



Expert witness statement of Dr Roger Drew Expert of Gunns Limited

**In the matter of the Bell Bay Pulp Mill Project: A project of State Significance
Resource Planning and Development Commission inquiry
Proponent: Gunns Limited**

1. Name and address

Roger Drew,
Toxikos Pty Ltd.,
PO Box 74,
East Caulfield,
Vic, 3145

2. Area of expertise

My qualifications and experience are detailed in Attachment 1.

I am a certified toxicologist with the American Board of Toxicology. Eligibility for certification requires demonstration of expert knowledge and experience in mammalian toxicology and ecotoxicology. Certification is by exam, with renewal by exam every 5 years. I was first certified in 1996 and to the best of my knowledge I am one of only three such certified toxicologists in Australia.

I have been conducting risk assessments for the impact of chemicals on human health and the environment for 25 years. This has included participation in World Health Organization task groups for evaluation of chemical risks. I have also been a member of standing committees of the National Health and Medical Research Council and the National Occupational Health and Safety Commission that were constituted for the evaluation of chemicals and establishing standards.

As a corporate toxicologist in the chemical industry, and as a consultant, I have conducted numerous impact assessments for chemicals in emissions, and for registration of industrial chemicals.

I have undertaken government contracts for review and development of health risk assessment techniques in Australia, and have been invited to participate in many Government sponsored workshops on the matter.

3. Scope

3.1 Instructions

Toxikos Pty Ltd was engaged by Gunns Limited to:

- Conduct a human health risk assessment for emissions to air from the proposed pulp mill during normal operation. This report is located in the Draft IIS at Appendix 21 of Volume 10.
- Conduct a human health risk assessment for chemicals in effluent released from the proposed pulp mill, during normal operation, into the marine environment. This is located in the Draft IIS at Appendix 22 of Volume 10.
- To write a commentary on the possible impacts of discharged mill effluent on a nearby seal colony, this is located in the Draft IIS at Appendix 23, Volume 10.

I am the principal author of the above reports and I adopt the contents except where qualified in this witness statement.

After the Draft IIS was produced, Toxikos was engaged to:

- Conduct a marine impact assessment for the proposed discharge of mill effluent, during normal operation, into Bass Strait. I am the principal author of this report which is at Attachment 2 of this statement.

In this witness statement I have been asked to respond to issues raised in submissions made during the public exhibition of the Draft IIS that are related to:

- The human health risk assessment of emissions discharged to air. In particular to respond to the comments raised by UniQuest (dated 13 November 2006) in their report to the RPDC;
- The human health risk assessment of effluent discharged into the sea. In particular, to respond to the comments and criticisms raised by Beca AMEC in their general review of the Draft IIS (dated 20th October 2006) and by the UniQuest review (dated 13 November 2006) in their reports to the RPDC;
- Commentaries on the possible impacts of discharging mill effluent on nearby seal colonies, particularly those from Beca AMEC (dated 13th October 2006) and the Tasmania Government Agency submission.

The substance of most submissions on marine issues, including that from whole of Government, relate to:

- The absence in the Draft IIS of considerations of discharged effluent on marine organisms; or
- The assessment of dioxin impacts, in particular issues pertaining to bioaccumulation and biomagnification.

These matters are addressed in the marine impact assessment at Attachment 2.

3.2 Information relied upon

Information relied upon in formulating the opinions in this statement is contained in my reports at Appendices 21, 22 and 23 of Volume 10 of the Draft IIS, and in Attachment 2 of this statement.

I have also read the witness statements of Robin Ormerod, Veronique Levy and Attachment 3 from the statement of Esa Vakkilainen.

3.3 Limitations and exclusions

In relation to the above named reports in the Draft IIS, and at Attachment 2, I am not aware of any limitations or exclusions that will materially alter the information or opinions contained in those reports.

With respect to the marine impact assessment at Attachment 2; the tight time constraints under which the report was constructed may have led to some expressions, concepts and opinions not being written with appropriate clarity for the lay reader. Nevertheless the issues and impacts have been carefully considered in all the detail reasonably possible given the prospective status of the proposed mill and time available.

4. Human Health assessment of emissions to air

4.1 Findings

The overall conclusions from the health risk assessment of predicted emissions to air from the proposed pulp mill are:

- Mill emissions are very unlikely to cause direct health effects, either alone or as a mixture.
- The low levels of dioxin released from the proposed mill are very unlikely to cause health effects.
- It is very unlikely odour events will be experienced by people living near the proposed mill, or that they will be annoyed by odour.
- Mill emissions have negligible incremental impact on existing health issues within Launceston.

