How humane are our pest control tools?

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Overall Summary

Duration of project

- Start date: 1 September 2009
- Completion date: 10 November 2010

Project Code

- BNZ 11326

Business/Institution

- Landcare Research New Zealand Limited with subcontract to AWSBC, Massey University for Part 1. Key staff were Penny Fisher and Bruce Warburton (Landcare Research), Ngaio Beaulsoeil and David Mellor (AWSBC, Massey University).

Programme Leader

- Penny Fisher

Programme Title

- How Humane are our Pest Control Tools?

- Part 1. Vertebrate toxic agents and kill traps in mammal species

- Beausoleil NJ, Fisher P, Warburton B, Mellor DJ 2010. How humane are our pest control tools? Part 1. Vertebrate toxic agents and kill traps in mammal species. Unpublished report prepared for Biosecurity New Zealand, Project No. 11326. 86 p.

- Part 2. In-burrow rabbit control methods

- Fisher P, Campion M 2010. How humane are our pest control tools? Part 2. In-burrow rabbit control methods. Landcare Research Contract report L0027, 26 p. Prepared for the Ministry of Agriculture and Fisheries, Wellington, New Zealand.

- Part 3. Leg-hold traps, rotenone, alphachloralose and DRC-1339

- Fisher P, Campion M, Warburton B, Booth L 2010. How humane are our pest control tools? Part 3. Leg-hold traps, rotenone, alphachloralose and DRC-1339. Landcare Research Contract report LC76, 33 p. Prepared for the Ministry of Agriculture and Fisheries, Wellington, New Zealand.

**Parts 1, 2 and 3 have been completed as separate reports that have been combined into this single report as chapters. A short overall summary is given below*

Goal

To provide MAF Biosecurity New Zealand with objective information on the relative welfare impacts ('humaneness') of the vertebrate pest control tools used in New Zealand. Specific objectives were:

1. To collate relevant current literature from New Zealand, Australia, and elsewhere, if available, and review it for its applicability and utility for assessing the animal welfare impacts on the agreed range of species and devices/tools used for pest control, using the framework developed in Australia (Sharp & Saunders 2008).
2. To apply the Australian framework to evaluate the relative animal welfare impacts of the nominated pest control tools used in New Zealand.
3. To identify current gaps in the information necessary to assess relative welfare impacts of pest control tools to guide future research.

Project Summary

Increasingly, information on the animal welfare impacts of pest control methods is required to inform the decisions of operators and policymakers. To date, there has been no comprehensive evaluation of the welfare impacts associated with methods used in New Zealand. The purpose of this project was to apply a recently developed Australian assessment framework to produce a ranking of the relative welfare impacts of lethal control methods used for vertebrate pest management in New Zealand. This was completed for a range of pest control methods and reported in three parts.

Part1 assessed vertebrate toxic agents (VTAs) and kill traps as control tools for a range of introduced mammal species. The VTAs and corresponding target and non-target mammal species assessed are shown in Table 1. For these control tools, relevant data were collated as reference material and also to identify information gaps. A review panel with expertise in animal welfare science, pest animal management, veterinary science and toxicology applied this reference material in conjunction with a framework developed in Australia (referred to throughout as Sharp & Saunders 2008) to produce an assessment of the relative welfare impacts of the selected control tools.

Part 2 (Fisher & Campion 2010) assessed in-burrow control tools for rabbits; the fumigants magnesium phosphide and chloropicrin and burrow destruction methods based on ignition of flammable gas (the 'Rodenator'). Literature review, data collation and application of the Sharp & Saunders (2008) framework were used to produce relative welfare impact rankings through expert opinion rather than a panel assessment.

Part 3 (Fisher et al. 2010) assessed leg-hold traps for possums, the avicides alpahchloralose and DRC-1339 for pest birds and the piscicide rotenone for pest fish. Literature review, data collation and application of the Sharp & Saunders (2008) framework were used to produce relative welfare impact rankings through expert opinion rather than a panel assessment.

Table 1: List of vertebrate toxic agents (VTAs) used in New Zealand and the relevant target and non-target mammalian species for which literature was reviewed. Primary non-targets are those that may ingest baits containing VTAs. Secondary non-targets are those that may ingest VTAs by consuming other poisoned animals.

