on atrazine has a high probability of mitigating the harm, given the evidence, and if this situation is overturned and the chemical is proven safe, the consequences will be minimal compared to the extinction of the Tasmanian devil.

Although economically Tasmania, like most of Australia, relies for its income on the service industry it is still nostalgically linked to the exploitation of its natural resources, such as forests and minerals. Plantation forestry and its expansion is viewed by environmental non-profit organizations such as the Tasmanian Wilderness Society as the solution to the logging of native and old growth forests in Tasmania. As such plantation forests are viewed as part of the agricultural and not the forestry industry. In the next chapter I reveal the conflict of interest that exists in Tasmania that impedes real progress in the scientific research into the Tasmanian devil cancer.

Chapter 9 – Plantation forestry in Tasmania - in whose interest?

9.1 Introduction

The north-east of Tasmania was the site of early development of agriculture and livestock grazing industries but more recently these land uses have been converted to plantation forestry. All these intensive commercial industries have had major impacts on the devils' habitat, not only in terms of its destruction but also its degradation through the use of fertilizers, pesticides and poisons. The north-east was however not the only region suitable for plantation forests; they have gradually spread across most of the state with the exception of the far southwest.

Volume 2 of the European Environment Agencies publication *Late lessons from early warnings* identifies conflicts of interest as a reason for the lack of progress in mitigating the harm caused by dangerous human activities. In this chapter I analyse the close relationship between Tasmanian government and the forestry industry in Tasmania. I establish that a conflict of interest exists within the Tasmanian Department of Primary Industries, Parks, Water and Environment (DPIPWE), when it oversees both the use and monitoring of all chemicals used in plantations and the Save The Devil Program (STDP) responsible for research into the devil cancer.

Whilst it is acknowledged that many pesticides used in forestry plantations are potentially hazardous I have focused on only two of those used in plantation forestry, atrazine and the poisonous compound sodium fluoroacetate (1080). These two chemicals were also selected by the DPIPWE in their limited list of chemicals for

toxicology studies as discussed in Chapter 5. Atrazine has been found to contaminate surface water in Tasmania and more recently ground water. As discussed in Chapter 7 atrazine is also the most commonly detected chemical in the habitats of three other wildlife species with cancers. The continued use of the atrazine and 1080 in Tasmania has also been controversial and has raised the most concern within the community.

A further hazard recently debated in relation to plantation forestry is the possibility that genetically modified (GM) eucalypts have been introduced. This will also be analysed. Although the existence of GM eucalypts is strongly denied by both the government and the forestry industry, it is not without precedent that GM crops have been introduced without the knowledge or consent of the relevant governments, e.g. GM rice into India and GM corn into Mexico.[1] The possibility that the eucalypts have been genetically modified further adds to the uncertainty as to the possible cause of the Tasmanian devil cancer.

9.2 The forestry industry in Australia

The *National Forestry Policy Statement* (NFPS) developed in 1992 is the overarching framework for forestry policy in Australia.[2] It supports the 1997 *Plantations 2020 Vision,* a policy to increase plantation development and improve regional wealth. Under the *NFPS* the Commonwealth and state governments introduced the *Regional Forests Agreements* (*RFAs*) to establish agreed approaches to sustainable management of native forests. *RFAs* included the Comprehensive, Adequate and Representative

[1] Ho, MW, 1999, *Genetic Engineering, Dream or Nightmare?* Gateway, Dublin,
[2] Thomson J & Kelly M, 2007, *Report Australia's forest industry in the year 2002,* Department of Agriculture, Fisheries and Forestry, Canberra

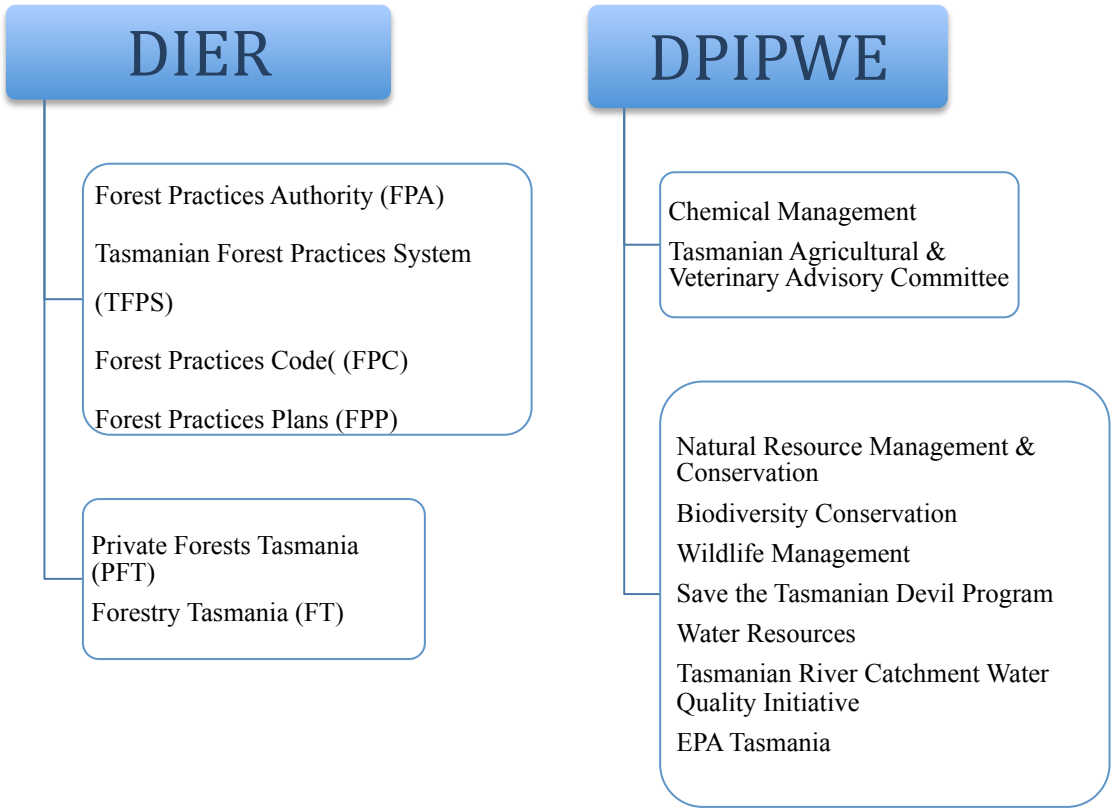
(CAR) reserve system to protect Australia's forest environment as previously noted in Chapter 6. The NFPS's primary role is to promote an enabling policy environment for forestry, with a focus on removing impediments to investment, particularly in plantations, and an expansion in the private sector through privatization of publicly owned resources.[3]

9.3 The forestry industry in Tasmania

In Tasmania at the state level, two departments administer forestry management, the conservation of natural resources and the protection of threatened species. The Department of Infrastructure, Energy and Resources (DIER) oversees the Tasmanian RFA, Forestry Tasmania, Private Forests Tasmania as well as the Forestry Practices Authority (FPA). The DPIPWE, on the other hand, is responsible for water quality monitoring, overseeing the Chemical Management Branch (which controls the use of agricultural chemicals) and solving the problem of the Tasmanian Devil Facial Disease through the STDP. The Environmental Protection Authority (EPA) Tasmania also operates under its auspices. The structure of the Tasmanian government regulatory authorities is shown in Figure 9:1 below.

[3] ibid.

Figure 9:1 Structures of Tasmanian government regulatory authority



9.3.1 Conflict of interest in Tasmania

The Tasmanian forestry industry operates under what appears to be a guise of regulatory control, which upon closer analysis exposes a conflict of interest both within government bodies and between government and industry. A "revolving door" exists between the state government and the forestry industry when the same personnel alternatively serve each sector. The Minister for DIER appoints the FPA board members who are supported by an advisory council and a team of scientists, advisors, compliance officers and administrative staff. The current chairman of the FPA Board is Professor Gordon Duff who is also the CEO of the Forestry CRC at the University of Tasmania (UTAS). One of the partners in the CRC for Forestry is Forestry Tasmania, a

government enterprise charged with managing Tasmania's state forests.[4] Board members include John Whittington who is also the Deputy Secretary of DPIPWE, formerly of Forestry Tasmania and currently on the Save the Tasmanian Devil Program Steering Committee. Other board members represent the Forest Industries Association of Tasmania, Forestry Tasmania, Tasmanian Farmers and Graziers Association; one sitting member is a Chief Forest Practices Officer. There is no independent environmental expert on the board. A list of members of the Forest Practices Authority and those sitting on the Forest Practices Tribunal is shown in 9:1 below which demonstrates that there exists no arms length separation between the government regulator and the forestry industry. The same departments responsible for the promotion of forestry interests also oversee the use of chemicals and the protection of wildlife thus constituting a conflict of interest.

Table 9:1 Governance of Forestry Practices Authority and Forest Practices Tribunal[5]

Directors of the FPA	**Affiliations**	**Further connections**
Professor Gordon Duff (Chairperson)	CEO Cooperative Research Centre for Forestry	
Dr John Whittington (Director)	Deputy Secretary, DPIPWE	
Ian Whyte (Director)	CEO Forest Industries Association of Tasmania	Senior Advisor to Tasmanian Farmers and Graziers Association
Stephen Luttrell (Director)	Retired forester	Forestry Commission and Forestry Tasmania
Meredith Roodenrys	Policy officer, Tasmanian Farmers & Graziers Association	
Members of Forest Practices Authority Council		
Jamie Bayly-Stark	State government	Former Director, Department of Premier & Cabinet
Alan Garcia	Local Government	

[4] Duff G, Commentaries & Speeches, Get Farming Australia. Available at: http://www.getfarming.com.au/pages/farming/speeches_view.php?sId=5398720110530174200 last accessed 6 July 2012

[5] Forest Practices Authority, nd, *The role of Board of the FPA*. Available at: http://www.fpa.tas.gov.au/the_fpa/programs/governance last accessed 18 September 2013

	Association of Tasmania	
Alex Schaap	Former Deputy Secretary DPIPWE	Director of EPA
Tom Fisk	Private Forests Tasmania, Chief Executive Officer	
John Hickey	Forestry Tasmania	
Terry Edwards	Forestry Industry Association of Tasmania (FIAT)	
Peter Bosworth		
Brett Hooper	Tasmanian Farmers & Graziers	Board member Private Forests Tasmania
Members of Forest Practices Tribunal	**Occupational areas**	
Keyran Pitt QC, Phillip Wright	Legal	
Marcus Higgs, Bert White, Donald Francombe	Forestry	
Robert Ellis, John Pretty, Rod Pearse	Forestry	
John Shoobridge, Neville Calvert, Robert Henry	Forestry	

Private Forests Tasmania (PFT) is also a government body, funded by both the Tasmanian government and private forestry; its role is the promotion of private forestry through providing strategic policy advice to government. It also provides staff to the Forest Practices Board (FPB) to undertake audits of Forest Practices Plans. The former chairman of PFT, Ian Dickerson, was also a member of the FPB and the Tasmanian Natural Resource Management Advisory Committee. Natural Resource Management in Tasmania was established in 2002 under the Federal Government *EPBC Act 1999* for assessment of water quality in estuaries and rivers and to implement protection for endangered species.[6] The chairman position on PFT is currently vacant.

Meanwhile, the primary endeavour of the Environment Protection Authority (EPA) under the *Environmental Management and Pollution Control Act 1994* (EMPCA) is the

[6] Natural Resource Management in Tasmania, *Monitoring and Evaluation.* Available at http://www.nrmtas.org/about/monitoringAndEvaluation.shtml last accessed 27 May 2007

prevention, reduction and remediation of environmental harm.[7] The EPA not only operates within the DPIPWE but its board members are also closely affiliated with government. It includes the chair, John Ramsay, a lawyer and previously Secretary to Department of Health and Human Services and a former DPIPWE Secretary. Alex Schaap is the Director and also a former DPIPWE deputy secretary, whilst also being the General Manager of the EPA Division of DPIPWE. Board member Dr Helen Locher is also a Principal Consultant at Hydro Tasmania and Louise Cherrie was formerly with the Department of Economic Development.

There has been a history in Tasmania of regulators not operating at arms length especially in regard to plantation forestry and its practices. This situation was exacerbated with the proposal by Gunns Limited to build a pulp mill in Bell Bay. Regulatory capture was again similarly characterized by "a revolving door" between the forestry industry and the government regulatory authorities. Evan Rolley whilst Secretary to the Premier's Department under the then Premier Paul Lennon was also a consultant to Forestry Tasmania. He was also a former Chairman of the Tasmanian Branch of the Institute of Foresters, an industry advocacy group.[8] In 2012 Rolley became the Executive Director of the private timber company Ta Ann.[9] In 2013 the secretary to the Department of Premier and Cabinet was Rhys Edwards, a former senior advisor to former Premier, Paul Lennon. The managing director of Forestry Tasmania Bob Gordon was the former head of the Pulp Mill Task Force, a government initiative

[7] Tasmanian Government, Environment Protection Agency, *Environmental Management and Pollution Control Act 1994*. Available at: http://epa.tas.gov.au/policy/empca last accessed 7 August 2013
[8] The Institute of Foresters of Australia, *Strategic Plan 2006-2008.* Available at: http://www.forestry.org.au/pdf/pdf-public/2006-008%20-%20Strategic%20Plan%20Only%20-%20Public%20website%20(as%20at%20171105).pdf last accessed 13 September 2007
[9] *Australian Broadcasting Corporation News*, 3 April 2012, Ex-forestry chief joins Ta Ann. Available at: http://www.abc.net.au/news/2012-03-14/20120314-rolley-new-ta-ann-boss/3889478 last accessed 6 July 2012

and according to the Wilderness Society he was the 'de-facto representative of Gunns' on the Task Force.[10]

9.4 Eucalypt plantations

In Tasmania the forest industry is a major contributor to the state economy mainly from the production of woodchips sourced from the controversial logging of native and old growth forests and more recently plantations.[11] The implementation in 1997 of the *Plantations 2020 Vision*[12] facilitated a tax-minimising plantation managed investment scheme (MIS), which led to an expansion in the scale of plantation forests.[13] Further motivations included off-shore manufacturing investment to secure pulpwood on short rotations and the future trading in carbon credits.[14] From 2004 the Tasmanian plantation estate rapidly expanded at an average rate of 13,500 ha/year most of which was hardwood.[15] In the period between 2006 and 2011, financed mainly through the MIS, plantation areas increased by 47 per cent or about 74,000 to 233,200 ha.[16] In 2012 the total area of forest converted to plantations covered 314,000 ha. The majority of hardwood plantations are grown on privately owned land.[17] The total extent of forest area in Tasmania is 3,388,000 ha with 1,172,000 ha consisting of conservation reserves.

---

[10] The Wilderness Society, *Bob Gordon's appointment to head up Forestry Tasmania*, Media Release. Available at: http://www.wilderness.org.au/campaigns/forests/tasmania/gunns_proposed_pulp_mill/bob_gordon last accessed 13 September 2007

[11] Australian Government, Bureau of Infrastructure, Transport and Regional Economics (BITR), 2008, *A regional economy: a case study of Tasmanian,* Report 116, Canberra, ACT

[12] *Plantations 2020 Vision*, A strategic partnership between Commonwealth, State and Territory governments and the plantation timber growing and processing industry Available at: http://www.plantations2020.com.au/vision/index.html last accessed 14 August 2009

[13] Ajani J, 2012, The untold story of the role of government in the rise and fall of Gunns, *The Conversation.* Available at: http://theconversation.edu.au/the-untold-story-of-the-role-of-government-in-the-rise-and-fall-of-gunns-9972 last accessed 31 January 2013

[14] Green G, 2004, *Plantation Forestry in Tasmania, The current resource, current processing and future opportunities*, Timber Workers for Forests, Hobart, Tasmania, p 17

[15] ibid.

[16] Forest Practices Authority, 2012, State of the forests Tasmania 2012. Available at: http://www.fpa.tas.gov.au/__data/assets/pdf_file/0009/82872/State_of_the_forests_Tasmania_2012_report.pdf last accessed 5 December 2013

[17] Green G, 2004, *Plantation Forestry in Tasmania, The current resource, current processing and future opportunities*, Timber Workers for Forests, Hobart, Tasmania

The remaining 2,215,000 ha are either covered by state forest or public land tenure (1,154,000 ha) or private tenure (1,061,000 ha).[18]

The Gunns Limited *State of the Forests Report* for 2006 records the total estate in which it had some form of interest was 273,931 hectares, making it the biggest private plantation owner.[19] Gunns' eucalypt plantations are managed solely for the production of pulpwood. Gunns' forest plantation development was funded through a combination of its own funds and joint ventures with customers such as Tamar Tree Farms (a partnership with Mitsubishi Paper Mills and Tokyo Electric Power Company).[20] According to forecasts from the 2012 Plantation Platform of Tasmania (PPT) – a partnership with Forestry Tasmania, Daio Paper, Kawasho International, Nakabayashi, Nissen, Nikkei BP, Kobunsha and NBS Ricoh - approximately 500,000 tonnes of wood would be available annually.[21] The plantation timber would be used for woodchip production and processed in Japan by Daio Paper.

The logging of old-growth forests and the proposed building of the pulp mill in the north of the state have been contentious issues in Tasmania. Plantation forestry, on the other hand, has been seen as a solution to old-growth forest logging because of its ability to supply woodchips to the pulp mill. As such it has gone uncontested except for the use of chemicals and their role in surface, drinking and ground water contamination.

---

[18] Forest Practices Authority, 2012, State of the forests Tasmania 2012. Available at: http://www.fpa.tas.gov.au/__data/assets/pdf_file/0009/82872/State_of_the_forests_Tasmania_2012_report.pdf last accessed 5 December 2013, p 9

[19] Expert witness statement of Mr Andrew Robert de Fegely, Expert of Gunns Limited. Available at: http://www.gunnspulpmill.com.au/iis/supp/robert_de_fegely_ews.pdf last accessed 5 June 2012

[20] Gunns Limited Forest Division, Plantations. Available at: http://www2.gunns.com.au/Forest/plantations.html last accessed 5 June 2012

[21] Green G, 2004, *Plantation Forestry in Tasmania, The current resource, current processing and future opportunities*, Timber Workers for Forests, Hobart, Tasmania, p 14

9.5 Pesticide use in plantation forestry in Tasmania

The use of chemicals and poisons in the Tasmanian environment to control pests has a long history. The period following the Second World War, as in the rest of the developed world, saw unprecedented growth in their use.[22] In Tasmania, this situation was exacerbated with the introduction and spread of plantation forests with their dependence on chemical pesticides. Whilst chemicals are used extensively in agricultural production in Tasmanian, it is the location of eucalypt plantations in otherwise pristine water catchments and the increased quantities and aerial spraying of chemicals that has made them a focus of attention.

In Australia, each individual state government oversees the use of chemicals whilst the Australian Pesticides and Veterinary Medicines Authority (APVMA), a Federal government body, is responsible for the registration of chemicals. The APVMA provided an 18-page list of registered products used as active ingredients in Tasmania.[23] However, this extensive list omitted terbuthylazine (a triazine), fluazifop and 1080 also used in plantation forestry in Tasmania. The definition of pesticide according to the publication *Pesticides in Plantations* is as follows:

> Any chemical or chemical mixture used for controlling weeds, insects, fungi, nematodes and animals, which adversely affect growth (quantity and quality) and the health of plantations.[24]

Eucalypt plantations in Tasmania are monocultures of mainly *Eucalyptus globulus* and *nitens* and as such rely heavily on pesticides to kill competing, mostly native, flora and

---

[22] Harrington J, 1996, The Midwest Agricultural Chemical Association: A Regional Study of an Industry on the Defensive, *Agricultural History*, Vol 70(2) pp 415-438

[23] Australian Government Senate Rural and Regional Affairs and Transport Legislation Committee, Answers to Questions on Notice, Budget Estimates May 2009, Response to Question on Notice, Question: APVMA06 Attachment 1, *Hansard*, 26 May 2009, p 96

[24] Jenkins BM & Tomkins B, 2006, *Pesticides in Plantations*, Forest and Wood Products Research and Development Corporation, Melbourne, Vic.

fauna species. Some of these pesticides, although designed to kill target species, are also known to cause harm including cancer to non-target species.