In formulating the above opinions I have relied upon the information on exposure levels derived by GHD from their air dispersion modelling of predicted mill emissions. Since that time additional dispersion modelling has been undertaken by Pacific Air & Environment (PAE)¹. The differences between the GHD modelling outputs and those of PAE are explained in the witness statement of Robin Ormerod. It is notable that GHD and PAE reach the same conclusions regarding the impact of mill emissions on local and regional air quality.

¹ PAE (2006). Supplementary air quality assessment of proposed pulp mill. Pacific Air & Environment, 8 August 2006, Job No 2238.

I have read the report of PAE and the witness statement of Robin Ormerod. I am of the opinion that the later air dispersion modelling work of PAE does not change the conclusions reached in Appendix 21, Volume 10 of the Draft IIS.

4.2 Response to community concerns and key submissions

The principal limitations raised by UniQuest in their 13 November report are echoed by a number of other submitters. The two main limitations are:

- The incomplete inventory of emissions from existing local industry restricts full assessment of the combined emissions from the proposed pulp mill with those of existing industry; and
- Lack of formal consideration of ultrafine particulates in the risk assessment.

Incomplete inventory:

The problem of incomplete information on background ambient air concentrations of all pollutants of interest was acknowledged in the risk assessment. However the issue cannot be quantitatively addressed until the 'missing' information is obtained. Nevertheless, in the context of the Draft IIS, the primary objective of the health risk assessment was to evaluate the health impacts of emissions from the proposed pulp mill and not those associated with the existing industry situation. To this end the assessment has shown negligible effects from the mill emissions on their own. In addition, for the criteria pollutants (for which there was information from the existing industries) the assessment showed the mill had negligible additional impact. Based on my experience from assessing industrial emissions, I would expect a similar finding (i.e. negligible additional impact of mill emissions) if all the 'missing' background pollutants from the existing industries were included in the combined assessment.

It should also be pointed out that the emissions inventory supplied by existing industry was 2004 data. Since that time one industry has installed additional emission controls and another firm has closed down. Consequently the background exposures of criteria pollutants predicted by the dispersion modelling for the Draft IIS do not reflect the air quality situation when the proposed mill may become operational. The exposure concentrations used in the risk assessment are biased to over predict the risks.

Furthermore the risk assessment inherently contains compounding conservatism. For example:

- As is apparent from Attachment 3 of Esa Vakkilainen's witness statement, Jaakko Poyry has been conservative in the emission estimations they provided to GHD for air dispersion modelling;
- Toxikos has also been conservative in the use of the ground level concentration predictions when evaluating the potential for air emissions from the proposed mill to cause direct health effects.

Some submissions have pointed to the combined risk assessment undertaken for a Georgetown location near the industrial precinct as providing confirmation that existing industries are already causing, or are likely to cause health effects. The risk assessment does not support such logic. The risk assessment does not suggest health effects due to existing industry on its own or in combination with emissions from the proposed mill are probable. Rather, because of the compounding conservatism and other limitations articulated in Appendix 21 of the Draft IIS, it was recommended that a

better understanding of industry related exposures in the area would address some of the limitations associated with the combined risk assessment.

Ultrafine particulates:

The question of assessment of ultrafine particulates (i.e. those less than 2.5 μm) is a similar predicament to the 'missing' background pollutants. At the moment there is not a requirement to collect data on ambient concentrations for these size particulates and data was not available to be used in a risk assessment.

In their report of the impact on air quality, GHD (2006)² applied an assumed ratio for $\text{PM}_{2.5} : \text{PM}_{10}$ for each industrial source to the dispersion modelling results of PM_{10} . The NEPM goal for $\text{PM}_{2.5}$ is 25 $\mu\text{g}/\text{m}^3$ as a 24 hour average (this is an advisory value for jurisdictions reporting ambient air monitoring results to the National Environment Protection Council). At all discrete receptors, except Georgetown A, the resulting 'indicative' $\text{PM}_{2.5}$ concentrations were less than the NEPM reporting comparison value. At Georgetown A the 'indicative' $\text{PM}_{2.5}$ concentration was 29 $\mu\text{g}/\text{m}^3$. The manner in which the $\text{PM}_{2.5}$ concentrations were estimated by GHD is not based in empirical data and it is my opinion the information should not be relied upon as suggestive of actual exposures or of potential health risks. In his evidence, Robin Ormerod provides additional discussion of the issues associated with $\text{PM}_{2.5}$.