		Non-target species	
VTA	Target species	Primary	Secondary
Sodium fluoroacetate (1080)	Possum	Pig	Ferret
	Rabbit	Deer	Stoat
	Rat	Wallaby	
	Cat		
Cyanide	Possum		
(NaCN and KCN)	Wallaby		
Cholecalciferol	Possum		
Brodifacoum	Possum		Ferret
	Rat		Stoat
	Mouse		Cat
Diphacinone	Rat		
	Mouse		
	Ferret		
Pindone	Possum		
	Rat		
	Rabbit		
Phosphorus	Possum	Pig	
	Rabbit		
Zinc phosphide*	Possum		
p-aminopropriophenone (PAPP)*	Stoat		
	Cat		
Sodium nitrite*	Pig		

* VTA not yet registered in NZ

Approach

For all assessments (Parts 1–3), literature searches were undertaken to compile reference information and to identify product registration, use patterns and 'best practice' application of the pest control tools in New Zealand. In particular, specific information about each VTA or control tool was sought regarding:

- Welfare impacts or humaneness either by review or research on captive animals
- The mode of toxic action or general toxic effects on mammals in general, including information on behavioural, physiological, and pathological responses
- Description of effects, time to death, pathology or other information relevant to evaluating welfare effects
- Toxic effects on humans

For assessment of the VTAs in mammals (Part 1), the review panel applied the principles of the Australian framework to avoid introducing personal bias and to ensure that rankings generated (number rankings using Part A of the framework, or letter rankings using Part B of the framework) would be broadly comparable with other assessments derived using the framework. Where possible, 'best practice' application of each control tool was described, and scores were based on the assumption of best practice being used. Before conducting the welfare assessment, the panel agreed on a number of modifications and qualifications to applying the Australian framework, largely to improve the suitability of the model for assessing VTAs. These modifications are described fully in Part 1, but in summary included scoring functional impairments only to the point of loss of consciousness to allow resolution of different lethal methods and the use of Part A of the framework described by Sharp & Saunders (2008) to assess the welfare impacts of VTAs. The scoring matrix used to derive number scores for Part A is shown in Appendix 1.

The panel decided that the Part A scoring process, applied to the actual mode of action of a VTA in a nominated species, allowed a more detailed consideration of available information about VTAs with respect to their different modes of toxic action and different welfare impacts across species. Consideration of the specific effects of a VTA across mammal species, using the 'domains' described in Part A, allowed a greater differentiation and a more transparent explanation of how rankings for VTAs were derived. The combined panel scores were also allocated 'confidence' ratings, as an indication of the degree of variance between the scores given by individual panel members – there was generally greater variance in the scores allocated when there was little information available about the effects of a particular VTA.

The panel also suggested several amendments to the terminology of the original process to better reflect current usage in animal welfare science. These included: replacing 'humaneness' with 'animal welfare impact' as truly humane control methods are rare; clearly differentiating impacts in domain 3 (functional impairment/pathology) from impacts in domain 5 (negative affective states associated with impairment/pathology); the addition of at least breathlessness, nausea, lethargy, and dizziness to the list of possible negative affective experiences, and clarification of the term 'distress'; and clarification of impacts associated with CNS dysfunction and level of consciousness in domain 3.

For the assessment of in-burrow rabbit control methods (Part 2), and the assessment of leg-hold traps, the avicides alphachloralose and DRC-1339 and the piscicide rotenone (Part 3), a phone and email survey of some product registrants was also undertaken in order to quantify the extent of use or sales of particular control tools. Compiled literature was used as the basis for an expert opinion (rather than review panel) assessment of the welfare impacts of these control tools, applying both Parts A and B of the Australian framework to evaluation of the welfare impacts of poisoning (where relevant), with the modifications/qualifications to the framework described in Part 1. This was to allow future comparisons with other assessments of the same control tools completed using the Australian framework.

Outcomes

The overall welfare impact scores determined in Part 1 for VTAs and animal species are summarised in Table 2.

Part 1: VTAs in mammals and kill traps

- Kill traps used in New Zealand are already subject to a guideline for assessment of humaneness (NAWAC 2005), such that assessment using the Australian framework was considered unnecessary and potentially confusing.

- Cyanide when used for killing possums or wallabies has the lowest relative welfare impact of the VTAs with animals losing consciousness within minutes of a lethal exposure.