In Tasmania chemical use in plantation forestry is self-regulated by the forestry industry and is monitored under the *Forest Practices Code 2000 (FPC 2000)*. The *FPC 2000* provides directions for the use of chemicals in plantations. In relation to the initial plantation development phase it contains the following provision – '[w]eed control carried out during site preparation will be planned to minimize the risk of soil erosion and the movement of chemicals off-site'.[25] In its basic approach to the use of chemicals, it states '[w]ithin 2 km upstream of a town water supply intake … specific prescriptions will be placed in Forest Practices Plan (and will be considered for catchments which are important for threatened aquatic fauna)…'.[26] It further states '[a]pplication of approved herbicides and other chemicals is only permitted in accordance with Section E2.'[27] The general principles under Section E2 assign the responsibility to protect people, water resources, karst systems and stock during the application of chemicals to the forest owners. It states that the use of chemicals will not impinge on the achievement of the water quality objectives. In relation to controlling pests and weeds in watercourses or along stream banks it states that wherever practical non-chemical means of control should be used but if chemicals are used, *Roundup Biactive* is preferred. [28]

[25] Forest Practices Board, 2000, *Forest Practices Code*, Forest Practices Board, Hobart, Tasmania, p 80
[26] ibid, p 57
[27] ibid, p 83
[28] ibid, p 89

Roundup Biactive is recommended because it is a special formulation with a built-in 'aquatically approved' surfactant but it contains the active constituent glyphosate.[29] Uncertainty exists as to the level of harm caused by glyphosate and its formulations. In a recent review of the data produced by independent scientists, as opposed to industry-sponsored studies, glyphosate exhibited teratogenicity[30] and reproductive toxicity to embryos of *Xenopus laevis* (African clawed frog) and chickens.[31]

The potential for pesticides from plantation forests to cause widespread contamination of the environment was first revealed in studies undertaken in Tasmania in 1994.[32] The results of these studies clearly demonstrated that following heavy rainfall chemicals used in plantation forestry had the potential to cause hazardous runoff. Hence an increase in plantation forests has correlated with an increase in the number of reported incidents of chemical contamination across Tasmania, particularly in the north east of the state. The issue of water contamination is further discussed in section 9.7 below. The next section covers aerial spraying of pesticides, which is a further cause for concern because of the potential for widespread dispersal of chemicals to non-target sites.

---

[29] Roundup Biactive Herbicide, Pest Genie. Available at: http://www.pestgenie.com.au/webservices/SearchProxy.asp?function=GetProduct&ProductID=975729&Details=Y&CompanyID=509228 last accessed 10 November 2013

[30] Teratogenicity – the capability of producing fetal malformation. Available at: http://medical-dictionary.thefreedictionary.com/teratogenicity last accessed 30 July 2013

[31] Antoniou M, Habib MEM, Howard CV, Jennings RC, Leifert C, Nodari RO, Robinson CJ & Fagan J, 2012, Teratogenic Effects of Glyphosate-Based Herbicides: Divergence of Regulatory Decisions from Scientific Evidence, *Journal of Environmental & Analytical Toxicology*, S4:006, pp 1-13

[32] Davies PE, Cook LSJ & Barton, 1994, Triazine herbicide contamination of Tasmanian streams: Sources, concentrations and effects on biota. *Australian Marine and Fresh Water Research,* Vol 45(2), p 209-226

9.6 Aerial Spraying of pesticides

It is acknowledged by regulators worldwide that there is a real potential for off-target movement of chemicals from aerial spraying that may affect public health and impact the environment.[33] The APVMA also acknowledges that 'measureable off-target spray drift can occur' in its 'Operating Principles in Relation to Spray Drift Risk'.[34] Assessment of chemicals that drift off-target is, according to the APVMA, a two-step process.[35] The first is to understand the nature of the hazard and the second is to understand the type of hazard and how much exposure is likely to occur. Chemicals are assessed for toxicity, persistence and accumulation properties. While exposure is also assessed according to the threshold for the chemical, with exposures above the threshold considered not acceptable. The APVMA, because it claims to incorporate large safety margins into its risk assessment, considers the risk for exposure below the threshold set for each chemical to be negligible. But when the hazard of a chemical is measured in terms of high or acute toxicity, it potentially ignores the hazard of a chemical that may be harmful at the non-toxic level (below the threshold), such as endocrine disrupters.

The Operating Principles prescribe a set of criteria to assess the risk from spray drift to humans and the environment. For human health when assessing a pesticide with high mammalian toxicity it states – 'such a risk can be evaluated by estimating the quantity of that pesticide falling at that distance per unit area, the amount of pesticides likely to be absorbed through the skin, transferred to the mouth and inhaled by a person over a

[33] Primary Industries Standing Committee Report, 2002, *Spray drift management: principles, strategies and supporting information*, CSIRO Publishing, Collingwood, Victoria, Australia
[34] Australian Government, Australian Pesticides and Veterinary Medicines Authority, 2008, *Operating Principles in Relation to Spray Drift Risk*, APVMA. Available at: http://www.apvma.gov.au/use_safely/docs/spraydrift_op_principles.pdf last accessed 28 February 2013
[35] ibid.

given period of time'.[36] This calculated potential dose of pesticide is then compared to the relevant health safety standard set by the Office of Chemical Safety. The APVMA states that '[f]ortunately, very few pesticides are sufficiently toxic to cause human health risks from these kinds of bystander exposures'.[37]

The hazards of drift from aerial spraying are well documented. In Tasmania experiments carried out by Davies et al found that spray drift was recorded at 400 meters from the target.[38] Hence, according to a review of aerial spraying, rivers, streams and lakes should be prescribed exclusion zones.[39] However, not all pesticide drift happens during or immediately after a pesticide application. Some pesticides continue to evaporate from fields for several days to several weeks after an application is completed.[40] Atrazine is of particular concern because it is persistent and is possibly subject to atmospheric transportation.[41]

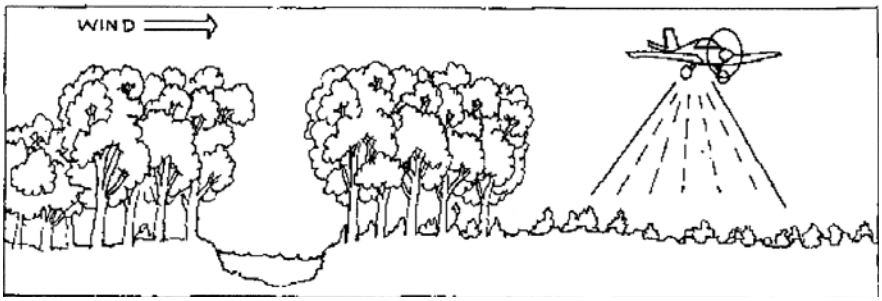
In Tasmania the *Code of Practice for Aerial Spraying* issued in June 2000 is administered by DPIPWE and published by Agricultural, Silvicultural and Veterinary Chemicals (ASCHEM) Council.[42] It prescribes the minimum standards for applying agricultural chemical products by aerial spraying in Tasmania. Current directions for

---

[36] ibid, p 7
[37] ibid.
[38] Davies, PE, Cook, LSJ & Barton, JL, 1994, 'Triazine Herbicide Contamination of Tasmanian Streams: Sources, Concentrations and Effects on Biota' *Australian Journal of Marine and Freshwater Resources,* No. 45, pp 209-226
[39] Agricultural, Silvicultural and Veterinary Chemicals Council. 2005, 'Review of the Code of Practice for Aerial Spraying' Summary of Submission, accessed 14/5/2007, http://www.dpiw.tas.gov.au/inter.nsf/Attachments/CART-6GT7SX/$FILE/Summary%20of%20Submissions%20to%20Aerial%20Code%20Review.pdf
[40] Pesticide Action Network North America, (PANNA), 2006 *Secondhand Pesticides: Airborne Pesticide Drift*. Available at: www.panna.org last accessed 12 January 2007
[41] Cites Barrett et al, 1991 in J Jackson, 1997*, State of Habitat Availability and Quality in Inland Waters,* Department of the Environment, Canberra
[42] Tasmanian Government, Department of Primary Industries, Parks, Water and Environment, 2000, *Code of Practice for Aerial Spraying.* Available at: http://www.dpiw.tas.gov.au/inter.nsf/Attachments/EGIL-555VL6/$FILE/Code%20of%20Practice%20for%20Aerial%20Spraying.pdf last accessed 7 August 2013

aerial spraying are shown in Figure 9.2 below.

Figure 9.2 Directions for aerial spraying[43]



In April 2005 in response to the continued contamination of surface and groundwater, the Tasmanian government DPIPWE called for submissions for a review of the *Code of Practice for Aerial Spraying.* In October 2005 a Summary of Submissions was released stating that the general view of the submissions was that the *Code of Practice* fails on many levels.[44] Proposed changes to the chemical spray regulations, to address some of the problems, were to go before the Tasmanian Legislative Council for further consultation but it was reported that the changes were withdrawn.[45] The practice of aerial spraying of these and other toxic chemicals continues and as Dr Alison Bleaney asks 'When is the review of aerial spraying of pesticides going to take place? It has been in progress now for seven years and two proposals have been produced – each withdrawn because of industry pressure!'[46]

---

[43] Source: Forests Practices Board, 2000, *Forest Practices Code,* Forest Practices Board, Hobart Tasmania

[44] Tasmania Agriculture, Silvicultural and Veterinary Chemicals Council, 2005, Review of the Code of Practice for Aerial Spraying, Summary of Submissions. Available at: http://www.dpiw.tas.gov.au/inter.nsf/attachments/cart-6gt7sx/$file/summary%20of%20submissions%20to%20aerial%20code%20review.pdf last accessed 12 November 2013

[45] Briscoe T, 2012, No change to chemical spray regulations at this stage, *Tasmanian Country Hour*, ABC Rural, Australian Broadcasting Corporation. Available at: http://www.abc.net.au/rural/tas/content/2012/06/s3521296.htm?site=northtas last accessed 11 June 2012

[46] Bleaney A, 2012, Chemicals: The Dismal Failure. Available at: http://tasmaniantimes.com/index.php?/weblog/article/how-chemicals-affect-us/ last accessed 11 June 2012

9.7 Pesticide contamination of water in Tasmania

In 2006, in response to continued pesticide contamination of surface, drinking and ground water the River Catchment Water Quality Initiative (RCWQI) was established.[47] The Initiative was jointly sponsored by the Federal and Tasmanian governments and was set up within DPIPWE. Its role was to monitor pesticide contamination in water catchments but with no provisions for controlling the use of pesticides. The UTAS was to assist the Initiative by undertaking water analysis whilst Forestry Tasmania was to assist in analyzing the historical data in relation to pesticide use.[48] DPIPWE is once again responsible for monitoring pesticide use through the Initiative whilst it is also the regulatory body responsible for overseeing pesticide use. There is no independent system operating, which means the principle of arms length separation is again being ignored.

The contamination of surface and drinking water was documented in 1994 when Davies and colleagues published a study that found between 1989 and 1992, 20 of the sampled 29 streams draining plantation forests contained detectable residues of atrazine and simazine.[49] Between 1992 and 1997 other incidents of contamination included:

- creeks in the Huon Estuary contaminated with atrazine;[50]
- domestic water contamination in Lorinna and Derby, with people poisoned;[51]
- Franklin River Creek contaminated with simazine.[52]

---

[47] Tasmanian Government Department of Primary Industry and Water, *Tasmanian River Catchment Water Quality Initiative*. Available at: http://www.dpiw.tas.gov.au/inter.nsf/WebPages/CART-6R7368?open last accessed 25 May 2007

[48] ibid.

[49] Davies, PE, Cook, LSJ & Barton, JL, 1994, 'Triazine Herbicide Contamination of Tasmanian Streams: Sources, Concentrations and Effects on Biota' *Australian Journal of Marine and Freshwater Resources,* No. 45, pp 209-226

[50] Commonwealth Scientific and Industrial Research Organisation, Marine Research, 2000, *Huon Estuary Study*. Available at: http://www.marine.csiro.au/research/sme/huonest/report/index.html last accessed 13 May 2007

[51] Sunday, 2004, television program, *Ninemsn*, 26 September. Available at: http://sunday.ninemsn.com.au/sunday/cover_stories/article_1649.asp last accessed 14 May 2007

Despite continued contamination incidents in Tasmania in 2003 a Federal government Senate Inquiry into plantations received a submission in which Gunns Limited claimed not to have found any trace of atrazine or other herbicides in any sampling they had undertaken. [53] Under a regime of self-regulation these water test results were not disclosed by Gunns, and as such there was no way to verify the claims.[54] The Senate Inquiry however recommended that governments call a halt to new plantation forests and make substantial changes to the management of Tasmanian forests.[55] The report also called for joint venture research to study environmental impacts of plantations particularly on water quality and quantity. [56]

In Tasmania, despite the recommendations of the Senate Inquiry, contamination of surface and drinking water continued. In 2004 geohydrologist David Leaman claimed that controls on aerial spraying were not being enforced and there were no assurances that the dosage or the use was under control.[57] Leaman's comments coincided with a major chemical spill in the north-east of the state at St Helens. It became the focus for the report by Drs Alison Bleaney (Area Medical Officer) and Marcus Scammell (marine ecologist), which made a correlation in time and space between the increase in plantations, oyster abnormalities and mass deaths, and the Tasmanian devil cancer. The

---

[52] Tillack G, nd, *Submission: Draft Impact Submission Statement – Gunns Ltd. Proposed Kraft Eucalypt Pulp Mill No. 571*. Available at: http://www.justice.tas.gov.au/__data/assets/pdf_file/0007/70000/571_Gemma_Tillack.pdf last accessed 13 May 2007

[53] Hayward J, Submission to the Senate enquiry on Plantations for Australia: The 2020 Vision. Available at: http://www.aph.gov.au/SENATE/committee/rrat_ctte/completed_inquiries/2002-04/plantation_forests/submissions/sub54.doc last accessed 14 August 2007

[54] ibid.

[55] Inquiry recommends brake on forest plantations, *The Age*, 2 September 2004. Available at: http://www.theage.com.au/articles/2004/09/02/1093939060279.html last accessed 14 August 2007

[56] ibid.

[57] 7.30 Report*,* 2004, television program, *Australian Broadcasting Corporation*, Sydney, 19 July. Available http://www.abc.net.au/7.30/content/2004/s1157381.htm last accessed 14 August 2007

next section provides an analysis of the subsequent events and the claims and counter-claims over the possible cause of the problems.

9.7.1 The St Helen's incident

In 2004 a record flood in the Georges River, in the north east of the state, coincided with the crash of the helicopter carrying chemicals used to aerially spray plantation forests in the catchment. The combination of chemical spill from the helicopter and the subsequent flood resulted in a mass mortality of oysters on the intertidal leases, plus the deaths of other aquatic and terrestrial organisms in the Georges Bay. Steve Percival, commissioned by DPIPWE, in a report of the incident, advised that a number of issues had been raised during the course of the investigation.[58] However, he concluded that it was not possible within time and budget constraints to confirm in any detail the validity or otherwise of any specific concerns in relation to the chemicals.[59]

The St Helen's Marine Farmers and Drs Bleaney and Scammell, frustrated with the inconclusive findings of the report and DPIPWE's apparent lack of concern, undertook an independent study. This study collated by Scammell became known as either the *Scammell and Bleaney Report (SBR)* or the *Scammell Report*.[60] The study was diligently undertaken and incorporated anecdotal evidence from the oyster farmers who reported extensive aerial spraying of chemicals in the month prior to the flood and helicopter crash in the upper Georges River catchment.

---

[58] Percival S, 2004, *Oyster Health in Georges Bay, Collation and analysis of data*, Tasmanian Department of Primary Industry, Water and Environment, Hobart
[59] ibid.
[60] Scammell M, 2004, Environmental Problems Georges Bay, Tasmania. Available at: http://www.tfic.com.au/domino/tfic/tficweb.nsf/vwTitle/07.04%20Scammell%20Report last accessed 13 May 2007

The content of the payload of chemicals dispersed from the helicopter on the day of the crash has never been fully disclosed. But the DPIPWE, in response to the *Scammell Report,* released the information shown in Table 9:2 below detailing the chemicals used in forestry operations in the Georges River catchment during 2003/4.

Table 9:2 Pesticides used in forestry operations in Georges River Catchment 2003/4

Chemical	Type	Total Quantity
Alpha-cypermethrin	Insecticide	29kg
Glyphosate	Herbicide	70.4kg
Sulfometuron-methyl	Herbicide	2.7kg
Terbacil	Herbicide	42.2kg

An assessment by DPIPWE of the soil at the crash site, taken sixteen weeks after the helicopter crash and chemical spill, identified the chemicals shown in Table 9:3 below.

Table 9:3 Chemicals identified in soil at crash site 5 April 2004

Chemical	Type	Total Quantity
Simazine	Herbicide	254mg/kg
Atrazine	Herbicide	75mg/kg
Chlorothalonil	Fungicide	1.25mg/kg

The DPIPWE gave two possible explanations for the discrepancy. Firstly, the presence of these chemicals was due to the spray-tank not being washed out properly, or secondly, it was residual spray from the time of the plantation establishment.[61]

[61] Christiaan Jonkers BSc (Forestry) ANU in an email dated 19 August 2004 to Dr Lloyd-Smith, National Toxic Network

The *Scammell Report* received highly critical reviews following its publication. Professor Paolo Ricci of the University of Queensland was commissioned by the DPIPWE to undertake a review with another undertaken by DIER, both Tasmanian government departments. They attacked the credibility and independence of the report. Ricci described the *Report* as an attempt at a manifesto based on unsound science.[62] Ricci's review later appeared on the website of CropLife Australia, an agrichemical industry funded group established to promote industry views.[63] The DIER review was published on the National Association of Forest Industries (NAFI) website together with claims that the *Scammell Report* had been 'labelled alarmist and unscientific by a Tasmanian State Government review'.[64] These reviewers' criticisms of a report that raised genuine concerns about the problems facing species exposed to environmental contaminants created confusion in the public debate and further contributed to the uncertainty as to the cause of the problems.

The Tasmanian government, the forestry industry and the chemical industries have a vested interest in avoiding criticism and continuing the expansion of plantation forests in Tasmania. The Tasmanian government through DPIPWE is responsible for Chemical Management and Pesticide Monitoring in water catchments and the preservation of native plants and animals including overseeing the Tasmanian Devil Facial Tumour Disease Program. DIER is responsible for encouraging the advancement of forestry production on both public and private land. CropLife Australia is a

---

[62] Ricci P, 2004, *Review of Drs A. Bleaney and M. Scammell Report (BSR), Compiled by Dr Scammell*. Available at: http://www.dpiw.tas.gov.au/intertext.nsf/Attachments/LBUN-63C9LL/$FILE/Review%20of%20Bleaney%20&%20Scammell%20Report%20Final.pdf last accessed 4 August 2007

[63] CropLife Australia, *Environmental Problems Georges Bay, Tasmania*. Available at: http://www.croplifeaustralia.org.au/default.asp?V_DOC_ID=1203 last accessed 1 August 2007

[64] Australia's National Association of Forest Industries, 'Oyster death study rejected by review'. Available at: http://www.nafi.com.au/news/view.php3?id=1108 last accessed 1 August 2007

subsidiary of CropLife International,[65] the peak representative body of the chemical industry established to promote their interests. [66]

The problem of water contamination is ongoing and the effort to bring accountability to the state government and the forestry industry continues. Of the many chemicals used in plantation forestry my focus remains on atrazine and the poison 1080 and their use in Tasmania.