$\text{PM}_{2.5}$ is a subset of PM_{10} and hence is inherently included in the health risk assessment of these latter particulates. It is recognised however that there is emerging health information suggesting many of the health effects of PM_{10} may be associated with the $\text{PM}_{2.5}$ fraction and that ideally it would be useful to formally address the risks of $\text{PM}_{2.5}$ particulates.

It is noted from Robin Ormerod's witness statement that a large proportion of measured PM_{10} at the Rowella monitoring station is sea salt. It would be anticipated that a large proportion of $\text{PM}_{2.5}$ would also be sea salt. ANSTO have been measuring the composition of $\text{PM}_{2.5}$ at a number of sites in Australia (ANSTO 2004, 2005, 2006)³. Over a 12 month period sea salt accounted for approximately 15 – 28%, and ammonium sulfate for 13 – 27%, of the $\text{PM}_{2.5}$ mass measured in Adelaide, Brisbane, Melbourne and Sydney. At Cape Grim in Northwestern Tasmania sea salt was 55.2% and ammonium sulfate 22.2% of the three year average mass of $\text{PM}_{2.5}$. From the geographical position of Georgetown, it would be expected that the sea salt and sulfate proportions of $\text{PM}_{2.5}$ would be between the values for the capital cities and Cape Grim. Importantly at environmentally relevant exposure levels, sea salt and sulfate salts are considered to be without adverse health effects in healthy or asthmatic individuals (RIVM 2002, Schliesinger and Cassee 2003)⁴.

² GHD (2006). Proposed Pulp Mill – Bell Bay. Impact on Air Quality. Report – Draft IIS, June 2006. (Located at Volume 9 Appendices of the Draft IIS).

³ ANSTO (2006). Fine particulate aerosol sampling. Australian Nuclear Science and Technology Organisation Newsletter Number 34, January 2006.

⁴ RIVM (2002). On health risks of ambient PM in the Netherlands. Netherlands Aerosol Programme, National Institute for Public Health and the Environment (RIVM), Report 650010 032, October 2002. Schliesinger, R.B. and Cassee, F. (2003). Atmospheric secondary inorganic particulate matter: The toxicological perspective as a basis for health effects risk assessment. *Inhal. Toxicol.* 15: 197 – 235.

Denier van der Gon et al. (2006)⁵ state that the European Commission has recently indicated that for the purposes of compliance reporting against PM₁₀ standards, measured ambient PM₁₀ concentrations may be corrected for natural contributions such as sea salt. The net effect is to lower the measured PM₁₀ and hence the calculated health risks will also be lowered. Because sea salt or sulfate are not included in the health risk predictions at Appendix 23 these considerations do not directly affect the health risk assessment. They will however influence calculated risks should measured PM levels be used in any further health risk assessment, or if the dispersion modelling were to be adjusted to match the measured PM at Rowella, or any other monitoring station in the Bell Bay area.

5. Human Health assessment of effluent discharge

5.1 Findings

The human health risk assessment concluded risks to the general public from chemical constituents in the proposed discharged effluent were negligible. The conclusions are based on:

- Lack of noteworthy uptake or accumulation of effluent constituents by fish in the immediate outfall area, and hence also at distances further away;
- Little potential for tainting of fish in the immediate outfall area;
- Low potential for the general public to consume fish that may be exposed to relatively high concentrations of effluent; and
- Within a short distance of the diffuser, water concentrations of effluent constituents being less than World Health Organization and Australian guidelines for recreational activities.

5.2 Response to community concerns and key submissions

The UniQuest report points to areas requiring clarification. Those for which the information is not already in the Draft IIS are:

- *Nutrients*: The impact of nutrients is addressed in Attachment 2. It relies on assessments undertaken by GHD that are included in Dr Veronique Levy's water quality assessment report.
- *Natural constituents of northern vs southern hemisphere woods*: This information is not pertinent to effluent composition as the biological effluent treatment will substantially change the constituent composition in effluent from that in wood. Information on metal concentration in the Bell Bay effluent was determined by Jaakko Poyry from analysis of Tasmanian woods that will be feedstock for the mill.