- The welfare impacts of 1080 were intermediate impact scores for all species considered. The suggestion that the neural effects of 1080 result in a progressive decline in the level of consciousness, and therefore reduced durations of negative experiences, requires further research.

- Anticoagulant poisoning has the highest relative impact on welfare of mammal species.

- For those VTAs in the development pipeline but not as yet registered for use in New Zealand, relative welfare impacts were intermediate relative to the registered VTAs assessed, noting that the panel produced a range of different scores. This was due to uncertainty (lack of information) about the welfare impacts of the corrosive metabolite of zinc phosphide in possums. Further research is required to better characterise the welfare impacts of zinc phosphide (possums), PAPP (stoats and feral cats) and sodium nitrite (pigs).

- For possums, cyanide has the least welfare impact while the anticoagulants and cholecalciferol have the highest relative impacts. Phosphorus, 1080 and zinc phosphide produce intermediate welfare impacts relative to the other VTAs assessed.

- For rodents, anticoagulants have the highest relative impacts while 1080 had intermediate impacts. The range of welfare impact scores assigned by the panel indicated relatively low confidence in the data from which scores were generated.

- For stoats, ferrets and feral cats anticoagulants have the highest welfare impacts, with 1080 producing intermediate impacts. Again, there was relatively low confidence in the data on which these scores were generated. The wide range of grades assigned for PAPP for carnivore species reflected the panel's uncertainty as to the animals' experiences of breathlessness and anoxic headaches before cerebral anoxia caused reduction or loss of consciousness.

- For rabbits, 1080 had intermediate impacts and pindone very high welfare impacts. There was insufficient information to evaluate the impacts of phosphorus on rabbits.

- For pigs, phosphorus and 1080 have intermediate welfare impacts. Scores assigned to sodium nitrite varied considerably between low and intermediate, reflecting the panel's uncertainty as to the animals' experiences of breathlessness and anoxic headaches before cerebral anoxia caused reduction or loss of consciousness.

- For deer and wallabies, the impacts of 1080 were considered to be less severe than for other species. However, this may reflect the stoic nature of some herbivores such as deer, and the paucity of information on which to base evaluation. There was insufficient information available to evaluate cyanide's impacts on wallabies.

Part 2: In-burrow rabbit control methods

- The overall score for chloropicrin was 5F, based on a combination of moderate to extreme negative welfare impacts (inhalation of toxic vapour causing extreme irritation and respiratory distress in rabbits) over a duration of minutes. The availability of recently published, formal evaluations of the humaneness of chloropicrin relative to other rabbit fumigants gave a high certainty to this assessment.

- The overall score for phosphine (the gas generated by magnesium phosphide) was 3D, based on a combination of mild to moderate negative welfare impacts (respiratory irritation in rabbits) over a duration of minutes. The availability of recently published, formal evaluations of the humaneness of phosphine relative to other rabbit fumigants gave a high certainty to this assessment.

- The overall score for the 'Rodenator' was 5E, based on a combination of welfare impacts ranging from none to extreme, with potentially extreme negative welfare impacts over a duration of immediate to seconds (consistent with close proximity of rabbits to a large explosion and very high temperatures). However, there was high uncertainty in this assessment, due to the lack of published scientific information about the range of effects on rabbits of this control method.

Part 3: Leg-hold traps, rotenone, alphachloralose and DRC-1339

- Based on the impacts on possums from Victor No. 1 padded and unpadded leg-hold traps and the duration of exposure to these impacts, the overall welfare score was 5E based on a combination of moderate domain impacts over a duration of hours (Part A) and extreme negative welfare impacts over a duration of seconds (Part B).

- For the use of alphachloralose to control pest birds, the overall welfare score was 5C, based on a combination of moderate domain impacts and negative welfare impacts over a duration of hours.

- For the use of DRC-1339 to control rooks or starlings, the overall welfare score assigned using the model was 7G, based on a combination of severe negative welfare impacts over a duration of hours. A high level of uncertainty in this score was noted, due to information gaps about some of the effects of DRC-1339 in birds.

- The overall welfare score for the use of rotenone for pest fish control was 5E, based on a combination of moderate negative welfare impacts over a duration of hours.

Conclusions and Recommendations

- Overall, the welfare impact assessment framework developed by Sharp and Saunders (2008) is highly applicable to the evaluation of the welfare impacts of New Zealand pest control tools.