9.8 Chemicals of concern – atrazine and 1080

In Tasmania atrazine is a widely used pesticide in both agriculture and forestry. The forestry industry claims that it has adopted atrazine for use in plantation forestry because its limited usage does not warrant expensive development and registrations of a more appropriate herbicide.[67] Atrazine is used in both softwood (pine) and in hardwood (eucalypt) forestry plantations. In hardwood plantations it is applied to control weeds for the first two years of planting, which includes a pre-plant broadcast over the whole plantation and a follow up one year later.[68] Atrazine is applied to eucalypts at the rate of 4,500 to 8,000 grams per hectare compared to agricultural crops, which is 2,000 – 2,900 grams per hectare.[69] The application of pesticides in plantations occurs in winter,

[65] Based in Brussels, CropLife International (formerly the Global Crop Protection Federation) is a global federation 'representing the plant science industry' and lead by: BASF, Bayer CropScience, Dow AgroScience, DuPont, FMC, Monsanto, Sumitomo and Syngenta. In 2003 Michael Pragnell, the chief executive of Syngenta, the world's largest agro-chemicals company became the president of CropLife International. Available at: http://www.lobbywatch.org/profile1.asp?PrId=159 last accessed 14 February 2011

[66] CropLife Board. Available at: http://www.croplifeaustralia.org.au/default.asp?V_DOC_ID=1895 last accessed 14 February 2011

[67] Jenkins BM & Tomkins B, 2006, *Pesticides in Plantations*, Forest and Wood Products Research and Development Corporation, Melbourne, Victoria

[68] Parsons M, Gavran M & Davidson J, 2006, *Australia's Plantations 2006*, Bureau of Rural Sciences, Canberra

[69] Jenkins BM & Tomkins B, 2006, *Pesticides in Plantations*, Forest and Wood Products Research and Development Corporation, Melbourne, Victoria

between the months of May and November.[70] This contributes to a greater potential for runoff than with agricultural use, because during winter the soils are wetter.[71]

The use of atrazine in forestry is highly controversial, particularly in relation to the danger it poses in the environment. In 2004 in an interview with *ABC* reporter Ticky Fullerton, John Gay, the then Chief Executive of Gunns Limited, admitted that Gunns did use atrazine in aerial spraying but claimed 'we only use it where it's very necessary and we use it where it's very, very safe'.[72] The timber industry lobby organisation, the National Association of Forest Industries (NAFI), has rejected reports that atrazine is harmful, stating that 'atrazine was declared a class 3 carcinogen by the World Health Organisation, putting it in the same class as talcum powder and coffee'.[73] They further suggested that reporting be based on scientific facts not 'the beliefs of scare mongers'. The volatility of the situation was summed up in a television program entitled *The Poisoning of Tasmania* when *Channel 9* reporter, Graham Davis, was told by a spokesperson for Gunns that any link made between the use of chemicals in the industry and human or animal health would result in legal action.[74]

---

[70] Davies PE, Cook LSJ & Barton JL, 1994, 'Triazine Herbicide Contamination of Tasmanian Streams: Sources, Concentrations and Effects on Biota' *Australian Journal of Marine and Freshwater Resources,* No. 45, pp 209-226

[71] Tasmanian Government, 1997, *State of the Environment*. Available at: http://soer.justice.tas.gov.au/2003/copy/111/index.php last accessed 15 August 2007

[72] Four Corners*,* 2004, television program, *Australian Broadcasting Corporation*, Sydney, 16 February 2004. Available at: http://www.abc.net.au/4corners/content/2004/s1134241.htm last accessed 14 August 2007

[73] National Association of Forest Industries, 2005, 'Atrazine necessary and safe' *NAFI e-news*, Issue 71, p 4. Available at: http://www.nafi.com.au/files/newsletter/NAFI%20eNews%20No.%2071.pdf last accessed 15 August 2007

[74] Davis G, 'The Poisoning of Tasmania/Tasmania: name your Poison', *news-tasmania.com.* Available at: http://www.news-tasmania.com/sept-04.html last accessed 14 August 2007,

In contrast Forestry Tasmania ceased using atrazine in 1995 as a consequence of a contamination incident.[75] It now controls weeds in plantation forestry through strip spraying and more intensive site preparation.[76] Aware of the potential for off-site contamination, Forestry Tasmania has adopted a State Policy on Water Quality Management, which includes as one of its principal objectives the application of the precautionary principle to achieve water quality objectives.[77] However, Gunns Limited and private plantations owners still continue to use atrazine. John Mollison, Registrar of Chemical Products, DPIPWE confirmed atrazine is used in plantations.[78]

DPIPWE's list of pesticides for monitoring includes atrazine as well as the triazines, cyanazine, hexazinone and simazine.[79] The rationale for monitoring these pesticides is based on their common usage, persistence and toxicology. In 2009 atrazine's potential to contaminate ground water in Tasmania was also evidenced in a pilot study when it was detected in both Port Arthur and Ross.[80] Atrazine remains a chemical of concern in Tasmania.

9.8.1 Poison of interest: Sodium Fluoroacetate (1080)

Sodium Fluoroacetate, more commonly known as 1080, is highly toxic to mammals including humans.[81] The use of 1080 was pioneered in Australia in the 1950s to control

---

[75] Elliott HJ & Hodgson BS, 2004 'Water sampling by Forestry Tasmania to determine presence of pesticides and fertilizer nutrients, 1993-2003' *Tasforests*, Vol 15, pp 29-42
[76] ibid.
[77] ibid.
[78] *Earthbeat with Alexandra de Blas*, 2004, radio program, Australian Broadcasting Commission. Available at: http://www.abc.net.au/rn/science/earth/stories/s1160346.htm last accessed 26 September 2007
[79] Department of Primary Industries, Parks, Water and Environment, Pesticides Monitored. Available at: http://www.dpiw.tas.gov.au/inter.nsf/webpages/cart-69stwk?open#PesticidesMonitored last accessed 24 May 2012
[80] Department of Primary Industries, Parks, Water and Environment, Ground Water, Bore Sample Results. Available at: http://www.dpiw.tas.gov.au/inter.nsf/Attachments/CART-7UG2JT/$FILE/Ground%20Water%20Monitoring%20Project.pdf last accessed 24 May 2012
[81] Sodium fluoroacetate (1080). Available at: http://toxipedia.org/display/toxipedia/Sodium+fluoroacetate+(1080) last accessed 10 June 2012

rabbit populations.[82] In Tasmania it has been used to control native browsing animals in plantations and more recently in a controversial fox eradication program. With the widespread increase in eucalypt plantations, the need to control native browsers also increased. The species most targeted because they are known to damage eucalypt seedlings were the brushtail possum (*Trichosurus vulpecular*), the red-bellied pademelon or rufous wallaby (*Thylogale billardierii*) and Bennett's wallabies, (sometimes called kangaroo; *Macropus rufogriseus*).[83] In the field 1080 is used at a concentration of 0.014 per cent of active ingredient in carrots for poisoning native animals.[84] The lethal dose of the target species and the Tasmanian devil are given in Table 9:4 below.

Table 9:4 Native species lethal dose of 1080[85]

Native Species	Lethal dose of 1080 mg/kg body weight
Bennett's wallaby	<0.2
Pademelon	0.13
Possum	0.7
Wombat	1.5
Eastern quoll	3.7
Tasmanian devil	4.2

In 1981 McIllroy conducted studies into the sensitivity of Australian animals to 1080 poison including marsupial and eutherian carnivores.[86] Included in the study were

[82] Australian Pesticides & Veterinary Medicines Authority (APVMA), 2008, *Sodium Fluoroacetate Final Review Report and Regulatory Decision,* APVMA, Canberra
[83] Farm Forestry, Browsing Damage to Seedlings, Technical Information Sheets No. 18, Level 2. Available at: http://www.privateforests.tas.gov.au/files/attachments/18BrowsingDamageToSeedlings2.pdf last accessed 10 June 2012
[84] Department of Primary Industries, Parks, Water and Environment, 1080 Poison. Available at: http://www.dpiw.tas.gov.au/inter,nsf/webpages/rpio-4zm7cx?open last accessed 10 June 2012
[85] ibid.
[86] McIlroy JC, 1981, The Sensitivity of Australian Animals to 1080 Poison II. Marsupial and Eutherian Carnivores, *Australian Wildlife Research*, Vol 8, pp 385-399

Tasmanian devils. Experiments were conducted on 5 male devils (mean weight 4.67 kg) from the southeast of the state and it was found that death occurred between 2.6 and 22.3 hours. McIllroy noted that devils responded to the ingestion of the poison by vomiting but there was still sufficient time for many of them to absorb a lethal dose. However there was considerable individual variability. He noted a number of limitations to the study including:

- the small sample size;
- the experiment did not indicate what would happen in a wild population;
- it did not take into account the distribution and density of baits in relation to the distribution and density of the target and non-target species; and
- the length of time the poison remained unleached in a field situation.[87]

In relation to secondary poisoning by eating poisoned animals, McIllroy noted there was not enough data available to form a theoretical assessment. 1080 itself is not toxic; its lethal action is due to conversion to fluorocitric acid. Fluorocitrate in the body inhibits the enzymes aconitase and succinate dehydrogenase; the accumulated citrate interferes with energy production and cellular function.[88] 1080 is readily absorbed through the gastrointestinal tract, mucous membranes, and pulmonary epithelia; once absorbed, it is uniformly distributed in the tissues.[89] Except for McIllroy's studies there appear to be no further studies into the short or long term effects of direct or secondary poisoning by 1080 on devils.

---

[87] ibid, p 396
[88] Eisler R, 1995, Sodium Monofluoroacetate (1080) *Hazards to Fish, Wildlife, and Invertebates: A Synoptic Review,* Patuxent Environmental Science Center, U.S. National Biological Service, Laurel, MD, p 9
[89] ibid.

As a result of the ongoing concern regarding the 1080 poisoning of non-target animals the Australian regulator, the APVMA, began a review in 2002. The Sodium Fluoroacetate Final Review Report and Regulatory Decision found that 1080 is used across mainland Australia to control feral animals such as rabbits, wild dogs, foxes and feral pigs.[90] In Tasmania, however, it is used to control native mammals (Bennett's wallaby, Tasmanian pademelon and brushtail possum) grazing on crops and tree seedlings.[91] The key outcomes of the Report were amendments to the labels and implementation of new conditions for registration.[92] Incongruously, in 2008 the APVMA in its Final Review Report on 1080 noted that 'Tasmanian devils …maintain stable and increasing populations in the face of baiting'.[93] Gunns reported it no longer uses 1080 to kill browsers as it has 'developed new strategies for protecting its plantations, which do not involve the lethal poison'.[94] This new strategy is to employ shooters who go out at night and use spotlights to detect and kill browsers.

Despite the lack of studies into the effects of direct or secondary 1080 poisoning of devils and the promised phasing out of 1080 poison to control herbivores in Tasmanian forestry plantations, its use continues in a baiting regime to control a suspected fox population. In 2001 the Tasmanian government established the Tasmanian Fox Free

---

[90] Australian Government, Australian Pesticides and Veterinary Medicines Authority, 2008, *Sodium Fluoroacetate Final Review Report and Regulatory Decision*, Australian Pesticides and Veterinary Medicines Authority, Canberra

[91] ibid.

[92] Australian Pesticide and Veterinary Medicines Authority, 2008, *Sodium Fluoroacetate, Final Review Report and Regulatory Decision, The Reconsideration of Registrations of Products Containing Sodium Fluroacetate (1080) and their Associated Labels*, APVMA, Canberra

[93] Australian Pesticide and Veterinary Medicines Authority, 2008, *Sodium Fluoroacetate, Final Review Report and Regulatory Decision, The Reconsideration of Registrations of Products Containing Sodium Fluroacetate (1080) and their Associated Labels*, APVMA,Canberra, p 30

[94] Personal communication.

Taskforce.[95] In 2006 the taskforce was expanded and renamed the Fox Eradication Program with $56 million in funding from Australian and Tasmanian governments for a 10-year strategy.[96] The well resourced "fox squads" used the latest technology to investigate sightings and gather evidence of fox activity, including footprints and possible den sites. It was reported in *The Mercury* newspaper in 2007 that the Tasmanian government had spent $5.1 million in the previous five years to set up the Taskforce with a contribution of $1.3 million from the Federal Government.[97] Concerns over the cost of the fox eradication program prompted a Tasmanian Parliamentary inquiry, which recommended that the program continue and that the precautionary principle should apply, stating 'as such this primary focus [to locate, bait and eradicate foxes] should not be unreasonably distracted by an on-going need to substantiate the presence of foxes'.[98]

By 2006 some 80,000 fox baits containing 1080 had been spread across the state in response to sightings and reports.[99] A DPIPWE map of fox locations across Tasmania and their evidence are shown in Figure 9:3 below.

---

[95] Saunders G, Lane C, Harris S & Dickman C, 2006, *Foxes in Tasmania: a Report on an Incursion of an Invasive Species*, Invasive Animals Cooperative Research Centre, Belconnen, ACT

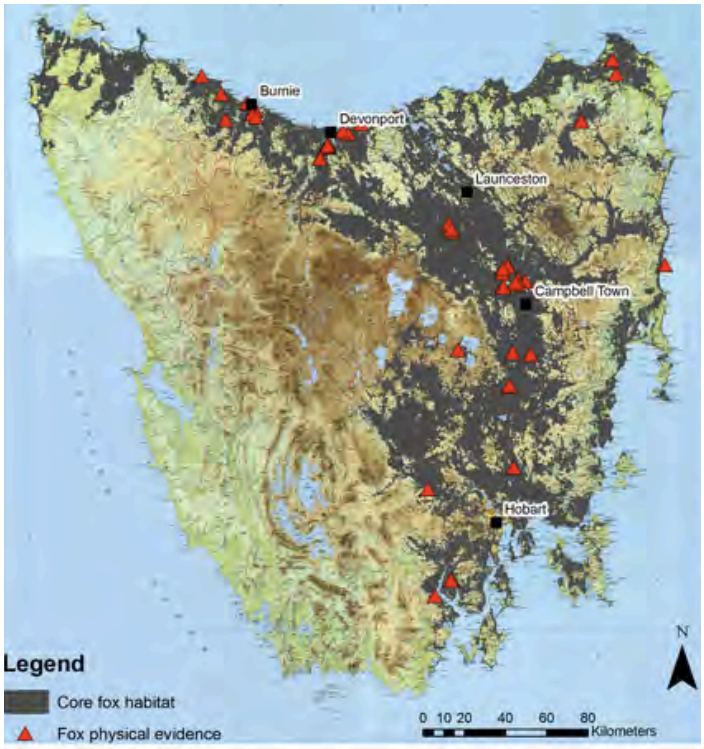
[96] Jeanes T, 2006, Tas Govt announces $56m fox eradication program, *The World Today*, ABC Radio. Available at: http://www.abc.net.au/worldtoday/content/2006/s1788366.htm last accessed 23 February 2013

[97] Neales S, 2997, Tassie 'hookwinked', *The Mercury*. Available at: http://www.news.com.au/mercury/story/0,22884,21582109-40007221,00.html last accessed 19 April 2007

[98] Parliamentary Standing Committee of Public Accounts, 2009, *Inquiry into the Efficiency and Effectiveness of the Fox Eradication Program in Tasmania,* Parliament of Tasmania, Hobart, Tasmania p 3

[99] Saunders G, Lane C, Harris S & Dickman C, 2006, *Foxes in Tasmania: a Report on an Incursion of an Invasive Species*, Invasive Animals Cooperative Research Centre, Belconnen, ACT

Figure 9:3 Locations of physical evidence of fox activity and identified core fox habitat in Tasmania[100]



Interestingly Saunders et al state '[t]he decline in devil populations occurs coincidently in the same area where most fox sighting reports have been received from the public'.[101] Foxes are targeted with 1080 baits of either dry kangaroo meat or commercially produced baits such as Foxoff ®. Baits are laid by aerial or ground operations at least four times a year at a rate of 5 baits per square kilometer. According to the Fox Eradication Program Fact Sheet, 3mg of 1080 per bait is used to target foxes.[102] According to the Fact Sheet a single fox bait 'is of no risk to a Tasmanian devil'.[103]

[100] Tasmanian government, Department of Primary Industries, Parks, Water and Environment, *DPIPWE – Fox Baiting Program*. Available at http://www.dpiw.tas.gov.au/inter.nsf/WebPages/SSKA-6H27T5?open last accessed 15 February 2013
[101] Saunders G, Lane C, Harris S & Dickman C, 2006, *Foxes in Tasmania: a Report on an Incursion of an Invasive Species*, Invasive Animals Cooperative Research Centre, Belconnen, ACT, p 30
[102] Fox Eradication Program, Fact Sheet 01, August 2010, Department of Primary Industries, Parks, Water and Environment, Hobart
[103] Department of Primary Industries, Parks, Water and Environment, Invasive Species, Frequently asked questions about the Fox Eradication Program, Will fox baits kill native wildlife. Available at:

Pilot studies into the effects of 1080 on native species including the Tasmanian devil and its close relative the spotted-tailed quoll suggest little damage to local populations.[104] A massive reduction in the number of eastern quolls however has been recorded in Tasmania with declines of 61-100% observed in some trapping surveys.[105]

Until the recent fox eradication program it was not legal to use 1080 in Tasmania for any form of predator control except for dogs. However, a Code of Practice for the use of 1080 against foxes under the current emergency situation was released in June 2002.[106] In 2004, in Tasmania an amendment to the *Agricultural and Veterinary Chemicals (Control of Use) Act 1995* made it illegal for government agencies to poison native wildlife using 1080 beyond December 2005. An exemption applies to the Fox Taskforce allowing fox control to continue until October 2006. The DPIPWE Fox Baiting Program Activity and Location statements indicate however that baiting is still occurring (2013). [107]

Considerable scientific uncertainty surrounds the effects of 1080, both short and long term, and from direct and secondary poisoning on the Tasmanian devil. The Tasmanian Government admits it does not know the volume of chemicals entering the state's waterways whilst the forestry industry is not obliged to divulge information on the use

---

http://www.dpiw.tas.gov.au/inter.nsf/WebPages/MMAN-7PY98B?open#Q7 last accessed 12 November 2013

[104] Department of Primary Industries, Parks, Water and Environment, Invasive Species, Effects of fox baiting on spotted-tailed quolls and Tasmanian devils. Available at: http://www.dpiw.tas.gov.au/inter.nsf/WebPages/JBRN-6VR882?open last accessed 12 November 2013

[105] Fancourt BA, Hawkins CE & Stewart CN, 2013, Evidence of rapid population decline of the eastern quoll (Dasyurus viverrinus) in Tasmania, *Australian Mammalogy*, Vol 35(2), pp 195-205

[106] Reported in the Saunders et al paper but I have not been able to locate such a document on the web.

[107] Department of Primary Industries, Parks, Water and Environment, Fox Baiting Program Activity and Location. Available at: http://www.dpiw.tas.gov.au/inter.nsf/WebPages/MMAN-7H32QE?open last accessed 14 January 2013

of chemicals having been exempted from Freedom of Information legislation.[108] To add further uncertainty to an already precarious situation for native species is the suggestion that plantation forestry eucalypts may be genetically modified.