⁵ Denier van der Gon et al. (2006). The contribution of sea salt aerosol to PM₁₀ in the Netherlands and a methodology to correct the annual PM₁₀ concentration and number of PM₁₀ exceedance days for sea salt aerosol. Geophysical Research Abstracts 8: 03978.

- *Knowledge of effluent dilution:* Effluent dilution and fate was determined by GHD in three reports where hydrodynamic modelling of the effluent has been progressively refined and improved. I have summarised these in the marine impact assessment report at Attachment 2.
 - *Breakdown of infrastructure for effluent treatment:* I understand the issue of infrastructure failure is being addressed by Jaakko Poyry.
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6. Effluent impact on seals

6.1 Findings

The commentary on the impact of discharge of the proposed effluent to seals concluded the viability of the seal colony at Tenth Island would not be adversely affected.

The additional work on this issue undertaken in Attachment 2 did not change the conclusions reached in the Draft IIS.

6.2 Response to community concerns and key submissions

Many submissions believed bioaccumulation/biomagnification of dioxins through the food chain of seals was inadequately addressed in the Draft IIS. These aspects have been further clarified in the marine impact assessment report at Attachment 2.

The Beca AMEC reports of 13th and 20th October 2006 agreed with the conclusions reached in Appendices 22 and 23 regarding impacts of dioxins on human health and seals but pointed to limitations of the argument used to support the conclusion. They considered the commentary on seals made a moderately strong case for low risk. However they were critical that the conclusion was based upon weak evidence for lack of bioaccumulation and biomagnification in marine food chains, and in their opinion unsubstantiated statements and assumptions, and lack of discussion of the foraging range of seals. The Government Agency submission made similar comments. That submission also argued that a quantitative assessment of the effects of dioxins, incorporating consideration of the diet of seals, should be undertaken rather than the qualitative evaluation of the commentary included in the Draft IIS at Appendix 23.

All these comments are addressed in Attachment 2.

7. Marine impact of discharged effluent

7.1 Findings

The marine impact assessment at Attachment 2 concludes the proposed discharge of mill effluent into Bass Strait will not adversely impact the survival, breeding and migration of fish, marine mammals, birds, or other organisms in Tasmanian or Commonwealth waters.

Threatened or protected species will not be adversely affected by the proposed discharge of effluent.

It is also concluded the existing primary productivity of the ecosystem(s) in the receiving marine environment will be unaltered, which, together with lack of direct toxicity to organisms, indicates ecological community structures and species diversity are unlikely to be adversely changed by the discharged effluent.

7.2 Response to community concerns and key submissions

With regard to effluent impacts on marine flora and fauna, many submissions, including the 13 October report of Beca AMEC and the Tasmanian Government Agency submission, were critical of the lack of, or exceptionally brief assessment in the Draft IIS.

In particular, Beca AMEC considered key outstanding issues included no assessment of the impact on the listed Gunn screw shell, ambiguity regarding the chronic effect of chlorate on seaweeds, toxicity testing of effluent on macroalgae not the same as tests quoted in the literature, inadequate consideration of cumulative effects and of potential chronic toxicity.

The above criticisms, plus others from a range of submissions, including the Tasmanian Government Agency submission, have been addressed in the marine impact assessment report at Attachment 2.

8. Declaration

I have made all the inquiries that I believe are desirable and appropriate and no matters of significance which I regard as relevant have, to my knowledge, been withheld from the Commission.



..... 30th January 2007
Roger Drew, PhD, DABT

Attachment 1

CURRICULUM VITAE

Personal Details

Name: Dr Roger DREW

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Place of Birth: England (Australian resident)

Date of Birth: 15th June, 1947

Professional Qualifications and Affiliations

1996 - Present Diplomate, American Board of Toxicology (D.A.B.T.,)

1994 - 1999 Adjunct Professor, Biochemical Toxicology, Key Centre for Applied and Nutritional Toxicology, RMIT University.

1990 - Present Associate Senior Lecturer, Department of Social and Preventive Medicine, Monash University.

1976 - 1978 Postdoctoral Training - Fogarty Visiting Fellow, Laboratory of Toxicology, National Cancer Institute, N.I.H., Bethesda, U.S.A.

1972 - 1975 Ph.D., Department of Human Physiology and Pharmacology, University of Adelaide.

1971 B.Sc. (Hons), Department of Human Physiology and Pharmacology, University of Adelaide.

1967-1970 B.Sc., University of Adelaide (Biochemistry and Pharmacology majors).

Employment Record

- 2000 – present Principal Consultant, Toxikos Pty Ltd., Toxicology Consultants.
- 1998 – 2000 Research Associate in Toxicology,
SHE Pacific Pty Ltd., Safety, Health and Environment Consultants.
- 1990 - 1997 Research Associate in Toxicology,
Manager, Toxicology Information Section,
Safety Health and Environment Division, ICI Australia.
- 1990 - 2000 Corporate Toxicologist, ICI/Orica Australia,
- 1984 - 1990 Senior Lecturer, Department of Clinical Pharmacology, School of Medicine,
Flinders University of South Australia.
- 1979 - 1983 Lecturer, Department of Clinical Pharmacology School of Medicine, Flinders
University of South Australia.

Professional Society Membership

- American Society of Toxicology (SOT)
- Australian Society of Clinical and Experimental Pharmacologists and Toxicologists (ASCEPT), (Founding member of Toxicology Section). (1978 - 1998)
- International Society of Regulatory Toxicology and Pharmacology (IS RTP).
- National Product Liability Association (NPLA)
- Australasian College of Toxicology and Risk Assessment

Professional Activities, Committees and Working Parties

- 2006 Presenter at “Advances in the Neurotoxicology and Risk Assessment of Volatile Organic Compounds”, a workshop organised by the Australasian College of Toxicology and Risk Assessment.
- 2005 Presenter at “Environmental Health Risk Assessment”, a short course organised by Entox (NRCET, National Centre for Environmental Toxicology), Queensland University.
- 2005 Presenter at “Toxicology in Public Health”, a short course organised by School of Public Health, Sydney University 22 -24 February.
- 2003 & 2004 Presenter of a Continuing Education Course, “Sensitisation: Biology, Testing and Classification. Australian Institute of Occupational Hygienists (AIOH), Annual General Meeting. Adelaide, December 2003. Repeated by request in 2004.
- 2002 Presenter of a Continuing Education Course, "Health based Risk Assessment", Australian Institute of Occupational Hygienists (AIOH), Annual General Meeting. Geelong, December 2002.
- 2002 - 2004 Associate Member of NHMRC Working Group on Toxicity and Risk Assessment.
- 2000 - Present Editorial Board, Regulatory Toxicology and Pharmacology.
- 1997 - Present Poisons Advisory Committee, Department Human Services, Victoria, Australia.

- 1997 - 1998 Technical Review Panel "Health Risk Evaluation" for "Towards a National Environment Protection Measure for Ambient Air Quality". The National Environment Protection Council (NEPC) of Australia.
- 1997 - 2001 Hazardous Substances Subcommittee, National Occupational Health and Safety Commission.
- 1994 - Present Policy and Management Board, NH&MRC National Research Centre for Environmental Toxicology, Queensland University, Brisbane.
- 1994 - 1997 Expert Working Group on Classification of Hazardous Substances, Worksafe Australia.
- 1994 - 1997 National Occupational Health and Safety Commission, Research Standing Committee.
- 1993 - 1997 Exposure Standards Reference Group, Worksafe Australia.
- 1993 - 1994 Expert Review Group for Model Regulations for the Control of Hazardous Workplace Substances. Part 2 Carcinogens, Worksafe Australia.
- 1991 - 2000 Course Advisory Committee, M.Sc (Toxicology), RMIT University, Melbourne.
- 1991 - 2000 Advisory Board for Key Centre of Applied & Nutritional Toxicology, RMIT University, Melbourne.
- 1991 - 1999 Australian Chemical Industry Council: NICNAS Industry Working Group, and Occupational Health Group.
- 1990 - 1993 Chemical Industry Advisory Committee, Centre for Environmental Toxicology, University of Technology, Sydney.
- 1990 Australian Standards Association: Standards for Pipes in Contact with Potable Water.
- 1988 - 1990 Ministerial Advisory Committee on Agricultural Chemicals, South Australian Department of Agriculture.
- 1987 NH & MRC: Plasticiser Working Group.
- 1987 South Australian Health Commission: Review of Level of Concern of Lead.
- 1981 NH & MRC Working Party: Toxicity of Fire Retardants.