9.9 Genetically modified eucalypts?

The *Scammell Report* initially raised the concern of the St Helens oyster growers that their oysters showed signs of abnormality and suffered occasional mass deaths; the issue is ongoing and remains unresolved. In 2005 the DPIPWE investigations had found no toxicity in water samples collected from the George River catchment.[109] However, foam samples collected with skimmer boxes following analyses revealed an unknown toxin. The DPIPWE concluded that naturally occurring eucalypt oils were likely to be responsible, but claimed the concentrations were well below those known to result in toxicity. The oyster growers were not convinced by DPIPWE's proposals that either their management practices or naturally occurring eucalypt oils were the cause of the problems. As a result Ecotox Services Australasia Pty Ltd (ESA) was commissioned to undertake further studies to identify the toxicity of foam samples. ESA undertook a Toxicity Identification Evaluation (TIE) study in order to determine the cause of the toxicity.

Between January 2005 and 2008 an unknown toxin was found to be present at hazardous concentrations, it was claimed not to be a known man-made chemical, but an organic chemical.[110] It was also neither a cineole or a pinene (naturally occurring eucalypt oils) as suggested by DPIPWE, nor was it a cyanobacterial toxin or a known

[108] Flanagan R, 2007, 'Rape of Tasmania' *The Bulletin*, Vol 121(50), 16 December 2003
[109] Analytical Services Tasmania, 2005, Toxicological Testing IQ-Toxicity Test, Report No. 24444A, Issue No. 1
[110] Bleaney A & Scammell M, 2010, George River Catchment Investigation (NE Tasmania). Available at: http://tasmaniantimes.com/images/uploads/Press_Releasex.pdf last accessed 13 June 2012

protein. But the unknown toxin was present in the Eucalyptus *nitens* leaves and the toxin in the leaves was the same as the toxin in the TIE experiment. Importantly, the toxin was not found in samples from undisturbed natural catchments.

Chris Hickey and Michael Stewart at the National Institute of Water and Atmospheric Research Ltd (NIWA) in New Zealand also undertook similar studies. Their findings confirmed the above results.[111] They also observed that the organisms were not only killed by the unknown toxins but were actually dissolved. This observation had not previously been seen in their laboratory. The toxicity was associated with particulate matter in foam samples and eucalypt leaf extracts.[112] At the Australasian Society for Ecotoxicology Conference in Adelaide in 2009 Hickey and Stewart made reference to research undertaken by Rosi-Marshall et al, which showed that byproducts, such as pollen and detritus from transgenic crops could be transported downstream.[113] Hickey and Stewart concluded that the issue needed a lot more investigation. To date no further investigations have been undertaken by either the Tasmanian government or independent researchers. No studies have been peer reviewed or published in relation to these findings.

The issue however went public in 2010 when the Australian Broadcasting Corporation (ABC) produced *Something in the Water,* a two-part series based on these investigations. In response to the program the then Premier of Tasmania David Bartlett wrote to the *ABC* Managing Director on 5 July 2010 enclosing the George River Water Quality

[111] Hickey C & Stewart M, 2010, *Catchment studies in Georges Bay, Tasmania: base-flow water and foam toxicity to cladocerans and blue-mussels,* National Institute of Water and Atmospheric Research, Hamilton, New Zealand
[112] Hickey C & Stewart M, Catchment studies in Georges Bay, Australia: base-flow water and foam toxicity to cladocerans and blue-mussels. A case of unintended consequences? (unpublished)
[113] Rosi-Marshall EJ, Tank JL, Royer TV, Whiles MR, Evans-White M, Chambers C, Griffiths NA, Pokelsek J & Stephen ML, 2007, Toxins in transgenic crop byproducts may affect headwater stream ecosystems, *PNAS*, Vol 104(41), pp16204-16208

Panel report and complained that the program 'had included allegations that were wrong and based on severely flawed science'.[114] In June 2013 the Tasmanian government, through DPIPWE Biosecurity section, commenced a review of the current policy on GMOs and called for public submissions.[115] A moratorium on the commercial release of GMOs has been in place in Tasmania since 2001.[116]

9.9.1 Support for genetically modified eucalypts

In 2001 the Rural Industries Research and Development Corporation published a paper on the risk of genetic pollution from farm forestry using eucalypt species and hybrids.[117] It noted traits being considered for eucalypts included modification for herbicide resistance, insect resistance, sterility, improvement of rooting ability, modification of lignin content and composition, amongst others. It further noted species and clones from which transgenic plantlets have been recorded, including from Eucalyptus *globulus*. But deployment of transgenic material from Eucalyptus *globulus* and Eucalyptus *nitens* (both used in forestry plantations in Tasmania) would have to await the development of efficient vegetative propagation systems. Genetic modification of eucalypt trees for plantation forests, if not established in Tasmania, is supported.

Dr Jim Peacock, former Chief of Division at the CSIRO Plant Industry, and a proponent of genetically modified organisms, at an Academy of Science Symposium in 2000, gave a brief overview of the potential for GMOs in economic and environmental

---

[114] ibid.
[115] Department of Primary Industries, Parks, Water and Environment, Biosecurity, Tasmania – Gene Technology and Primary Industries 2013 Review of the Current Policy on Genetically Modified Organisms. Available at: http://www.dpiw.tas.gov.au/inter.nsf/WebPages/EGIL-53876E?open last accessed 26 June 2013
[116] Macquarie Franklin, 2012, *Market Advantage of Tasmania's GMO-free Status*, Devonport, Tasmania
[117] Potts BM, Barbour RC & Hingston A, 2001, Genetic Pollution from Farm Forestry using eucalypt species and hybrids, Rural Industries Research and Development Corporation. Available at: http://diversitynativeseeds.com.au/attachments/Eucalyptus_Hybridisation_Research_Summary.pdf last accessed 13 June 2012

sustainability. Peacock was enthusiastic in proposing developments in plantation forestry 'through genetic engineering, eucalypt species with enhanced productivity and quality characteristics'.[118] He went on further to state:

> Our understanding of gene expression is being applied to genetic engineering of plantation eucalypts for sterility and insect resistance. Not only as a landscape dewatering option, but also because of pressure to move away from logging in native forests is increasing in Australia and forest industries must seriously consider plantation Eucalypt production to remain competitive in the world market. Insect pests are a serious problem but the economics of insecticide usage in plantations is prohibitive, so a genetic solution like that being used in cotton is being developed using transgenic Eucalyptus species.
>
> An important component is the development of transformation systems for commercially important Eucalypt species. Because of the tendency for eucalypts to outcross, it is essential that transgenic pest tolerant varieties produced are sterile to avoid the possibility of deleterious impacts of escaping genes on the native forest ecosystems that will inevitably surround commercial eucalypt plantations.
>
> Important genes in the regulation of flowering in eucalypts are being isolated and genetic engineering is being used with these genes to interfere with the key events in floral initiation and development to produce completely sterile plants that can be used with safety near our native forests.

Peacock's interests and work would also engage him in further genetic enhancement of Australian crops. In 2003 commenting on Sygenta's announcement that it would join Grain*gene*[119] he stated '[w]e are looking forward to working with Syngenta's researchers…[a]ccess to Syngenta's genomic tools and intellectural property will complement Grain*gene's* ability to deliver value added products in Australian cereals'.[120] Peacock was poised to have a close working relationship with the

---

[118] Peacock J, Sustainable Agriculture, National Science Academy Symposium, Sustainable Australia? November 2000. Available at: http://www.atse.org.au/index.php/index.php?sectionid=544 last accessed 28 Feb 2010

[119] Graingene – Australian national crop genetics research consortium.

[120] Media Release, 2003, New Commercial Focus for Grain Research, Graingene. Available at: http://www.awb.com.au/investors/companyannouncements/mediareleases/2003mediareleases/05.03.03NewCommercialFocusForGrainResearch.htm last accessed 31 January 2013

manufacturers of atrazine. In 2007 as the Chief Scientist of Australia Peacock would play an active role in assessing the proposed Gunns Limited pulp mill in Tasmania.[121]

In an article by Desmond Stackpole and Brad Potts of the Cooperative Research Centre (CRC) for Forestry, at the University of Tasmania and colleague, Kelsey Joyce of Gunns Limited, they noted that the Eucalyptus *nitens* and Eucalyptus *nitens x globulus* eucalyptus trees are F1 clonally replicated genotypes.[122] There is also an abstract by Naomi Glancy, Julianne O'Reilly-Wapstra and Brad Potts on breeding to enhance the resistance of Eucalyptus *nitens* to marsupial browsing.[123]

The Southern Tree Breeding Association (STBA) manages the improvement programs for Eucalyptus *globulus*. It was formed in 1983 as a not-for-profit cooperative. STBA genetic material is extensively tested in trials spread across the plantation estate in temperate Australia. Genetically improved seed and plants can be obtained directly from STBA Members and/or *seedEnergy* Pty Ltd, a licensed seed producer.[124] Members of STBA include Forestry Tasmania, Gunns Ltd, Hancock Victoria, CRC for Forestry, CSIRO Forestry and Forest Products, Scion, Norske Skog Paper Mills (Australia) Ltd and the University of Melbourne (School of Forest and Ecosystem Science).

---

[121] Australian Government, Department of the Environment and Water Resources, 2007, Summary of Advice Provided by the Chief Scientist of Australia on the Gunns Limited Proposed Pulp Mill (EPBC 2007/3385). Available at: http://www.environment.gov.au/epbc/notices/assessments/2007/3385/pubs/summary-chief-scientist.pdf last accessed 8 August 2013

[122] Stackpole D, Joyce K & Potts B, Correlated response of pulp-wood profit traits following differential fertilization of an *Eucalyptus nitens* clonal trial, *Australasian Forest Genetics Conference*, 11-14 April 2007, Hobart, Tasmania, p 46

[123] Glancy N, O'Reilly-Wapstra J & Potts B, Breeding to enhance the resistance of Eucalyptus nitens to marsupial browsing, *Australasian Forest Genetics Conference*, 11-14 April 2007, Hobart, Tasmania, p 52

[124] The Southern Tree Breeding Association, Program Book, *Australasian Forest Genetics Conference,* 11-14 April 2007, Hobart, Tasmania,

Notwithstanding the support for genetically modified eucalypts, the Tasmanian government has vehemently denied the proposition that the trees might be genetically modified. Tasmania currently claims to be a GM free state.

9.10 Conclusion

The Tasmanian state government and the forestry industry have invested heavily, both politically and economically, in realizing a forest plantation industry. The inherent risks associated with operating and managing the plantations have been discounted in an effort to achieve these outcomes. Plantation forests are critical to the Tasmanian government's long-term solution to the controversy over the logging of old-growth and native forest in Tasmania. Recognition of a correlation between chemicals used in plantation forestry and the spread of Devil Facial Tumour Disease would be detrimental to this plan. Therefore, there has been government resistance to adoption of precautionary regulation, as it would mean restricting use of these chemicals in plantation forests.

Since the scientists researching the Devil Facial Tumour Disease in Tasmania are employed by the government they have avoided an examination of an alternative hypothesis to the allograft theory that might implicate plantation forests. Independent scientists would find it difficult to secure devil samples to conduct their own studies, as theses samples are controlled by the government.

The DPIPWE has produced evidence that Tasmanian waterways are regularly contaminated with chemicals used in agriculture but mainly from plantation forestry. Despite this ongoing evidence the Tasmanian government continues to allow the use of these chemicals, as it is determined the detected levels are safe. A conflict of interest is

apparent however when the two government departments DIER and DPIPWE oversee the promotion of forestry plantation, the use of chemicals and the protection of endangered species. All forestry plantation management, whether in respect to chemicals or aerial spraying, operate under a regime of self-regulation by the industry. These practices are exacerbated by a revolving door between the regulators and the industry.

Notwithstanding the lack of action to limit or prevent the continued use of these chemicals it has now been acknowledged that an unknown toxin has been found in these same waterways. This unknown toxin has been suggested to be either one of the natural occurring eucalyptus oils from the plantation trees or a product of genetic enhancement of the trees. Neither of these claims has been substantiated by scientific evidence. Similar unknown toxins have recently been identified outside Tasmania where genetic modification of crops has occurred. The debate over the unknown toxin, although important, should not become a diversion from the real issue of chemical contamination of rivers systems, ground water and drinking water. It is for this reason that, although I have addressed the issue of the unknown toxin and described the authorities' failure to fully research the problem, the issue of contamination by chemicals is the focus of this research.

The threatened extinction of the Tasmanian devil appears to be treated as irrelevant compared to industry progress and profit making. Whether pesticides used in plantation forestry, or GM trees, contributed to the Tasmanian devil cancer has not been tested. A more overt lack of protection for the Tasmanian devils is evident in the destruction of the devils' habitat through forestry and mining practices. The Tasmanian government

has also failed to implement a Recovery Plan under the *Environmental Protection and Biodiversity Conservation Act 1999*. These issues are the topic of the next chapter.

Chapter 10 – An inadequate policy response to the Tasmanian devil disease

10.1 Introduction

Habitat destruction by human activities has been identified as the major cause of biodiversity loss and species extinction.[1] In this chapter I will demonstrate that the Tasmanian government has not only failed to fully investigate the immediate threat of a deadly cancer, Devil Facial Tumour Disease (DFTD) but they have also neglected to protect devils' habitat. Forestry practices including logging of native forests and the establishment and maintenance of plantations, as does the practice of fox baiting, pose significant threats to the long-term survival of devils in Tasmania. A further threat comes from the proposal to expand mining in the area known as the Tarkine where the last remaining DFTD-free devils are said to exist.

The Tasmanian government's adoption of the precautionary principle in the eradication of foxes, justifying the use of 1080 to minimize their impact on Tasmania's biodiversity, exposes the principle's vulnerability to misuse and a possible weakness due to its many interpretations. Despite the Tasmanian government's willingness to adopt the precautionary principle in the eradication of foxes the same level of protection is lacking for native species in plantation forests including the Tasmanian devil. The listing of the Tasmanian devil as endangered under the *Environment Protection and Biodiversity Conservation Act 1999* (*EPBC Act*) underpinned by the precautionary principle warrants action to mitigate harm in the face of plausible scientific evidence.

[1] Chivian E, 1993, Species Extinction and Biodiversity Loss: The Implication for Human Health in E Chivian, M MCCally, H Hu & A Haines, (eds) 1993, *Critical Condition: Human Health and Environment*, MIT Press, Cambridge Mass. And London

Devil Facial Tumour Disease (DFTD) is evidence of harm, possibly irreversible, to the devil population whilst uncertain but plausible scientific evidence exists that atrazine, used in plantation forestry, might play a role in disease.

The use of the precautionary principle in the eradication of foxes is not supported by plausible scientific evidence. There has been no credible evidence of either the presence of a fox population or any harm inflicted by foxes on Tasmania's biodiversity.[2] Only recently has a continuing lack of evidence of the presence of foxes in Tasmania led to a reduction in the operations of the fox eradication program.

The Tasmanian government, through the Department of Primary Industries, Parks, Water and the Environment (DPIPWE) management of the Save the Tasmanian Devil Program (STDP), has shaped the research towards finding answers to a transmissible cancer, effectively evading an alternative hypothesis that forestry practices including the use of chemicals in plantations, may have contributed to the disease. In this chapter I argue that the Tasmanian government has been negligent in not pursuing all the scientific studies in relation to the devil cancer and in failing to protect the Tasmanian devil and its habitat. Although some management strategies under the *Tasmanian Threatened Species Act* have been adopted, there exists a serious lack of protection with the failure to implement a Recovery Plan under the *EPBC Act*, which would ensure the devil's protection from forestry practices and mining ventures.

In 2009 a continuing significant decline in devil numbers prompted the then Federal Environment Minister, Peter Garrett, to upgrade the *EPBC Act* listing of the Tasmanian

[2] Street J, 2013, Tasmania scraps fox baiting program, ABC News. Available at: http://www.abc.net.au/news/2013-06-13/fox-baiting-winds-down/4752636 last accessed 23 November 2013

devil to endangered in order to secure its protection.[3] A draft Recovery Plan prescribed under the *EPBC Act* for the protection of the Tasmanian devil has been drafted by DPIPWE and public comments have been sought but the implementation of the Plan is still awaited, as of December 2013. Consequently forestry practices plans for clear felling and logging do not take into consideration the need for protection of devil habitat. Whilst the Draft Recovery Plan recognizes the importance of habitat as critical to the long-term survival of the devils there is no assessment of the possible impacts of pesticide use in plantations on native wildlife including devils.

Forestry practices in Tasmania are controlled by the Regional Forests Agreements (RFAs), which include the development of nationally agreed criteria to protect forest biodiversity, old-growth forests and wilderness areas through the creation of world-class Comprehensive, Adequate and Representative (CAR) reserve systems. This system does not allow for RFAs to be exempt from the *EPBC Act.* RFA's are in fact understood to constitute a form of assessment and approval for the purposes of the *EPBC Act*.[4] This arrangement circumvents the need for the Commonwealth to be involved in every assessment of logging practices on a coupe[5] by coupe basis which was deemed administratively impracticable. The forestry industry, however, operates under a self-regulatory regime as discussed in the previous chapter.

---

[3] Australian Government, Department of Sustainability, Environment, Water, Population and Communities, Sarcophilus harrisii – Tasmanian Devil Listed as Endangered. Available at: http://www.environment.gov.au/cgi-bin/sprat/public/publicspecies.pl?taxon_id=299 last accessed 9 August 2012

[4] Australian Government, The Australian Environment Act: Report of the Independent review of the *Environment Protection and Biodiversity Conservation Act 1999*, Final Review, Chapter 10, Regional Forest Agreements. Available at: http://www.environment.gov.au/epbc/review/publications/final-report.html last accessed 8 August 2012

[5] Coupe is an area of forest with established boundaries which has been set aside for commercial forestry activities. Available at: http://www.daff.gov.au/rfa/publications/deferred/kit/glossary last accessed 9 August 2012

Mining operations also present a further potentially threatening process in the north west of Tasmania, in an area known as the Tarkine. Applications have been submitted for several proposed open-cut tin and tungsten mines in the region.[6] The last remaining DFTD-free devils are to be found in the north west of the state and it is critical for the long-term survival of the species that this habitat is maintained. Tony Burke, the then Environment Minister, approved the Shree Minerals' mine with a requirement that the developers donate $350,000 to the Save the Tasmanian Devil Program Appeal to compensate for the damage caused to the environment by the mine.[7] Hamish McCallum, former senior scientist with Tasmania's Save the Devil Program, observed this was an explicit recognition of the impact of the mine.[8]

An analysis of the relevant legislation reveals that there is little protection afforded the Tasmanian devil in either the state or private forests under Forestry Tasmanian or Private Forests Tasmania operations.

10.2 *Tasmanian Threatened Species Protection Act 1995 (TSP Act)*

The *Threatened Species Protection Act 1995 (TSP Act)* is a state government act, which sets out special protection measures for native animals and plants that are considered 'threatened'. It does not include the precautionary principle as a guide for action. Species declared as threatened are listed in the schedules of the *TSP Act* according to the nature of their threatened status. The Tasmanian devil was listed as 'endangered' – extinct or in danger of extinction (Schedule 3) - under the *TSP Act* in May 2008. The

[6] Denholm M, 2012, Tarkine the next forest flashpoint, *The Australian*. Available at: http://www.theaustralian.com.au/news/features/tarkine-the-next-forest-flashpoint/story-e6frg6z6-1226341076652 last accessed 14 January 2013
[7] McCallum H, 2013, Tarkine mines could be last straw for Tasmanian devils, *The Conversation*. Available at: http://theconversation.edu.au/tarkine-mines-could-be-last-straw-for-tasmanian-devils-11483 last accessed 30 January 2013
[8] ibid.