NH & MRC Standing Committees

- 1993 - 1994 NH & MRC: Panel on Cosmetics
- 1986 - 1990 NH & MRC: Committee on Toxicity
- 1984 - 1986 NH & MRC: Consumer Products Safety Committee
- 1983 - 1986 NH & MRC: Pesticides & Agricultural Chemicals Committee

World Health Organisation/International Programme on Chemical Safety

- 1996 WHO/IPCS Environmental Health Criteria for Hexachlorobenzene. Observer Geneva, Switzerland.
- 1991 WHO/IPCS Environmental Health Criteria. Document and Health and Safety Guide for 1,3-dichloropropene, 1,2-dichloropropane and mixtures. Chairman of Task Group, Hanover, Germany. EHC Document 146.
- 1990 WHO/IPCS Environmental Health Criteria on Acrolein. Task Group Member, Geneva, Switzerland. EHC Document 127.
- 1989 WHO/IPCS Environmental Health Criteria on 1- and 2-Propanol. Task Group Member, Charlshalton, England. EHC Documents 102 and 103.
- 1985 WHO/IPCS Environmental Health Criteria for Butanols-Four Isomers. Chairman of Task Group, Geneva, Switzerland. EHC Document 65.
- 1984 WHO/IPCS Environmental Health Criteria for Organochlorine Pesticides Other than DDT. Task Group Member, Ottawa, Canada. EHC Documents 40, 41 & 42.

Symposia Organisation

- 1998 –2001 Scientific Committee, IXth International Congress on Toxicology, Brisbane, 2001
- 1996 - 1998 Scientific Committee, VIIIth International Congress on Toxicology, Paris, 1998.
- 1995 "Health-Based Risk Assessment of Contaminated Land: Focus on Carcinogens", ASCEPT Toxicology Section, Melbourne, February 27-28, 1995.
- 1991 - 1993 Member of Steering Committee for "Sixth International Conference on Environmental Mutagens", Melbourne, February 1993.
- 1990 "Health Effects of Environmental Contamination" with S. Tepe, ASCEPT Toxicology Section, Melbourne, August 1990.
- 1989 "Alternatives to *In Vivo* Toxicity Testing" with A. Harman, ASCEPT Toxicology Section, Melbourne, June 1989.
- 1988 "Interpreting Toxicity Tests" with S. Tepe, ASCEPT Toxicology Section, Sydney, March 1988.
- 1987 7th International Symposium on "Microsomes and Drug Oxidations" with J.O. Miners, D.J. Birkett, B.K. May and M.E. McManus, Adelaide, August 1987.
- 1981 "Metabolite Mediated Toxicity" with B.G. Priestly ASCEPT Annual General Meeting, Adelaide, December 1981.

Invited Lectures

- Too numerous to list.

Tertiary Teaching

This section includes lecture series in general and mechanistic toxicology, risk assessment and risk communication for the following courses, it does not include courses taught at Adelaide University or Flinders Medical Centre while employed by Flinders University of South Australia. These latter lectures and tutorials to science and medical students, dealt with general toxicology, safety testing of medicines, and pharmacology of a range of drug groups.

- 2002 – 2004 Toxicology, B.Vet Sc Course, Melbourne University.
- 1993 - Present Principles of toxicology and target organ toxicity. BSc Course.
Risk Communication. Master Public Health.
Monash University.
- 1992 - 1996 Ph.D research student supervision, RMIT.
- 1991 - 1995 Industrial Toxicology, Deakin University.
- 1990 - 2001 Master of Applied Science in Toxicology.
Royal Melbourne Institute of Technology.
- Supervision of M.Sc (Toxicology) & PhD research projects.
- 1990 - Present Master of Public Health, Monash University.

Publications

(Publication of abstracts, and papers presented at national and international scientific meetings are not included)