Secretary of DPIPWE carries responsibility for ensuring that the requirements of the *TSP Act* are implemented, although a number of key decisions are ultimately at the discretion of the Minister. Under the *TSP Act* there are five principal ways in which a species is protected:

- preparing a statewide strategy for the conservation of the threatened species in Tasmania – The Threatened Species Strategy
- preparing listing statements and implementing species recovery plans and threat abatement plans
- implementing land management plans, including special agreements with landowners and public bodies such as Forestry Tasmania
- declaring interim protection orders
- declaring critical habitats

Under the *TSP Act* it is prohibited to 'take' (kill, injure, damage or destroy)[9] a listed species without a special permit. However, a person or corporation can apply to the Secretary of DPIPWE for a permit under the *TSP Act* to take a threatened species or to take an action that is likely to result in harm. Forestry activities, which are likely to impact on threatened species, are exempt from the *TSP Act* under the following conditions:

> A person acting in accordance with a certified forest practices plan or a public authority management agreement may take, without a permit, a specimen of a listed taxon of flora or fauna, unless the Secretary, by notice in writing, requires the person to obtain a permit.

According to the Tasmanian Environmental Defenders Office (EDO) the *TSP Act* has all the tools necessary to protect the State's species and habitats. However, its implementation has been very slow and the *TSP Act* has yet to prove that it will

[9] Under the *Tasmanian Threatened Species Protection Act 1995* 'take' includes to kill, injure, catch, damage, destroy and collect a protected species. As the Tasmanian devil is a species listed under the *Act* as endangered it was also illegal to 'take' devils or samples for research. Available at: http://www.austlii.edu.au/au/legis/tas/consol_act/tspa1995305/s3.html last accessed 16 August 2012

successfully achieve what it sets out to do.[10] The EDO further states that the *Act* has key deficiencies including:

> Poor implementation and under-resourcing has meant that, at the time of writing [July 2010], many listing statements remain outstanding, no prosecutions have been commenced, no interim protection orders or critical habitats have been declared and no land agreements have been entered into.[11]

A determination for a critical habitat, under the *TSP Act*, is provided for by a map, which is then registered in the central plan office, under the *Survey Co-ordination Act 1994*. According to *Tasmania's Threatened Fauna Handbook, What, Where and How to Protect Tasmania's Threatened Animals* TASMAP contains map sheets of Tasmania showing where threatened species occur, their localities and areas containing potential habitat.[12] There is no record of a map describing critical habitat for Tasmanian devils but the handbook has not been updated since its publication in 1999. Concurring with the EDO's statement above, the *TSP Act* makes provision for land management plans, threat abatement plans and a recovery plan. However, no provisions are current for the Tasmanian devil.

All decisions under the Tasmanian *TSP Act* are referred to and made by a Scientific Advisory Committee. It is through this decision making process that threatened species are protected by determinations of critical habitat, strategies and plans. In relation to Tasmanian devils these determinations cover devil habitat in native forests and plantation forests but are not limited to these areas as devils occupy other habitats, such as coastal plains and scrublands. When a Committee making these decisions is not

---

[10] Environmental Defenders Office Tasmania, *The Environmental Law Handbook*, Chapter 7. Available at: http://www.edohandbook.org/doku.php?id=ch7#how_can_private_land_be_protected last accessed 15 August 2012

[11] ibid, p 16

[12] Bryant S & Jackson J, 1999, *Tasmania's Threatened Fauna Handbook, What, Where and How to Protect Tasmania's Threatened Animals*, Threatened Species Unit, Parks and Wildlife Service, Hobart, Tasmania

operating at arms length from either the Tasmanian government or the forestry industry then these processes are potentially compromised. Members of the Committee and their close association with the forestry industry are shown in Table 10.1 below.

Table 10.1 Scientific Advisory Committee

Committee Member	Affiliation
Raymond Brereton (Chair)	Formerly with Forest Practices Board and DPIPWE
Patrick Dalton	School of Plant Science, UTAS
Dr Christine Crawford	Tasmanian Aquaculture and Marine Institute
Dr Greg Jordan	School of Plant Science, UTAS working with Forestry Tasmanian CRC for Forestry
Dr Jean Jackson,	Formerly with DPIPWE
Dr Niall Doran	Formerly Senior Policy Analyst DPIPWE
Mark Wapstra	Formerly Forest Practices Authority

10.3 DPIPWE Strategies for the conservation of Tasmanian devils

In accordance with the *TSP Act* the DPIPWE implemented a number of strategies for the conservation of the Tasmanian devil through the *Save the Tasmanian Devil Program (STDP)*. The Commonwealth Government committed funding of $10 million over the 5 years to 2013.[13] The strategies progressed through three stages. The *STDP* commenced with Stage 1 from 2004-2006, which focused on understanding the nature of DFTD, recording the impacts of the disease and initiating research. Stage 2 was implemented between 2006-2008 devising a number of plans and strategies - a *Strategic Plan*, an *Insurance Population Strategy*, a *Business Plan* for 2007-2008 and a *5-year Business Plan* 2008-2013. The *Strategic Plan* was developed in 2007 and its vision was to establish an enduring and ecologically functional population of Tasmanian devils in

[13] Darby A, 2008, Tasmanian devil listed as endangered, *Sydney Morning Herald*. Available at: http://www.smh.com.au/news/environment/conservation/tasmanian-devil-listed-as-endangered/2008/05/21/1211182852173.html last accessed 12 August 2013

the wild.[14] The principles underlying the Plan were an understanding of DFTD in order to inform disease management based on sound science and peer review. These actions would be consistent with, and guided by, a statutory *Tasmanian Devil Recovery Plan* proposed to be implemented by 2008.[15] The *Strategic Plan* also included the development and implementation of a comprehensive insurance population strategy.

The *Insurance Population Strategy* was prepared by DPIPWE in conjunction with the Australasian Regional Association of Zoological Parks and Aquaria (ARAZPA).[16] The *Strategy* provides a framework to establish and maintain a healthy, viable insurance population of Tasmanian devils for 25 years, that:

- is free of DFTD;
- is genetically representative of the species;
- is able to sustain a harvest of animals for release to the wild; and
- provides for the maintenance of the suite of associated flora and fauna (commensal, symbiotic and parasitic) and wild behaviours wherever possible, to facilitate reintroduction to the wild.

A number of proposals to secure the Tasmanian devil population have been proposed and implemented. These include:

- the establishment of Australian mainland populations in various locations including the Devil Ark;[17]
- a proposal to construct a fence to protect devils from the disease;[18]
- Devil Island Project to establish a population offshore in Tasmania on Maria Island[19]

---

[14] Save the Tasmanian Devil Program: Strategic Plan, 2007. Available at: http://www.tassiedevil.com.au/tasdevil.nsf/file/82C18864F5819337CA2576CB0011569B/$file/STDP%20Strategic%20Plan%202007.pdf last accessed 20 August 2013

[15] Australian Parliament House, Senate Standing Committee on the Environment, Communications and the Arts, Answers to questions on notice, Environment, Water, Heritage and the Arts, Additional Budget Estimates 2008-2009, Question No: 43, Tasmanian Devil.

[16] Save the Tasmanian Devil Program: Insurance Population Strategy. Available at http://www.tassiedevil.com.au/tasdevil.nsf/file/82C18864F5819337CA2576CB0011569B/$file/STDP_Insurance%20Population%20Strategy_290707.pdf last accessed 9 August 2012

[17] Devil Ark founded by STDP and supported by the Australian Reptile Park and the New South Wales government Department of Environment and Heritage. Available at: http://www.devilark.com.au/our-partners last accessed 24 November 2013

[18] *ABC News*, 2012, Bid to fence in healthy devils. Available at: http://www.abc.net.au/news/2012-09-13/bid-to-fence-in-healthy-devils/4258766?section=tas last accessed 14 September 2012

In 2010 the *STDP* released its Stage 3: 2008-2013 *Communication Strategy*.[20] The strategy identifies target audiences and communication tools. It also covers media management and coordination for all media releases and publications. The Strategy directs the coordination of marketing, sponsorship and fundraising and makes provision for the support and facilitation of education and community awareness. The Program's objectives also include a commitment to conduct all research in a scientifically rigorous manner and to make all research results available to the public and stakeholders as soon as possible through publications in refereed scientific journals, technical reports and newsletters. The STDP emphasises that communication is critical to its success.

In 2011 the *STDP* prepared a *Monitoring Strategy* with its stated aim 'to ensure that the activities conducted by the monitoring and management sub-program fit into the overall strategic plan and business plan of the Program.'[21] The monitoring framework consisted of three streams. The first two relate specifically to Tasmanian devils and disease epidemiology and the third stream addresses the ecological implications resulting from a loss of Tasmanian devils from the landscape.

Notwithstanding the implementation of the above strategies for the conservation of the Tasmanian devil forestry practices in Tasmania continue to threaten the devils' survival as outlined in the following sections.

---

[19] The Devil Island Project, Devil Island Project Group Inc. Available at: http://devilislandproject.org/the-tasmanian-devil/devil-facial-tumour-disease/ last accessed 14 April 2013

[20] Save the Tasmanian Devil Program, 2010, Communication Strategy, Stage 3: 2008-2013, Department of Primary Industries, Parks, Water and Environment. Available at: http://www.tassiedevil.com.au/tasdevil.nsf/file/82C18864F5819337CA2576CB0011569B/$file/STDP_Communication_Strategy_180210.pdf last accessed 11 August 2013

[21] Save the Tasmanian Devil Program Monitoring Strategy, 2011. Available at: http://www.tassiedevil.com.au/tasdevil.nsf/downloads/82C18864F5819337CA2576CB0011569B/$file/STDP_Monitoring_Strategy_2011.pdf last accessed 9 August 2012

10.4 Lack of protection for Tasmanian devils in forestry practices in Tasmania

Under the Tasmanian *Regional Forest Agreement* (RFA) there is an exemption for forestry practices from the provisions of the *EPBC Act* but there remain conditions for the protection of endangered species under the *CAR* system. In plantation forestry operations Tasmanian devils and their habitat are most likely to be impacted in the initial stages of logging and clearing of native forests and at harvesting. But risks also exist during the growing and maintenance of plantation trees due to a pesticide regime to deter competition and predation.

The *Forestry Act 1920* and the *Forest Practices Act 1985* are the two principal Acts governing forestry operations in Tasmania. The *Forestry Act 1920* established the forestry corporation Forestry Tasmania, which controls forestry practices in State forests. The *Forest Practices Act 1985* regulates all forestry practices on both public and private land. The Forestry Practices Authority (FPA) regulates forestry operations in Tasmania and its functions include:

- establishing forests (including regeneration of native forests);
- growing or harvesting timber (including plantations);
- clearing trees (for any purpose);
- clearing and converting threatened native vegetation communities;
- harvesting tree ferns; and
- works associated with growing, harvesting or clearing trees, such as constructing roads and operating quarries.

The FPA requires a Forestry Practices Plan (FPP), site-specific operational plans, to be developed before logging commences in a designated coupe identifying any possible impediments. They describe measures for the protection of soils, water and natural and cultural values. FPPs must be certified before activities take place and prepared in accordance with the *Forest Practices Code (FPC)*. The *Code* provides for 'reasonable

protection' of areas subject to forestry practices. It is also integral to developing and managing forest plantations.[22] Its goal is 'sustainable management of Crown and private forests with due care for the environment …'.[23] There are however only general principles for fauna conservation with a basic approach to be implemented during the preparation of a FPP. According to the *Code* the proposed operational area will be assessed to determine:

- the known occurrence and potential habitat for threatened species;
- the presence or requirements for wildlife habitat strips;
- the requirements for wildlife habitat clumps;
- the presence of or requirements for special management zones for fauna.

Plans are to be prepared in consultation with statutory authorities such as the Environmental Protection Authority (EPA) Division and the Threatened Species Unit of the DPIPWE. There are, however, currently no prescriptions under these Plans to take into account Tasmanian devil habitat or maternal dens. Meanwhile, the conservation group *Still Wild Still Threatened* claims devil dens in new logging coups have been bulldozed.[24] According to Peter McQuillan 'it is claimed that because devils are wide-ranging animals it is inappropriate to declare critical habitat for them' and that 'no special effort is made to identify and protect devil den sites in Forest Practice Plans'.[25] But in his view 'secure denning sites for Tasmanian devils are a relatively scarce resource and should be declared critical habitat in order to protect them'.[26]

---

[22] Sadanandan Nambiar EK, Philip J, Smethurst R, Raison J, House APN & Moggridge B, 2012, *Assessment of Code of Practice for Plantation Forestry: Tasmania*, Australian Government Department of Agriculture, Fisheries and Forestry, Canberra.
[23] Forest Practices Board, 2000, *Forest Practices Code*, Forest Practices Board, Hobart, Tasmania, p 1
[24] Still Wild Still Threatened, Baby devil found in area threatened by logging. Available at: http://www.stillwildstillthreatened.org/node?page=3 last accessed 25 November 2013
[25] McQuillan PB, 2012, IVG Forest Conservation Report 9A, Report to Professor Jonathan West, Chair of the Independent Verification Group. Available at: http://www.parliament.tas.gov.au/ctee/Council/Submissions/ET%202.36.pdf last accessed 25 November 2013, p 29
[26] ibid.

Provisions are made under a Forest Practices Tribunal (FPT) to hear objections related to a limited range of forest practices disputes. The FPT has been incorporated into the Registry of the Resource Management and Planning Appeal Tribunal. This Tribunal consists of three members: a lawyer and two members with experience in harvesting, road construction and forest management. If an appeal is likely to raise questions about threatened species the Tribunal must include a person with a conservation science background (nominated by the Minister).

FPA specialists undertake research, often in collaboration with other researchers including university students, in order to develop new planning tools and management prescriptions. The scientific knowledge acquired is said to be essential for underpinning and improving the *Forestry Practices Code*. A specific FPA Biodiversity Program exists that conducts research primarily on threatened or priority listed flora and fauna and associated habitats. The main research areas currently undertaken are directed at 'distribution, ecology and impacts of forestry practices on flora and fauna species of high conservation significance, and their habitats'.[27] The Tasmanian devil is not included in this program.[28]

In 2011 FPA produced a scientific report entitled *Developing a framework for the conservation of habitat of RFA priority species – background report 3. A report on the on-ground implementation of current forest management prescriptions for the*

[27] Forest Practices Authority, Biodiversity research and monitoring. Available at: http://www.fpa.tas.gov.au/research_and_monitoring/biodiversity_program_research_and_monitoring last accessed 15 August 2012

[28] Forest Practices Authority, FPA Biodiversity program. Available at: http://www.fpa.tas.gov.au/research_and_monitoring/biodiversity_program_research_and_monitoring#bio res1 last accessed 15 August 2012

conservation of RFA priority species.[29] Part of this report was *Milestone 20* described as a '[m]idterm progress report detailing the on ground implementation of current forest management prescriptions (landscape and coupe level) to protect and assist recovery of RFA priority species in Tasmania.'[30] The only species listed for monitoring projects were the Swift parrot, Simson's stag beetle and the Ben Lomond leek-orchid.

The Swift parrot Recovery Plan was adopted in 2001 with an emphasis on the need to 'identify and protect key habitats and sites; the implementation of management strategies to protect breeding-habitat; and the maintenance or enhancement of existing habitat.' [31] However, established plantations are not currently subject to the recommendations in the *Swift parrot decision tree*[32], unless a plantation harvest operation involves the harvesting of forest remnants within the plantation.

The Tasmanian devil is not listed on the RFA priority species list and no recovery plan is in place. Therefore it is therefore difficult to know how the FPP can take into consideration the needs for protection of the Tasmanian devil habitat. The *Code's* basic approach is to develop an agreed procedure with endorsed management prescriptions for protection of a threatened species through consultation between landowners, Forest Practices Officers and specialists within the FPA and DPIPWE. It would appear that this process has not taken place.

---

[29] Chuter AE & Munks SA, 2011, *Developing a framework for the conservation of habitat of RFA priority species – background report 3. A report on the on-ground implementation of current forest management prescriptions for the conservation of RFA priority species,* Forest Practices Authority, Hobart

[30] ibid, p 1

[31] Chuter AE & Munks SA, 2011, *Developing a framework for the conservation of habitat of RFA priority species – background report 2 A review of the approach to the conservation of RFA priority species in areas covered by the Tasmanian forest practices system* Forest Practices Authority, Hobart, p 62

[32] Swift parrot decision tree is a guide used to make decisions on habitat management of the Swift parrot in areas covered by the Forest Practices System.

Meanwhile, in many rural areas of Tasmania, forestry is a 'permitted use' requiring Councils to issue a permit for forest practices but if forest practices are listed as a 'discretionary use' a permit is required.[33] Private land can also be declared a Private Timber Reserve (PTR) for the purposes of establishing plantation forests, harvesting timber and compatible activities through the FPA. PTRs are granted with respect to certain criteria being complied with, including land suitability, no land occupier, neighbor or owner is disadvantaged and it is not contrary to public interest. Objections to the issue of a PTR can be made to the Forest Practices Tribunal. If an application is refused, based on natural or cultural impacts, the FPA may require entry into a 'conservation covenant' to protect those values and may provide compensation for any loss of property value. However, participation in land conservancy for the protection of natural landscapes and native species is according to the Tasmanian Land Conservancy wholly voluntary.[34]

The forestry practices in plantation forests, under a self-regulatory system, provide no protection for endangered species habitat, including the Tasmanian devil. This situation is further compounded when DFTD-free areas of Tasmania are threatened by mining activities.

10.5 Lack of protection for Tasmanian devils in the Tarkine

The proposal to increase mining in the north west of the state, in an area known as the Tarkine, where the last remaining DFTD-free devils are located is a further threat to the long-term survival of the Tasmanian devil. Despite a critical need to protect this habitat for the future of the Tasmanian devils there have so far been threats to construct a

[33] Anonymous Contributors, 2013, Ch 8, *The Environmental Law Handbook*. Available at: http://www.edohandbook.org/doku.php?id=ch8&rev=1379475675 last accessed 15 August 2012
[34] Tasmanian Land Conservancy, Protected Areas on Private Land. Available at: http://www.tasland.org.au/majorprogrammes/papl last accessed 27 November 2013

tourist/forestry road and more recently applications for enlarging existing mines and proposals for new open-cut mining leases. A number of environmental reasons exist for preserving this unique area. In my view, the protection of the Tasmanian devil deserves the highest priority. To continue to destroy and degrade the devils' habitat reflects a serious lack of concern for its long-term future by the Tasmanian government and the forestry and mining industries.

10.5.1 The Tarkine road

The proposal for a tourist road was first initiated by the former Tasmanian premier Paul Lennon to be built at a cost of $23 million by Forestry Tasmania. Ken Jeffreys of Forestry Tasmania in an interview with Felicity Ogilvie supported the road because 'it makes sense' - economic sense with 1,600 jobs and $70 million to be created by the construction of the road.[35] The road however was not popular with local residents in the north-west with only one out of seven councils supporting its construction. Many tourism operators were also against the road as they said it would detract from the area, whilst conservations said it would open up the area for further logging of native forests.

The controversial proposal to build a tourist road was however stalled by Federal Environment Minister Peter Garrett's decision to use his emergency powers to bring forward its assessment for registration on the National Heritage List. This decision followed the referral of the matter as a controlled action under the *EPBC Act 1999* to the Minister for assessment. The result was that the proposed road was subject to two Federal government decisions. The listing of the Tarkine on the National Heritage List according to Garrett "does not in itself prevent the road being built and does not amount

[35] Ogilvie F, Controversial Tarkine road to be assessed by Federal Government, *ABC PM Program*, 20 March, 2009

to a decision on the road".[36] But it meant that the then Bartlett government needed to prove that the proposed road would not detrimentally affect the Tarkine natural heritage values. Garrett assured the Tasmanian government that the assessments would not interfere with forestry operation or possible mining in the Tarkine. The Tasmanian government remained confident that the road, to be constructed by the Department of Infrastructure, Energy and Resources, would be approved by the Minister.