1. Boggiano, B.G. and Drew, R. Aspirin-paracetamol interactions in tablets. Aust. J. Pharm. 51: 602-604 Sci Supp. No. 84, 1970.
2. Drew, R. and Priestly, B.G. Microsomal drug metabolism during - naphthylisothiocyanate-induced cholestasis. Toxicol. Appl. Pharmacol. 35: 491-499, 1976.
3. Drew, R. and Priestly, B.G. Hexobarbital sleeping time and drug metabolism in rats with ligated bile ducts - a lack of correlation. Biochem. Pharmacol. 25: 1659-1663, 1976.
4. Drew, R., Priestly, B.G. and O'Reilly, W.J. Hexobarbital pharmacokinetics in rats after ligation of the common bile duct. J. Pharmacol. Exp. Ter. 201: 534-540, 1977.
5. Mimnaugh, E.G., Waring, R.W., Sikic, B.I., Magin, R.L. Drew, R., Litterst, S.L., Gram, T.E. and Guarino, A.M. The effects of whole body hyperthermia on the disposition and metabolism of adriamycin in rabbits. Canc. Res. 38: 1420-1425, 1978.
6. Gram, T.E. Sikic, B.I., Litterst, C.L., Mimnaugh, E.G., Drew, R. and Siddick, Z.H. The role of the lung in the metabolism and disposition of xenobiotics. In "In vivo aspects of biotransformation and toxicity of industrial and environmental xenobiotics", Ed. I. Gut, Excerpta Media, Amsterdam pp 53-58, 1978.
7. Drew, R. and Priestly, B.G. Failure of hypertrophic hypoactive smooth endoplasmic reticulum to produce cholestasis in rats. Toxicol. Appl Pharmacol. 45: 191-199, 1978.
8. Drew, R. and Priestly, B.G. The effect of chlorpromazine and erythromycin on bile salt-induced cholestasis in the rat. Pharmacology, 18: 202-209, 1979.

9. Siddik, Z.H., Drew, R., Sikic, B.I., Mimnaugh, E.G., Litterst, C.L. and Gram, T.E. Lack of correlation between cortisol-induced precocious activation of the fetal rabbit lung and drug metabolism. *Biochem. Pharmacol.* 28: 683-685, 1979.
10. Drew, R. and Gram, T.E. Vehicle alteration of paraquat toxicity in mice. *Toxicol. Appl. Pharmacol.* 48: 479-487, 1979.
11. Drew, R., Siddik, Z.H. and Gram, T.E. Effect of chlorpromazine on paraquat toxicity and accumulation by rat lung. *Toxicol. Appl. Pharmacol.* 50: 443-449, 1979.
12. Drew, R., Siddik, Z.H. and Gram, T.E. Uptake and efflux of ¹⁴C-paraquat by rat lung slices: The effect of imipramine and other drugs. *Toxicol. Appl. Pharmacol.* 49: 473-478, 1979.
13. Drew, R. Sikic, B.I. Mimnaugh, E.G., Litterst, C.L. and Gram, T.E. The distribution of ¹⁴C-imipramine in mice bearing Lewis Lung Tumour. *Life Sci.* 25: 1813-1820, 1979.
14. Drew, R. and Priestly, B.G. Choleric and cholestatic effects of infused bile salts in the rat. *Experientia*, 35: 809-810, 1979.
15. Mimnaugh, E.G., Siddik, Z.H., Drew, R., Sikic, B.I. and Gram, T.E. The effects of -tocopheral on the toxicity, disposition and metabolism of adriamycin in mice. *Toxicol. Appl. Pharmacol.* 48: 119-126, 1979.
16. Mimnaugh, E.G., Siddik, Z.H., Trush, M., Drew, R. and Gram, T.E. The effect of unilateral pneumonectomy on in vitro drug metabolism by the contralateral lung of rabbits. *Drug Metab. Dispos.* 7: 208-210. 1979.
17. Drew, R. and Siddik, Z.H. The effect of a specific 5HT uptake inhibitor (citalopram) on drug accumulation by rat lung slices. *Pharmacol.* 20: 27-31, 1980.
18. Siddik, Z.H., Drew, R., Litterst, C.L., Mimnaugh, E.G., Sikic, B.I. and Gram, T.E. Hepatic cytochrome P-450-dependent metabolism and enzymatic conjugation of foreign compounds in vitamin A deficient rats. *Pharmacol.* 21: 383-390, 1980.
19. Siddik, Z.H., Drew, R. and Gram, T.E. The metabolism and biliary excretion of sulfobromophthalein in vitamin A deficient rats. *Biochem. Pharmacol.* 29: 2583-2588 1980.
20. Drew, R., Siddik, Z.H., Mimnaugh, E.G. and Gram, T.E. Species and dose differences in the accumulation of imipramine by mammalian lungs. *Drug Metabolism Disposition*, 9: 322-326, 1981.
21. Drew, R. and Siddik, Z.H. Drug uptake by lung slices from paraquat pretreated rats. *Experientia*, 37: 1093-1095, 1981.
22. Drew, R. Volatile Solvents. In "Stimulants depressants and analgesics" Royal Commission in to the Non-Medical Use of Drugs. South Australia. Research Paper 9 pp 92-100, 1979.
23. Drew, R., Rowell, J. and Grygiel, J.J. Cimetidine: A specific inhibitor of hepatic aryl hydrocarbon hydroxylase (AHH) in the rat. *Res. Commun. Chem. Pathol. Pharm.* 33: 81-93, 1981.
24. Grygiel, J.J., Miners, J.O., Drew, R. and Birkett, D.J. Differential effects of cimetidine on theophylline metabolic pathways. *Eur. J. Clin. Pharmacol.* 26: 335-340, 1984.
25. Miners, J.O., Drew, R. and Birkett, D.J. Effect of cimetidine on paracetamol activation in mice. *Biochem. Pharmacol.* 12: 1996-1998, 1984.

26. Drew, R. and Miners, J.O. The effects of buthionine sulphoximine (BSO) on glutathione depletion and xenobiotic biotransformation. *Biochem. Pharmacol.* 33: 2989-2994, 1984.
27. Miners, J.O., Drew, R. and Birkett, D.J. Mechanism of action of paracetamol protective agents in mice in vivo. *Biochem. Pharmacol.* 33: 2995-3000, 1984.
28. Drew, R. and Knights, K.M. Postulated reactive intermediates of NSAID's. *Agents and Actions* 17: 127-133, 1986.
29. Knights, K.M., Cassidy, M.R. and Drew, R. Benoxaprofen induced toxicity in isolated rat hepatocytes. *Toxicol.* 40: 327-339, 1986
30. Knights, K.M., Cassidy, M.R., Ryall, R.G. and Drew, R. Interaction of benoxaprofen with rat erythrocytes: Effects on oxidative metabolism and membrane ATPase activities. *Biochem. Biophys. Res. Comm.* 54: 227-236, 1986.
31. Knights, K.M. and Drew, R. A radiosotopic assay of picomolar concentrations of coenzyme A in liver tissue. *Analyt. Biochem.* 168: 94-99, 1988.
32. Knights, K.M., Drew, R. and Meffin, P.J. Enantiospecific formation of fenoprofen CoA thioester in vitro. *Biochem. Pharmacol* 37: 3539-3542, 1988.
33. Fraunfelder, F.T., Coster, D.J. and Drew, R. Severe, chronic eye injury resulting from acute exposure to methyl ethyl ketone peroxide. *Amer. J. Ophthalmol* 110:635-640, 1990.
34. Knights, K.M. and Drew R. Effects of ibuprofen enantiomers on hepatocyte intermediary metabolism and mitochondrial respiration, *Biochem Pharmacol* 44; 1291 - 1296, 1992.
35. Drew, R. Toxicology of Plastics. In 'Occupational Toxicology' Ed. N. Stacey. Taylor & Francis Ltd, pp. 213-232, 1993.
36. Drew, R. Between the sheets: Understanding material safety data sheets. *Aust. Safety News* 64, 40-43, 1993.
37. Voskoboinik, I., Drew, R. and Ahokas, J.T. The effect of peroxisome proliferator nafenopin on the cytotoxicity of dihaloalkanes in isolated hepatocytes. *Toxicology in Vitro.* 19: 577-578 (1996).
38. Voskoboinik, I., Drew, R. and Ahokas, J.T. Differential effect of peroxisome proliferators on rat glutathione S-transferase isoenzymes. *Toxicology Letters.* 87: 147-155 (1996).
39. Voskoboinik, I., Drew, R. and Ahokas, J.T. Peroxisome proliferator nafenopin potentiated cytotoxicity and genotoxicity of cyclophosphamide in the liver and bone marrow cells. *Chemico-Biological Interactions.* 105: 81-97 (1997).
40. Voskoboinik, I., Ooi, S.G., Drew, R and Ahokas, J.T. Peroxisome proliferators increase the formation of BPDE-DNA adducts in isolated rat hepatocytes. *Toxicology* 122: 81-91 (1997).
41. Drew, R. and Frangos, J. (2007). The concentration of no toxicological concern: a risk assessment screening tool *J. Toxiol. Environ. Hlth* (In Press).

Attachment 2