The proposal to build the Tarkine Road was referred to the then Environment Minister under the *EPBC Act 1999*. The Referral included a survey of flora and fauna conducted by Barker Ecosystem Services for the state government.[37] Scott Jordan from the Tarkine National Coalition said that "crucial information regarding the projected numbers of devil roadkill, on the number of dens in the area, the impact on spotted tail quoll, on wedge tailed eagle and on threatened botanical species in the area, are just missing from the report". A total of 70 public submissions were received, however they were not made public.

Twenty-six scientists signed an open letter expressing their concern at the construction of the Tarkine Road. The Tarkine is home to 24 species of native land mammals, more than two-thirds of Tasmania's native mammal species. Devils are known to use roads to travel in search of food and as scavengers are especially attracted to roadkill. Other native species likely impacted by the construction of the road include the critically endangered orange-bellied parrot and the endangered wedge-tailed eagle and swift parrot.

---

[36] Neales S, 2009, Protection move for Tarkine, *The Mercury*. Available at: http://www.themercury.com.au/article/2009/12/11/115161_tasmania-news.html 12 December 2009

[37] Australian Government, Department of the Environment, Water, Heritage and the Arts, Referral of Proposed action, *Environment Protection and Biodiversity Conservation Act 1999*, 001 Referral of proposed action vJAN09

The *Save the Devil Program* research scientists also claimed that the construction of the road would have a negative impact on the devils and facilitate the spread of the devil cancer. This is confirmed by the state government's own claim that "The Tarkine area has a large number of Tasmanian devils that have not been affected by the Devil Facial Tumour Disease so the department will undertake a study into any possible spread of the disease".[38] However, they proposed that the risk to the Tasmanian devil posed by Tarkine road is minimal. Meanwhile, the Greens and the Tasmanian state Liberals oppose the road project in line with the scientists' view.

Evidence for the danger posed to the Tasmanian devils by the construction of the road was also provided as part of the CRC for Sustainable Tourism. A paper was produced titled *Reducing the incidence of wildlife roadkill: improving the visitor experience in Tasmania* documenting the devastating effects new roads and widening of roads had on Tasmanian devils.[39] Although targeting the experience of tourist and wildlife business operators two studies showed negative impacts on native species including devils following the widening and sealing of roads. The first case was the Woolnorth Road in far north-west Tasmania which resulted in the disruption of a Tasmanian devil viewing/filming business due to dramatically reduced numbers of Tasmanian devils. The second case occurred along the access road to Cradle Mountain, part of the World Wilderness Heritage Area, where Eastern quoll (*Dasyurus viverrinus*) population was eliminated and the Tasmanian devil population halved. Tasmanian devils and quolls are

[38] Carter P, 2009, Garrett to assess Tarkine road plan, *Sydney Morning Herald*, 13 November 2009, accessed 14.11.2009, http://news.smh.com.au/breaking-news-national/garrett-to-assess-tarkine-road-plan-20091113-ie8w.html

[39] Magnus Z, Kriwoken LK, Mooney NJ & Jones ME, 2004, *Reducing the incidence of wildlife roadkill: improving the visitor experience in Tasmania*, Cooperative Research Centre for Sustainable Tourism, Hobart Tasmania

often attracted to the road to scavenge on carcasses of herbivores, which are attracted to the roadsides by the green grass growing due to run-off from the road.[40]

The proposed new road development in the Tarkine wilderness constitutes unacceptable and irreversible environmental degradation threatening the survival of endangered species, including the Tasmanian devil. A more serious threat to the area than a tourist/forestry road would however be the proposal to extend mining and approve further open-cut mines.

10.5.2 Mining in the Tarkine

Tasmania is one of the most highly and diversely mineralized areas in the world and mining and mineral processing make major contributions to the Tasmanian economy. The regulatory regime in Tasmania is supportive of mining through the *Mineral Resources Development Act 1995* and the government actively seeks development of new projects. A West Australian mining company, Venture Minerals, has been granted a lease to mine for hematite at Livingstone in the Meredith Ranges Regional Reserve near Tullah.[41] Venture Minerals is also developing a hematite prospect at nearby Riley Creek as well as the Mount Lindsay tin and tungsten project. Shree Minerals also applied for an open cut iron ore mine at Nelson Bay River. In total ten new mines are proposed for development in the area over the next 3-5 years from a total of 56 current exploration licences granted over the Tarkine.[42] The scale of the mining operations is shown in Figure 10:1 below.

---

[40] ibid, p 20
[41] *ABC News*, Green light for Tarkine mining lease, 29 May 2012. Available at: http://www.abc.net.au/news/2012-05-28/new-mining-lease-for-tarkine/4038146 last accessed 8 August 2012
[42] Tarkine National Coalition, Mining. Available at: http://tarkine.org/mining/ last accessed 8 August 2012

Figure 10:1 Savage River iron ore mine[43]

According to Grange Resources Annual Report for the year ended 31 December 2012 the Savage River mine is set to continue operations until 2030 with potential to further extend the mine life.[44] Confirmation of Tasmanian devils in the vicinity of the Savage River mine was provided in Grange Resources Tasmanian Pty Ltd Notice of Intent for the construction of a South deposit tailings storage facility in 2012.[45] In a survey undertaken by North Barker Ecosystem Services in March 2012 it was noted that '[t]he Tasmanian devil is likely to be present on site based on scats observed during the survey'.[46] It was also acknowledged that the construction of the tailings storage facility would result in the loss of Tasmanian devil habitat, potentially including dens. In 2012

[43] Engines serving it up at Savage River mine, *The Australian Mining Review*. Available at: http://australianminingreview.com.au/wp-content/uploads/2012/07/l1.jpg last accessed 3 September 2013
[44] Grange Resources Limited, 2012, 2011 Annual Report. Available at: http://www.grangeresources.com.au/clients/grange/downloads/item151/grange_resources_annual_report_year_ended_31_december_2012_-_3rd_april_2012_updated.pdf last accessed 8 February 2013
[45] Caloundra Environmental Pty Ltd. & Grange Resources Tasmanian Pty Ltd, 2012, *Notice of Intent Construction of South Deposit Tailings Storage Facility*. Available at: http://epa.tas.gov.au/documents/grange%20resources%20savage%20river%20south%20deposit%20tsf%20notice%20of%20intent.pdf last accessed 8 February 2013
[46] ibid, p 13

Venture Minerals funded a University of Tasmania (UTAS) scholarship, to undertake an honours project to 'demystify devil dens'.[47] Dr Menna Jones of UTAS and *Save the Tasmanian Devil Program* will be the supervisor. According to Rebecca Cuthill of the Save the Tasmanian Devil Appeal '[t]hrough the generous support provided by Venture Minerals we are one step closer to saving our iconic devil from extinction'.[48]

Environmentalist, conservationist and community members are however strongly opposed to further mining in the Tarkine and the protection of the Tasmanian devils as illustrated in Figure 10:2 below.

Figure 10:2 Protest banner in the Tarkine[49]

10.6 Public participation and lay knowledge

Decision makers in Tasmania, although willing to implement the precautionary principle in the eradication of foxes, have failed to do so to protect the Tasmanian devils under the *EPBC Act*. In order to have a more inclusive and transparent policy outcome

[47] Media Release, 19 April 2012, *'Scholarship recipient to demystify devil dens'* University of Tasmania, Hobart
[48] ibid.
[49] Source: Image provided by protestors.

it may be appropriate to include greater public participation in the decision making process. By also incorporating lay knowledge a better appreciation of the situation at the local level, as occurred in the compilation of the *Scammell Report,* may also be of benefit. There exists a considerable body of knowledge surrounding the concepts of public participation and lay knowledge, which is beyond the scope of this thesis. Possible further studies incorporating these concepts could investigate their role in overcoming undone science for political reasons and ensure that the precautionary principle is implemented in the future.

10.7 Conclusion

In 2009 the Tasmanian devil was listed as endangered under both the Tasmanian and Federal threatened species legislation but still there have been no practical steps implemented in Tasmania to protect either the devil or its habitat. The DPIPWE has established the STDP and implemented strategies, which focus on the conservation of the devil and studies into the deadly cancer DFTD. These strategies include monitoring and management of the disease and the establishment of an insurance population in captivity. But several important criteria under the legislation, including declaring and mapping critical habitat, implementing recovery and threat abatement plans and land management plans, have not been undertaken. This lack of commitment to protecting the devil and its habitat results in exposure to logging and plantation development plans with no prescriptions for the devil or its habitat to be protected.

The Tasmanian government's commitment to save the Tasmanian devil appears merely symbolic given its failure to comprehensively study all avenues of research in relation to DFTD, as described in the previous chapters, coupled with the continued lack of protection for the devils' habitat. Thus it would appear from the evidence given in this

chapter that for the Tasmanian and Federal governments the enabling of the forestry and mining industries in Tasmania is a much higher priority than the protection of the Tasmanian devil.

Conclusion

The Tasmanian devil is a carnivorous marsupial, which belongs to a distinctive group of pouched mammals that arrived in Australia when it was part of Gondwana. It is unique both because it is found nowhere in the world except Tasmania and because it is facing extinction from a deadly cancer said to be contagious. The cancer, termed Devil Facial Tumour Disease (DFTD), was first observed in 1996 in the north east of the state of Tasmania, and has resulted in a loss of over 80% of devil populations in some locations. In 2003 the then Premier of Tasmania, Jim Bacon, declared every effort would be made to prevent the Tasmanian devil going the way of the Tasmanian tiger, which became extinct in the 1930s. A meeting of scientists was held, without public access, to devise a program of scientific studies. Research into the cancer was established through the Save the Tasmanian Devil Program (STDP) with funding from the Australian and Tasmanian governments and public contributions to the Department of Primary Industries, Parks, Water and Environment (DPIPWE) Save the Tasmanian Devil Fund.

Using David Hess's concept of alternative pathways and undone science I have analysed the published scientific articles into the devil cancer. Hess's analyses found that political and economic elites, such as governments, foundations and private corporations, use their funding power to direct scientific studies according to their interests, which results in areas of enquiry being neglected.[1] His studies showed that conventional energy sources and methods of food production receive more funding and hence are supported by more studies than their alternatives, renewables and organic farming. I have found that scientific studies concerning DFTD have been directed

[1] Hess DJ, 2007, *Alternative pathways in science and industry, Activism, Innovation, and the Environment in an Era of Globalization*, MIT Press, Cambridge, Massachusetts, London, England.

along a pathway that has avoided, abandoned or left undone research findings that may in some way link commercial industry practices to the devil cancer. If the devil cancer is linked to plantation forests, as suggested by the *Scammell Report,* it would also potentially create a backlash against plantations as the solution to the logging of Tasmania's native and old-growth forests. I have found no practical reasons, such as lack of technical facilities or theoretical frameworks, to explain the lack of studies.

The research pathway selected in support of the transmissibility of the devil cancer produced several findings that have since been disproved. These include the claim of similarities between the dog and the devil cancers, the proposal that west coast devils would be resistant to the cancer, and the claim that devils' lack of genetic diversity was the reason for the spread of the cancer. Claims proposed regarding the spread of the cancer have also oscillated between being dependent on density (the number of devils in the population) or frequency (the number of times devils come into contact with a diseased devil). However, testing of the transmission hypothesis was abandoned. More importantly for showing the role of undone science, studies into a plausible competing hypothesis, that chemicals used in plantation forestry may be linked to the cancer, were abandoned following an initial pilot study.

Furthermore, an analysis of the role of the forestry industry and its support from both Federal and Tasmanian governments reveals close ties between government agencies, chemical companies and the forestry industry as well as questionable practices.

Tasmanian devil cancer research pathways and undone science

The concept of undone science as a tool for analyzing scientific research has proved valuable in highlighting how funding moves studies along a prescribed pathway. In my

investigation, undone science has been used as a probe to analyse whether or not the research into DFTD has been abandoned or neglected for practical or political reasons. In this analysis I have engaged with the scientific research as an individual seeking to understand how certain research pathways are avoided. This thesis contributes to a growing body of research informed by the concept of undone science. Other comprehensive studies have been undertaken by David Hess in Can Bacteria Cause Cancer?[2] And Robert Proctor in Cancer Wars: How Politics Shapes What we Know and Don't Know About Cancer.[3] Both studies engage with the controversial nature of the causes of cancer. The contagious nature of the Tasmanian devil cancer is, however, not contested by scientists. Also, unlike other studies of undone science by David Hess[4] and Frickel et al[5] there are no social groups pushing for research to be undertaken. In this non-controversy, as a social analyst, I have therefore systematically examined the published literature identify either areas of research that have been pursued or those that have been initiated, abandoned and left undone. Whilst this approach may have limitations in revealing why certain areas of study are abandoned or ignored it demonstrates its value by identifying politically motivated actions that lie behind the undone science. There is another approach, which would add an extra dimension to the concept - to analyse scientific research from the perspective of activists. Often activists are those seeking but not obtaining answers to questions about environmental problems. However, there were no dissenting scientists or questioning activists on whose behalf I could approach the science into DFTD. Hence, I proceeded as a non-scientist

---

[2] Hess, DJ, 1997, *Can Bacteria Cause Cancer? Alternative Medicine Confronts Big Science*, New York University Press, New York and London

[3] Proctor RN, 1995, *Cancer Wars: How Politics Shapes What We Know and Don't Know About Cancer,* BasicBooks, New York

[4] Hess DJ, 2009, The Potentials and Limitations of Civil Society Research: Getting Undone Science Done, *Sociological Inquiry*, Vol 79(3), pp 306-327

[5] Frickel S, Gibbon S, Howard J, Kempner J, Ottinger G & Hess D, 2009, Undone Science: Charting Social Movement and Civil Society Challenges to Research Agenda Setting, *Science, Technology, & Human Values*, Vol 35(4), pp 444-473

investigating the scientific studies to determine whether a competing hypothesis, that toxins in the environment had contributed to the cancer, had been tested.

The research pathway chosen by the STDP scientists centred around the hypothesis that the devil cancer was contagious and spread by biting. Anne Maree Pearse and Kate Swift made this hypothesis public when they published an article in the Brief Communications section of the prestigious journal *Nature* in 2006. Their claim that a chromosomal anomaly existed in the cells of one devil but was absent in that devil's tumour cells, a claim that underpinned the hypothesis, has never been tested. A transmission study was commenced, resulting in the publication of an abstract in a conference paper, but no further studies have been published. It was proposed that the dog sexual transmissible tumour was a precedent for the devil cancer but, as I have shown in Chapter 4 in a comparison between the research programs, the devil cancer lacks fundamental similarities with the sexually transmitted dog tumour. The claim of stability of the devil cancer cells, unlike the instability in normal cancer cells, was the basis of the transmission hypothesis, but this has since been contradicted by Pearse et al in a paper published in 2012.[6] In this paper it is claimed DFTD cells are regarded as unstable by comparison with the much older transmissible cancer, canine transmissible venereal tumor (CTVT).[7] Pearse and Swift noted it would be necessary to DNA-fingerprint tumours to reveal the disease's toxicology, progression and epidemiology. PhD student Hannah Bender was to undertake these studies at the Australian National University (ANU) in Canberra in 2007 as part of a project between the ANU and the DPIPWE. The DNA fingerprinting studies were however not undertaken until 2012.

---

[6] Pearse AM, Swift K, Hodson P, Hua B, McCallum H, Pyecroft S, Taylor R, Eldridge MDB & Belov K, 2012, Evolution in a transmissible cancer: a study of the chromosomal changes in devil facial tumor (DFT) as it spreads through the wild Tasmanian devil population, *Cancer Genetics*, Vol 205, pp 101-112

[7] ibid, p 101

Research studies were also undertaken to investigate the anomaly in the devil immune system – how did the tumour become established in the devil when it had a competent immune system? This research also investigated the proposed resistance in the DFTD-free west coast devils and a test for a vaccine. However, the claim that west-coast devils were resistant to the cancer was subsequently disproved. The immunological studies, on the other hand, remain inconclusive. They have so far not accounted for the lack of an immune response to DFTD, observed by Richmond Loh in his initial study, if as claimed the devils have a competent immune system. Other research undertaken by the STDP team investigated the population dynamics of the devil and conservation issues.

The need to test devil tissue and fat for the presence of chemicals detected in the environment was proposed in the initial DPIPWE Report. Toxicity studies were at first delayed. Then, following a pilot study, which showed devil tissue contained high levels of PBBs (flame retardants) toxicity studies were abandoned. This was contrary to the earlier reports, and contrary to the advice of two scientific reviewers of the pilot study results who agreed further testing was needed. An analysis of the research published revealed that only one paper, that by Vetter *el al* published in the journal *Rapid Communications in Mass Spectrometry,* related to the alternative hypothesis.

The scientific research pathway chosen for investigation into the devil cancer appears to have been directed away from the possibility of implicating the toxins used in the plantation forests, either in the initiation or progression of the cancer or their role in immune suppression. Research independent of the supervision of the STDP was limited

and even for this research the DPIPWE supplied all devil research material. No practical reasons exist for the lack of studies into the toxicology of the disease.

Early in the research, it was suggested and confirmed by a GIS report, that the spread of the cancer may have been due to an artifact of reporting the disease. It is therefore possible that DFTD is a cancer cluster in the devil population, similar to those occurring in other wildlife species - the Beluga whales in the St Lawrence River Estuary, the California Sea Lions in the San Francisco Bay and the Green Sea Turtles in Moreton Bay. But as I have demonstrated, toxicology studies into these cancers, like the devil cancer, remain undone except for two pilot studies. It is acknowledged that the sheer complexity of the environment means a vast number of variables could be contributing factors in the initiation and promotion of cancer, but this should not inhibit toxicology studies: it should in fact be the driver for more. Some of these factors as mentioned in chapter 7 are only now being recognised, such as the effects of endocrine disrupters and epigenetics. The uncertain but plausible scientific evidence that the endocrine disrupter atrazine may play a role in all four wildlife cancers suggests that the precautionary principle should be adopted to mitigate the possibly irreversible harm and prompt further toxicology studies. These scientific studies could then extend the boundaries of knowledge in relation to the harm caused by chemicals such as atrazine to wildlife and probably human populations.

Australia is a signatory to the United Nations *Convention on Biological Diversity (CBD)*, which is legislated through the *Environment Protection and Biodiversity Conservation Act 1999 (EPBC Act),* underpinned by the precautionary principle. It requires the protection of endangered species through the establishment of a recovery

plan. The Tasmanian devil was listed as vulnerable in 2006 and reclassified as endangered in 2009 prompting the need for such a plan. A draft recovery plan has been drafted by the DPIPWE and public submissions have been sought but it remains to be adopted. The Plan however also fails to address the possible role of environmental toxins in a list of threats to the recovery of the devil. Meanwhile, the DPIPWE has in place strategies for the monitoring of DFTD, the conservation of the devil through an insurance population strategy, a business strategy and a communications strategy. My analysis of the role of government agencies, forestry and chemical industries in plantation forestry practices attempts to explain why scientific findings that might prove negative to their interests are avoided.

Forestry in Tasmania

The forestry industry in Tasmania is a key economic resource and both the government and the industry enjoy reciprocal benefits. The forestry industry is enabled through government support and industry funding supports political parties at elections. In Tasmania, as elsewhere in Australia, there has been considerable controversy over the logging of native and old-growth forests, which is ongoing, but plantation forestry is seen by some as a potential substitute and solution. The industry was preoccupied until recently by the Gunns Limited proposal to build a chlorine free pulp mill, aimed at adding value to the logging and woodchipping of plantations.

In Tasmania the introduction of the *Plantations 2020 Vision* and the proposed increase in plantation estate had the potential to be problematic because of the reliance on pesticides and poisons to control predators and weeds. As early as 1984 studies on atrazine demonstrated its potential to contaminate streams. Ongoing reporting of various forms of pesticide contamination of surface water such as rivers, streams and

tanks eventually prompted the DPIPWE to initiate a monitoring regime. Under the *Forestry Practices Code* monitoring of contamination was undertaken by the forestry industry in a self-regulatory process. Over the years little action has been taken to properly address these issues. An abrupt change came about in 2003 with a helicopter crash and the spill of its payload of chemicals in the George Bay catchment, near St Helens on the east coast of Tasmania.

The resulting death of commercial oysters and native species prompted the local oyster growers to undertake, with local activist Dr Alison Bleaney and ecologist Dr Marcus Scammell, their own enquiry. The outcome was the *Scammell Report*, which found a correlation in time and space between chemical use in plantations, the ongoing oyster problems and the devil disease. It called for the adoption of the precautionary principle to halt aerial spraying of chemicals until further scientific studies could be undertaken. The response to the report by the Tasmanian government and the chemical industry was scathing, with the DPIPWE consultant declaring the report a manifesto based on unsound science. It was this report and the subsequent reactions that prompted my interest in the Tasmanian devil cancer.

Undue influence?

There are no practical reasons such as lack of funding, technology or theories preventing the further investigation of the possible role of chemicals in the devil cancer. There is evidence however that Syngenta, the manufacturer of atrazine, attempted to influence the US EPA's decision on atrazine. Likewise the Australian regulator, the Australian Pesticides and Veterinary Medicines Authority (APVMA) has been slow to address concerns in regard to further restricting the chemical. In Tasmania the

DPIPWE's role in overseeing both the use of chemicals in plantations and the Save the Devil Program constitutes a conflict of interest.

Following ten years of research into this deadly cancer a likely reason why DFTD still threatens the extinction of the Tasmanian devil is a deficit of relevant knowledge, a consequence of scientific studies avoided for political reasons.

Broader implications

My investigation into the role of undone science in the case study of the Tasmanian devil cancer was at times hampered by my limited access to the STDP scientists and my lack of scientific authority. My role as a social scientist trained in critical analysis has enabled me to overcome many of these shortcomings. By using the concept of undone science I have interrogated the broader social and political forces impacting externally and internally on the DFTD scientific community. I have found that important studies into a competing hypothesis that environmental toxins played a role in the cancer were abandoned. I have also been able to show that the elite in Tasmania choose to fund a particular pathway, the allograft theory, to investigate the devil cancer and that scientific inquiry into the transmission of the cancer was also abandoned.

To overcome deficits of knowledge due to political reasons, more in-depth analyses of the wildlife cancers in the Beluga whales, the San Franciscan seal lions and the Green Sea turtles, touched on in this thesis, should be undertaken. Others areas may also warrant further investigation.

The role of government in the Tasmanian devil cancer should be to develop public policy and to act, to make decisions in order to prevent the extinction of a species. The

Tasmanian devil is listed under the *EPBC Act* as endangered, facing the threat of extinction. The *EPBC Act* is informed by the precautionary principle, which is a tool for decision makers to act to mitigate harm. In order to trigger the precautionary principle there must first be a body of scientific evidence supporting an action, even if that knowledge is confounded by uncertainty, and a commitment to further scientific research. But a lack of certainty or research should not be used as a reason for delaying action. The precautionary principle presently does not make allowance for undone science or science that is abandoned. Undone science as a form of ignorance or non-knowledge undermines the application of the precautionary principle, which relies on research findings about potential risks, even if they are uncertain or contested. A fruitful field of future inquiry is to investigate the role of undone science in the scientific uncertainty weighting on regulators and decision makers when considering the adoption of the precautionary principle. This could include the failure to fund further studies required by the precautionary principle, which could be probed to determine if the reasons are practical or political.

Recommendations

Based on the findings of this thesis, I recommend:

1. Completion of the transmission studies in both the laboratory and field to determine the mechanism of action for the establishment of the cancer in the new devil host.
2. Comprehensive toxicology studies to determine if there is a link between the chemicals, in particular atrazine, used in plantation forestry in Tasmania and the devil cancer.
3. Comprehensive studies to determine if these same chemicals interfere with the normal functioning of the devil immune system.

4. Comprehensive studies to investigate if the instability in the DFTD tumour cells is linked to toxins or poisons used in plantation forestry. These studies were first listed for undertaking in the initial DPIPWE Progress Report but remained undone because it was claimed that the DFTD tumour cells, like the dog transmissible cancer, were stable. This claim has since been acknowledged as false.
5. Regulatory risk assessment needs to incorporate chemicals such as endocrine disrupters, which operate at non-toxic levels and at particular times in the development of an organism, into their testing. Currently chemicals such as atrazine, which is classified as non-toxic, is not assessed as a hazard to humans or the environment.
6. Funding is needed for independent studies including toxicology (the effects of pesticides on native species), immunology, and a broader scope for detecting chemicals in Tasmanian waterways.
7. The Federal government should play a more decisive role in the application of the *EPBC Act* in relation to species listed as endangered in order to avoid extinction, including the drafting of a new Recovery Plan for the Tasmanian devil.
8. The chemical regulator, the APVMA, and the state regulatory bodies should establish more community consultative committees with powers to investigate breeches of the codes of practice and these should be developed to meet international standards of performance for sustainability.
9. A public register of chemicals used, when, where and by whom, should be established in order to increase accountability and to help trace non-point sources of contamination.

10. The testing of chemicals for regulatory purposes requires a shift in paradigm, from a focus on the toxicity of individual chemicals to the more relevant synergistic effects as well as non-toxic effects.
11. The precautionary principle under the *EPBC Act* should be implemented to mitigate further harm and the possible extinction of the Tasmanian devil, by further restricting or banning atrazine.
12. More public participation and inclusion of lay knowledge in environmental studies, such as undertaken in the *Scammell Report*, should be encouraged to increase the knowledge base particularly when dealing with local issues.

Postscript

The Save the Tasmanian Devil Program failed to secure federal government funding of $4 million over the next four years.[8] The US National Science Foundation will however spend $2.25 million to study DFTD as an Emerging Infectious Disease (EIDs).[9]

[8] *ABC News*, 2013, Greens claim funding decision will condemn Tasmanian Devils to extinction. Available at: http://www.abc.net.au/news/2013-08-30/the-federal-govt-accused-of-snubbing-devil-program/4924296?section=tas last accessed 30 August 2013

[9] Starr P, 2013, Feds devote $2.5 million to study Tasmanian Devil Facial Tumour disease, *CNS News*. Available at: http://cnsnews.com/news/article/feds-devote-225-million-study-tasmanian-devil-facial-tumor-disease last accessed 27 August 2013

Appendix A

COPY

Vol 439|2 February 2006 549

Transmission of devil facial-tumour disease

An uncanny similarity in the karyotype of these malignant tumours means that they could be infective.

The Tasmanian devil, a large carnivorous Australian marsupial, is under threat from a widespread fatal disease in which a malignant oral–facial tumour obstructs the animal's ability to feed1. Here we show that the chromosomes in these tumours have undergone a complex rearrangement that is identical for every animal studied. In light of this remarkable finding and of the known fighting behaviour of the devils2, we propose that the disease is transmitted by allograft, whereby an infectious celline is passed directly between the animals through bites they inflict on one another.

The cancer, known as devil facial-tumour disease, now affects devils (*Sarcophilus harrisii*) in more than half of Tasmania1. The growth of the tumours, which ulcerate and become friable, eventually causes the devils to starve. As the tumour cells are easily dislodged and because almost all bites from the devils' frequent fighting occur around the mouth2, we investigated whether the disease might be transmitted by allograft between animals. We studied tumours that included early neoplasms, huge primary cancers and secondary cancers. The cancers were sampled from animals throughout eastern Tasmania, Australia, over a 12-month period (for methods, see supplementary information).

The normal number of chromosomes in the devil is 14, including the XX or XY sex chromosomes (Fig. 1a). We found that the facial tumours contained only 13 chromosomes and that these were grossly abnormal (Fig. 1b). The number and appearance of the chromosomes (the karyotype) indicated that both sex chromosomes, both chromosomes 2 and one chromosome 6 were absent. There was also a deletion of the long arm of one chromosome 1, and four unidentified marker chromosomes were present. Most important, these anomalies were the same in the facial tumours from every animal (*n*_11).

These rearrangements are complex, but no intermediate stages were found between normal and tumour chromosomes, even in small primary cancers. In human cancers, there is generally a common breakpoint (first event)3, irrespective of whether the neoplasm is caused by viral insertion (as in Burkitt's lymphoma4) or arises spontaneously (as in Ewing's sarcoma3); complex rearrangements occur in solid tumours as a result of further clonal evolution5. However, the identical chromosomal rearrangements that we found in the facial tumours of each devil are too complex for a common breakpoint to have occurred3. Indeed, the rearrangements do not conform to any human model, particularly given the loss of sex chromosomes in all the tumours of devils of both sexes.

Further support for the allograft theory of disease transmission derives from the serendipitous observation of a pericentric inversion of chromosome 5 in the constitutional karyotype of one animal. This constitutional anomaly was found in all

cultures of that devil's normal tissues, but was not present in either of the chromosomes 5 in his facial-tumour cells, where it would have been found had the neoplasm arisen from his own tissue.

Cases of transmissible venereal sarcoma in dogs6 also show similar chromosomal defects among tumours, leading to the proposition that this sarcoma may develop from "a clone capable of a parasitic existence"7 — a description that also fits the features of the devil's facial tumour. We suggest that the devils' cancer (like the dogs') is infective and that the infective agent is a rogue cell line that initially evolved in a tumour of unknown origin.

Humans, too, can accidentally infect each other with cancer, through cell implantation in patients that have received organ transplants8; such cancers then develop according to their usual course9. Organ transplants are less likely to be rejected if the donor is a close relative who has a matching tissue type; by analogy, the low genetic diversity and high degree of kinship among devils10 might help to reduce their immune response to cancer cells implanted during biting. Although the devil's immune system is poorly understood, preliminary investigations indicate that there is little immune reaction between lymphocytes taken from devils from within and outside local populations (G.Woods, personal communication).

To obtain further insight into the transmission of the devil's facial-tumour disease, it will be necessary to DNA-fingerprint tumours and clarify their derivation by using whole-chromosome painting probes, as well as searching for oncogenes. This should reveal the disease's toxicology, progression and epidemiology.

A.-M. Pearse, K. Swift
Department of Primary Industries, Water and Environment. PO Box 46, Kings Meadows, Tasmania 7249, Australia
e-mail: anne-maree.pearse@dpiwe.tas.gov.au

1. Hawkins, C. E. *et al. Biol. Cons.* (submitted).
2. Hamede, R. Thesis, Univ. Tasmania (Hobart, 2004).
3. Mitelman, F. *Mutat. Res.* 462, 247–253 (2000).
4. Zech, L., Haglund, U., Nilsson, K. & Klein, G. *Int. J. Cancer* 17, 47–56 (1976).
5. Mitelman, F., Johansson, B., Mandahl, N. & Mertens, F. *Cancer Genet. Cytogenet.* 95, 1–8 (1997).
6. Das, U. & Das, A. K. *Vet. Res. Commun.* 24, 545–556 (2000).
7. Murray, M., James, Z. H. & Martin, W. B. *Res. Vet. Sci.* 10, 565–568 (1969).
8. Kauffman, H. M. & McBride, M. A. *Transplantation* 74, 358–362 (2002).
9. Forbes, G. B. *et al. J. Clin. Pathol.* 34, 109–115 (1981).
10. Jones, M. E., Paetkau, D., Geffen, E. & Moritz, C. *Mol. Ecol.* 13, 2197–2209 (2004).

Supplementary information accompanies this communication on *Nature*'s website.
Received 27 September 2005; accepted 17 January 2006.
Competing financial interests: declared none.
doi:10.1038/439549a

Appendix A

BRIEF COMMUNICATIONS

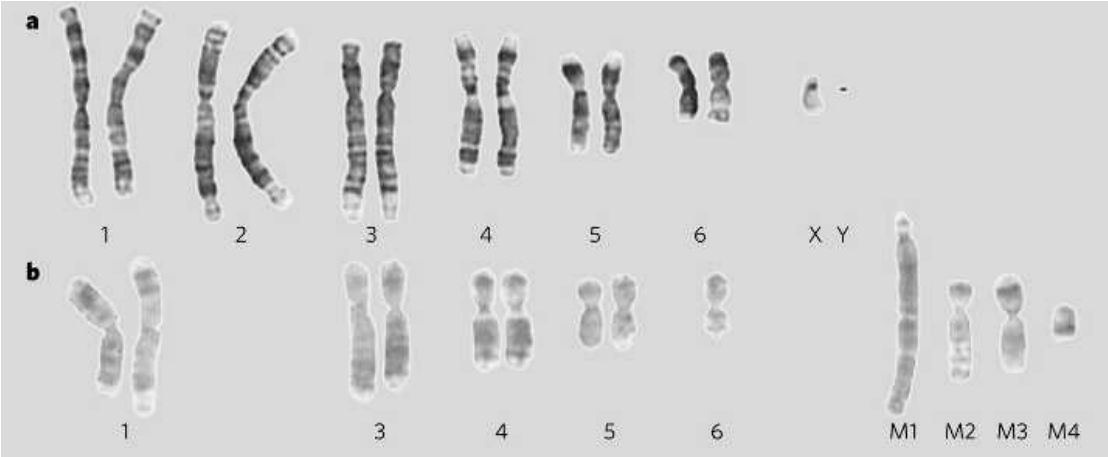


Figure 1 | **Chromosomes of facial tumours from Tasmanian devils. a,**Normal karyotype for a male Tasmanian devil (14 chromosomes, including XY). **b,**Karyotype of cancer cells found in each of the facial tumours of all 11 animals studied (13 chromosomes, with no sex chromosomes, no chromosome-2 pair and only one chromosome 6; the long arm of one chromosome 1 was deleted; four additional marker chromosomes were present (M1–M4).

Appendix B

Tasmanian devil publications associated with Devil Facial Tumour Disease to July 2011

AUSVET. 2005. Tasmanian devil facial tumour disease response. Technical Workshop: 29-31 August 2005. Final Report, Department of Primary Industries, Water, and Environment, Hobart (Tasmania).

Belov K. (2011) The role of the Major Histocompatibility Complex in the spread of contagious cancers. Mammalian Genome 22: 83-90

Bode, M., C. Hawkins, T. Rout, and B. Wintle. 2009. Efficiently locating conservation boundaries: Searching for the Tasmanian devil facial tumour disease front. Biological Conservation 142:1333-1339.

Bode, M. and B. Wintle. How to build an efficient conservation fence: Contributed paper. Conservation Biology 24:182-188.

Bradshaw, C. J. A. and B. W. Brook. 2005. Disease and the devil: density-dependent epidemiological processes explain historical population fluctuations in the Tasmanian devil. Ecography 28:181-190.

CBSG. 2008. Tasmanian Devil PHVA Final Report. Apple Valley, MN.

Church, J. S., D. J. Evans, and A. L. Woodhead. 2009. The characterization of Tasmanian devil Sarcophilius harrisii pelage fibres and their associated lipids. Acta Zoologica 90:380-389.

Czarny, N. A. and J. C. Rodger. Vitrification as a method for genome resource banking oocytes from the endangered Tasmanian devil (Sarcophilus harrisii). Cryobiology 60:322-325.

Fassati, A. and N. A. Mitchison. 2009. Testing the theory of immune selection in cancers that break the rules of transplantation. Cancer Immunology, Immunotherapy:1-9.

Hamede, R., J. Bashford, H. McCallum, and M. Jones. 2009. Contact networks in a wild Tasmanian devil (Sarcophilus harrisii) population: using social network analysis to reveal seasonal variability in social behaviour and its implications for transmission of devil facial tumour disease. Ecology Letters 12:1147-1157.

Hamede, R., H. I. McCallum, and M. E. Jones. 2008. Seasonal, demographic and density-related patterns of contact between Tasmanian devils: Implications for transmission of Devil Facial TumourDisease. Austral Ecology 33:614-622.

Hawkins, C. E., C. Baars, H. Hesterman, G. J. Hocking, M. E. Jones, B. Lazenby, D. Mann, N. Mooney, D. Pemberton, S. Pyecroft, M. Restani, and J. Wiersma. 2006. Emerging disease and population decline of an island endemic, the Tasmanian devil Sarcophilus harrisii. Biological Conservation 131:307-324.

Hesterman, H. and S. M. Jones. 2009. Longitudinal monitoring of plasma and fecal androgens in the Tasmanian devil (Sarcophilus harrisii) and the spotted-tailed quoll (Dasyurus maculatus). Animal Reproduction Science 112:334-346.

Hesterman, H., S. M. Jones, and F. Schwarzenberger. 2008. Pouch appearance is a reliable indicator of the reproductive status in the Tasmanian devil and the spotted-tailed quoll. Journal of Zoology 275:130-138.

Hesterman, H., S. M. Jones, and F. Schwarzenberger. 2008. Reproductive endocrinology of the largest dasyurids: Characterization of ovarian cycles by plasma and fecal steroid monitoring. Part I. The Tasmanian devil (Sarcophilus harrisii). General and Comparative Endocrinology 155:244-254.

Holtcamp, W. 2007. Sympathy for the devil. Ideas emerge to save the dying Tasmanian devil. Scientific American 296:27.

Izzard, L., S. Graves, E. Cox, S. Fenwick, N. Unsworth, and J. Stenos. 2009. Novel rickettsia in ticks, Tasmania, Australia. Emerging Infectious Diseases 15:1654-1656.

Jackson, A. E. In this issue - May 2010. Australian Veterinary Journal 88:157-159.

Jones, M., P. Jarman, C. Lees, H. Hesterman, R. Hamede, N. Mooney, D. Mann, C. Pukk, J. Bergfeld, and H. McCallum. 2007. Conservation management of Tasmanian devils in the context of an emerging, extinction-threatening disease: Devil Facial Tumor Disease. EcoHealth 4:326-337.

Jones, M. E. 1993. Road mortality of eastern quolls and devils on the Cradle Mountain tourist road., Department of Transport and Works and Department of Environment and Land Management, Parks and Wildlife Service.

Jones, M. E. 2000. Road upgrade, road mortality and remedial measures: impacts on a population of eastern quolls and Tasmanian devils. Wildlife Research 27:289-296.

Jones, M. E., A. Cockburn, R. Hamede, C. Hawkins, H. Hesterman, S. Lachish, D. Mann, H. McCallum, and D. Pemberton. 2008. Life-history change in disease-ravaged Tasmanian devil populations. Proceedings of the National Academy of Sciences of the United States of America 105:10023-10027.

Jones, M. E., D. Paetkau, E. Geffen, and C. Moritz. 2003. Microsatellites for the Tasmanian devil (Sarcophilus laniarius). Molecular Ecology Notes 3:277-279.

Jones, M. E., D. Paetkou, E. Geffen, and C. Moritz. 2004. Genetic diversity and population structure of Tasmanian devils, the largest marsupial carnivore. Molecular Ecology 13:2197-2209.

Jones M. E. and H. McCallum (2011) The Devil's Cancer. Scientific American 304: 72-77

Appendix B

Jones, S. M., T. J. Lockhart, and R. W. Rose. 2005. Adaptation of wild-caught Tasmanian devils (Sarcophilus harrisii) to captivity: evidence from physical parameters and plasma cortisol concentrations. Australian Journal of Zoology 53:339-344.

Kreiss, A., N. Fox, J. Bergfeld, S. J. Quinn, S. Pyecroft, and G. M. Woods. 2008. Assessment of cellular immune responses of healthy and diseased Tasmanian devils (Sarcophilus harrisii). Developmental and Comparative Immunology 32:544-553.

Kreiss A., C. Tovar, D. L. Obendorf, K. Dun and G. M. Woods (2011) A Murine Xenograft Model for a Transmissible Cancer in Tasmanian Devils. Veterinary Pathology 48: 475-481

Kreiss, A., D. L. Obendorf, S. Hemsley, P. J. Canfield, and G. M. Woods. 2009. A histological and immunohistochemical analysis of lymphoid tissues of the Tasmanian devil. Anatomical Record 292:611-620.

Kreiss, A., B. Wells, and G. M. Woods. 2009. The humoral immune response of the Tasmanian devil (Sarcophilus harrisii) against horse red blood cells. Veterinary Immunology and Immunopathology 130:135-137.

Lachish S., P. Abraham and M. E. Jones (2011) A new PCR assay for reliable molecular sexing of endangered Tasmanian devils (Sarcophilus harrisii) from non-invasive genetic samples. Conservation Genetics Resources 3: 279-281.

Lachish, S., M. H., D. Mann, C. E. Pukk, and M. E. Jones. 2010. Evaluating selective culling of infected individuals to control Tasmanian devil Facial Tumor Disease. Conservation Biology.

Lachish, S., M. Jones, and H. McCallum. 2007. The impact of devil facial tumour disease on the survival and population growth rate of the Tasmanian devil. Journal of Animal Ecology 76:926-936.

Lachish, S., H. McCallum, and M. Jones. 2009. Demography, disease and the devil: Life-history changes in a disease-affected population of Tasmanian devils (Sarcophilus harrisii). Journal of Animal Ecology 78:427-436.

Lachish, S., H. McCallum, D. Mann, C.E. Pukk and M. E. Jones (2010) Evaluation of Selective Culling of Infected Individuals to Control Tasmanian Devil Facial Tumor Disease. Conservation Biology 24: 841-851.

Lachish, S., K. J. Miller, A. Storfer, A. W. Goldizen, and M. E. Jones. 2010 Evidence that disease induced population decline changes genetic structure and alters dispersal patterns in the Tasmanian devil. Heredity 106: 172-182.

Loh, R., J. Bergfeld, D. Hayes, A. O'Hara, S. Pyecroft, S. Raidal, and R. Sharpe. 2006. The pathology of Devil Facial Tumor Disease (DFTD) in Tasmanian Devils (Sarcophilus harrisii). Veterinary Pathology 43:890-895.

Loh, R., D. Hayes, A. Mahjoor, A. O'Hara, S. Pyecroft, and S. Raidal. 2006. The immunohistochemical characterization of Devil Facial Tumor Disease (DFTD) in the

Tasmanian Devil (Sarcophilus harrisii).Veterinary Pathology 43:896-903.
Lunney, D., M. Jones, and H. McCallum. 2008. Lessons from the looming extinction of the Tasmanian Devil. Pacific Conservation Biology 14:151-153.

McCallum, H. 2008. Tasmanian devil facial tumour disease: lessons for conservation biology. Trends in Ecology & Evolution 23:631-637.

McCallum, H. and M. Jones. 2006. To lose both would look like carelessness... Tasmanian Devil Facial Tumour Disease. Public Library of Science. Plos Biology 4:1671 - 1674.

McCallum, H. and M. Jones. 2010. Sins of omission and sins of commission: St Thomas Aquinas and the devil. Australian Zoologist.

McCallum, H., M. Jones, C. Hawkins, R. Hamede, S. Lachish, D. L. Sinn, N. Beeton, and B. Lazenby. 2009. Transmission dynamics of Tasmanian devil facial tumor disease may lead to disease-induced extinction. Ecology 90:3379-3392.

McCallum, H., D. M. Tompkins, M. Jones, S. Lachish, S. Marvanek, B. Lazenby, G. Hocking, J. Wiersma, and C. E. Hawkins. 2007. Distribution and impacts of Tasmanian devil facial tumor disease. EcoHealth 4:318-325.

McGlashan, N. D., D. L. Obendorf, and J. S. Harington. 2007. Update on the devil facial tumour in Tasmania. European Journal of Oncology 12:75-80.

Miller, W., V. M. Hayes, A. Ratan, D. Petersen, N. E. Wittekindt, J. Miller, B. Walenz, J. Knight, J. Qi, F. Zhao, Q. Wang, O. Bedoya, N. Katiyar, L. P. Tomsho, L. M. Kasson, R-A. Hardie, P. Woodbridge, E. Tindall, M. F. Bertelsen, D. Dixon, S. Pyecroft, K. M. Helgen, A. M Lesk, T. H. Pringle, N. Patterson, Y.
Zhang, A. Kreiss, G. M. Woods, M. E. Jones, and S. C. Schuster (2011) Genetic diversity and population structure of the endangered marsupial Sarcophilus harrisii (Tasmanian devil). Proceedings of the National Academy of Sciences in press.

Morner, T., D. L. Obendorf, M. Artois, and M. H. Woodford. 2002. Surveillance and monitoring of wildlife diseases. Rev. sci. tech. Off. int. Epiz. 21:67-76.

Murchison, E. P. 2008. Clonally transmissible cancers in dogs and Tasmanian devils. Oncogene 27.

Murchison, E. P. 2009. Clonally transmissible cancers in dogs and Tasmanian devils. Oncogene 29:S19-S30.

Murchison, E. P., C. Tovar, A. Hsu, H. S. Bender, P. Kheradpour, C. A. Rebbeck, D. Obendorf, C. Conlan, M. Bahlo, C. A. Blizzard, S. Pyecroft, A. Kreiss, M. Kellis, A. Stark, T. T. Harkins, J. A. M. Graves, G. M. Woods, G. J. Hannon, and A. T. Papenfuss. 2010. The Tasmanian devil transcriptome reveals Schwann cell origins of a clonally transmitted cancer. Science 327:84-87.

Obendorf, D. L. and N. D. McGlashan. 2008. Research priorities in the Tasmanian devil facial tumour debate. European Journal of Oncology 13:229-238.

Pearse, A.-M. and K. Swift. 2006. Transmission of devil facial-tumour disease. Nature 439:549.

Phillips, B. T. and S. M. Jackson. 2003. Growth and development of the Tasmanian devil (Sarcophilus harrisii) at Healesville Sanctuary, Victoria, Australia. Zoo Biology 22:497-505.

Pyecroft, S. B., A.-M. Pearse, R. Loh, K. Swift, K. Belov, N. Fox, E. Noonan, D. Hayes, A. D. Hyatt, L. F.Wang, D. B. Boyle, J. S. Church, D. J. Middleton, and R. J. Moore. 2007. Towards a case definition for Devil Facial Tumour Disease: What is it? EcoHealth 4:346-351.

Reppas, G., P. Nosworthy, T. Hansen, M. Govendir, and R. Malik. Inguinal panniculitis in a young tasmanian devil (Sarcophilus harrisii) caused by Mycobacterium mageritense. Australian Veterinary Journal 88:197-200.

Siddle, H. V., A. Kreiss, M. D. B. Eldridge, E. Noonan, C. J. Clarke, S. Pyecroft, G. M. Woods, and K.

Belov. 2007. Transmission of a fatal clonal tumor by biting occurs due to depleted MHC diversity in a threatened carnivorous marsupial. Proceedings of the National Academy of Sciences of the United States of America 104:16221-16226.

Siddle H. V., J. Jolanta; Y. Cheng, M. E. Jones and K. Belov (2010) MHC gene copy number variation in Tasmanian devils: implications for the spread of a contagious cancer. Proceedings of the Royal Society B-Biological Sciences 277: 2001-2006.

Siddle, H. V., J. Marzec, Y. Cheng, M. Jones, and K. Belov. 2010. MHC gene copy number variation in Tasmanian devils: Implications for the spread of a contagious cancer. Proceedings of the Royal Society of London B (accepted 9 Feb 2010; on-line 10 March 2010).

Siddle, H. V., C. Sanderson, and K. Belov. 2007. Characterization of major histocompatibility complex class I and class II genes from the Tasmanian devil (Sarcophilus harrisii). Immunogenetics 59:753-760.

Stewart, N. J., S. S. Bettiol, A. Kreiss, N. Fox, and G. M. Woods. 2008. Mitogen-induced responses in lymphocytes from platypus, the Tasmanian devil and the eastern barred bandicoot. Australian Veterinary Journal 86:408-413.

Tovar, C., D. Obendorf, E.P. Murchison, A. T. Papenfuss, A. Kreiss, and G. M. Woods (2011) A tumour specific diagnostic marker for transmissible facial tumours of Tasmanian devils: Immunohistochemistry studies. Veterinary Pathology, in press.

Vanherberghen, M. and D. Desmecht. 2007. The transmissible facial tumor of the Tasmanian Devil. La tumeur faciale transmissible du Diable de Tasmanie 151:107-111.

Appendix B

Vetter, W., R. Von Der Recke, R. Symons, and S. Pyecroft. 2008. Determination of polybrominated biphenyls in Tasmanian devils (Sarcophilus harrisii) by gas chromatography coupled to electron capture negative ion tandem mass spectrometry or electron ionization high-resolution mass spectrometry. Rapid Communications in Mass Spectrometry 22:4165-4170.

Vilcins, I. M. E., J. M. Old, and E. Deane. 2009. Detection of a Hepatozoon and spotted fever group Rickettsia species in the common marsupial tick (Ixodes tasmani) collected from wild Tasmanian devils (Sarcophilus harrisii), Tasmania. Veterinary Parasitology 162:23-31.

Woods, G. M., A. Kreiss, K. Belov, H. V. Siddle, D. L. Obendorf, and H. K. Muller. 2007. The immune response of the Tasmanian devil (Sarcophilus harrisii) and devil facial tumour disease. EcoHealth 4:338-345.

KREISS, A. & WOODS, G. M. - Immunogenic studies with captive devils; immunisation with killed tumour cells and subsequent challenge

Three western population devils were used in a captive trial to assess the capability of devils to develop an immune response to a DFT vaccination followed by live tumour cell challenge.

TD 111 a female trapped at *Woolnorth* with four pouch young - AKA *'Christine'*; TD 146 - one of TD 111 pouch young when originally wild-caught - AKA *'Cedric'*; TD 145 - another pouch young of TD 111 from a subsequent mating in captivity to an Arthur River male - AKA *'Kinky'*

CHRISTINE - **TD 111** [ex Woolnorth population] MHC class I - **type A**; MHC class II - diverse phenotype

Developed no detectible humoral immune response to DFT vaccination and did develop tumours 16 weeks post challenge; challenged in week 32, tumours palpable by week 4; by week 53 the tumour measured `2.3 -2.4 mmm in diameter. Was re-immunized against strain 3 (first booster) in week 51 and against strains 1,2 & 4 in week 53. Tumours present on both the left and right sides of the cheek were surgically removed in week 58. In week 70 no palpable tumours; however, by week 75 - recurrent DFT nodule in right cheek subcutis near incision line.

CEDRIC - **TD 146** [male offspring from wild mating] - MHC class I - **type A**; MHC class II - normal phenotype; different MHC class I epitopes to the DFT cells.

Developed no humoral immune response to DFT vaccination (3 week vaccination followed by a booster @ week 8); at week 30 booster vaccination slight increase in antibody titre) and at week 41 challenged with strain 2 of DFT (subcutaneously into right cheek; sub-gingivally into oral mucosa next to left M1) and again at week 68 with strain 3 (**again** subcutaneously into right cheek; and also subcutaneously into the left cheek) - and did develop tumours in week 90.

Kriess (Chapter 6) states that no DFT tumour developed after first challenge 'suggesting that the immunisation had been effective' but 'not strong'; that was up to week 68 or just under 7 months! However in week 68 Cedric was re-challenged with live DFT cells this time a different strain - and tumours were detected at both sites (i.e. left and right cheek inoculation sites) by week 90 - 22 weeks later!

CLINKY - **TD 145** [male offspring from captive mating with an Arthur River male **TD 8 & TD 13**] - MHC class I - **type L**; MHC class II - diverse phenotype; many bands patterns in the MHC epitopes similar to eastern devil population and DFT tumour cells.

Developed strong humoral immunity to DFT vaccination (3 week vaccination followed by a booster @ week 8); challenged at week 30 (no obvious antibody response detected) - and at week 41 challenged with strain 2 of DFT and at week 53 (12 weeks after this challenge) developed DFT tumours at both inoculation sites (subcutaneously into right cheek; sub-gingivally into oral mucosa next to left M1).

Appendix C

Gingival tumour ulcerated by week 65. At week 71 the cheek tumour was removed surgically and Kreiss suggests a 3-fold increase in antibody by week 75. by week 79 devil euthanased after displaying vomiting & weight loss with post mortem detection of DFT metastasis in right submandibular LN and regrowth of tumour at the site of surgical resection. Cause of the devil's vomiting and weight loss not determined at necropsy.

Cedric and ***Clinky*** are half brothers - same mother ***Christine***

18 November 2008 (Week 58): [with Alex K and Barry W] surgically removed tumours from Christine. The right cheek tumour was ulcerated due to prior biopsy (see digital image); the left cheek tumour nodules were subcutaneous (see digital image).

29 January 2009 (Week 70): [with Alex K and Barry W] checked ***Christine*** and ***Cedric*** for resurgence of DFT at sites of resection. None detected - both remain under observation.

6 March 2009 (Week 75): ***Christine*** is found comatose and thin; she is euthanased; no weight taken. Common bile duct obstruction close to cystic bile duct junction (?infiltrative neoplasia) (digital image); jaundiced liver; extremely enlarged gall bladder; excessively yellow sclera and bodily fat reserves.

Right cheek removed - one prominent DFT nodule (digital images) at or near surgical excision line; possible one smaller DFT nodule ~ 2mm in diam. Left cheek removed…no obvious DFTD nodules detected (both cheeks fixed in 10% F-S for sectioning and periaxin staining) Regional LN appear normal; no metastases in major organs or body cavities; ?fat necrosis/lipomas in omentum. Did not check the lower lumbar spine for any vertebral/spinal lesions associate with clinical hind limb paresis. Euthanased 6 March 2009). Process histology 8 April 2009 including serial sections in vicinity of the excised larger DFTD-like nodule [7mm x 5mm x 4mm] and surgical excision site.

Nestin staining expressed strongly in DFTD cells - nestin is expressed in stem cells in mature mammals and within developmental embryos and foetuses in especially neuronal tissues. Nestin is down-regulated in mature/adult resting cells.

22 March 2009 DFT aggregates in deep subcutis of right cheek on H&E; all three sections in vicinity of the live tumour inoculation and excision line. Biliary hyperplasia and obstruction in common bile duct.

Working on this section below……

DFT tumours fail to express MHC class II antigens

All **eastern devils** tested in 'in vivo' allograft experiments - total of 8 animals - all showed host-graft or graft-host rejection.

MLRs which effectively measures MHC class II was used as a secondary 'in vitro' test of immunogenic recognition - 1st pair from different regions of eastern Tasmania low MLR coefficient ~1; 2nd pair MLR coefficient - 17; 3rd pair MLR - 3-4; 4th pair .

WEST PENCIL PINE STUDY SITE -

Appendix C

Erica - MHC class I - **Type-L** developed DFT naturally; there are no? strain **Type 1** DFTD in the West Pencil Pine population . Ercia was like ***Clinky*** a **Type-L** devil.

?Another **Type-L** devil at WPP did develop an DFTD-specific antibody response as a result of the natural 'vaccination'.

Gabby's Honours work - MLR between NW devils - *'low response';* 'a good MLR response with a **Type-1** individual.

Menna Jones - 19 March 2009

The Mt William NP devil population has very low genetic diversity. As a result of selection pressure of DFTD in this population for over 10 years - the remaining devils are showing a higher degree of 'observed heterozygosity'. The MHC class 1 types in this population are **Type-1; Type-A and Type-G**.
The Forestier Peninsula Disease Suppression Trial - begun in 2004 with 2006 being the year that new infection rate (what Menna calls the "transition rate" had reached 15%)
By 2008 there was no difference in the devil demographics to that seen at Freycinet!
Wants to change the suppression trial to one in which all adult devil irrespective of disease status are removed from the population.

Cathy Belov & Hannah Siddle - 19 March 2009

Currently there are 26 MHC class I types - this is very low number of MHC polymorphisms and the differences between these types are *'minimal'. 'All but two of these types have been sequenced.'*

Type-L types *'have extra variations'.*

Belov mentions that within the Tasmanian devil MHC class I genes there are between 2 and 10 alleles!

There is a *'lack of a critical couple of alleles'* in these genes in the NW devil population.

They are examining 'back-libraries' from ***Spirit*** and ***Cedric*.**

WHO IS ***SPIRIT*** AND WHERE DOES IT COME FROM?

There is diversity present in the MHC class II but only one family of genes has been examined; and then there's class III. This class II diversity give validation for Kreiss's uniform host-graft rejections in the skin graft experiments.

MHC class II are found on the immunologically competent stem cells and their prgenitors - they help to recognise exogenous antigens (microbiological/parasitological/viral).

Belov mentions that Murchison identified 200-300 'immuno-function genes' in the devil; this is quite low cf. humans ~1500 immuno-function genes.

Jody,

This has a lot in it.....you are correct about the selective referencing. Several scientists have contacted me about this matter and consider it highly unusual. They suggest even if they disagree with our publications, they are relevant and should be referred to and if necessary challenged....but they choose to just disregard them outright!

I hope to get some other background information (referenced) on eco-toxics and disease in marsupials up on Sourcewatch and PIT in the near future. The selective science down here is rather worrying.

The origins of the PBBs especially the products that contain one congener in highest concentration in some devil fat (PBB 153) needs to be researched. Only 16 devils done in the Pyecroft pilot study!! Not good enough.

In 2006 when one of my co-authors, Dr Neil McGlashan contacted the then DFTD Manager - Alistair Scott - about some data to include in our first devil paper, Scott demanded he be sent the draft to review and then when Neil declined he demanded to know which journal we were sending the MS to for consideration. He blatantly suggested the Government & its scientists had a right to contact the journal's editor and get the opportunity to referee or veto this paper.

When this paper and the AJV letter that we had published in May 2006, the Government took quite some prodding to place the papers on the list of publications for DFTD and devils.

Attached is my speech at the launch of the Tasmanian Eco-toxicology Research Fund from last Friday.

Kind regards,

David

Appendix E

Dear all

As some of you know, I have for some time been very concerned at the potential for individual members of the APVMA's Community Consultative Committee to use their membership of this committee as a platform to publicly pursue their own organisations' agendas. This is particularly so when these individuals hide behind the skirts of the APVMA to do so, and also receive sitting fees generously furnished by CropLife members through the APVMA's full cost recovery processes.

Today's story on Simazine in the Australian is the realisation of my concern. Credence is given to Anthony Amis, the "committee's environmental representative", when in fact he is an employee of Friends of the Earth which is one of the most extreme environmental activist groups operating in Australia.

Who is Mr Amis to talk about a public health matter when the committee is chaired by Dr Heather Yeatman from the Public Health Association of Australia?

The answer is that environmental activists are working with green politicians and pseudo scientists in Tasmania and in Queensland to place pressure on the APVMA to ban atrazine, simazine and other triazines. This is a concerted campaign that has been strategically pursued by the activists (particularly the National Toxics Network) in the Sydney Morning Herald last year and now the Australian.

They continue to achieve hyperbolic headlines with absolutely no scientific evidence to back it up. That is why the APVMA finalised its review of atrazine last year and allowed its continued use.

The APVMA is to be commended for its patient and ongoing rebuttals of the alarmist accusations being generated on a non-stop basis by the activists.

But I believe we must seriously consider whether the Community Consultative Committee is the most appropriate way for the APVMA to engage in a meaningful dialogue with the community about pesticide use. The CCC is no more than a convenient vehicle for activists to legitimise their outlandish and misleading campaigns.

Paula

Paula Matthewson
Chief Executive Officer

Tel: +61 2 6230 6399
Fax: +61 2 6230 6355

Appendix E

Email: paula.matthewson@croplifeaustralia.org.au
Website: www.croplifeaustralia.org.au