- Assessments and assignation of scores using the Australian framework assumed 'best practice' application of a lethal control tool for a nominated pest species. Thus welfare impacts potentially associated with poor application or efficacy of a control tool – e.g. sublethal exposure to VTAs, failure to recover birds affected by alphachloralose for euthanasia – were not evaluated here but could be in future.

- Variable amounts of information were available in relation to the effects on mammals, birds and fish of the respective lethal control tools assessed, which affected the certainty

with which scores could be allocated to domain impacts. A number of particular information gaps were identified, addressing which would improve the robustness of relative assessments of humaneness.

- Priority information gaps for control tools, with regard to the likely number of animals potentially affected are:
  - In mammal species other than possums, for many VTAs data on the time between onset of symptoms and loss of consciousness (duration of negative experiences) during toxicosis are lacking, as is information on the level of consciousness during critical events, e.g. convulsions, respiratory compromise. Those combinations where data are lacking are shown as NDD in Table 2 below.

  - Assessments of welfare impacts of various leg-hold trap types (excepting Victor No. 1) for possums are lacking, as are assessments of the effects of leg-hold traps in general on feral cats and ferrets.

- Formal surveys to better determine the extent of use of chloropicrin and the 'Rodenator' for in-burrow rabbit control in New Zealand, and use of alphachloralose and DRC-1339 as avicides, would assist in estimating the number of animals potentially affected by control methods.

References

NAWAC 2005. National Animal Welfare Advisory Committee guideline http://www.biosecurity.govt.nz/animal-welfare/nawac/policies/guideline09.htm (accessed 4 November 2010).

Sharp T, Saunders G 2008. A model for assessing the relative humaneness of pest animal control methods. Canberra, ACT, Australian Government Department of Agriculture, Fisheries and Forestry.

Table 2: Welfare impact scores derived by review panel for VTAs in target pest mammals, except where NT (non-target) is specified. Using the matrix defined in Part A of the Australian framework, numerical scores from 1 to 8 were assigned as a combination of welfare impact scores and the duration of the welfare impact(s). The higher the score, the higher the relative overall welfare impact. Shaded spaces indicate no assessment was made for the species–VTA combination, NDD = no direct data for VTA–species combination (information gap), *indicates a VTA not yet registered for use in New Zealand.

Species	1080	Cyanide	Cholecalciferol	Brodifacoum	Diphacinone	Pindone	Phosphorus	Zinc phosphide*	PAPP*	Sodium nitrite*
Possum	6	4	8	8		7.5	6	6		
Rodents	6			7.5	NDD	NDD				
Stoat	6.5 (NT)			NDD/NT					5.5	
Ferret	6.5 (NT)			7.5 (NT)	NDD				NDD/NT	
Feral cat	6.5			7.5 (NT)					5.5	
Rabbit	6					8	NDD			
Pig	6.5 (NT)						7			5.5
Deer	NDD/NT									
Wallaby	5.5 (NT)	NDD								

1. Vertebrate toxic agents and kill traps in mammal species

by N.J. Beausoleil (AWSBC, Massey University), P. Fisher (Landcare Research), B. Warburton (Landcare Research) and D.J. Mellor (AWSBC, Massey University)

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1.1. ABSTRACT

Increasingly, information on the animal welfare impacts of pest control methods is required to inform the decisions of operators and policy makers. To date, there has been no cohesive evaluation of welfare impacts associated with Vertebrate Toxic Agents (VTAs) used in NZ. The purpose of this project was to apply a recently developed Australian welfare assessment framework to produce a ranking of the relative welfare impacts of vertebrate toxic agents on their mammalian targets and other non-target mammals. A selection of devices (VTAs and kill traps) and species were agreed upon, and relevant data were collated to provide reference material for the assessment and to identify gaps in existing knowledge. This review was used by a panel to apply the framework. As kill traps used in NZ are already tested according to a NAWAC guideline, we decided that assessment using the framework was unnecessary and potentially confusing. For all species exposed (possums, rodents, carnivores, rabbits), lethal anticoagulant poisoning has the highest relative impact on welfare. In contrast, cyanide as used for possum control has the lowest relative welfare impact. In general, 1080 and phosphorus produce intermediate impacts. For VTAs currently unregistered in NZ, opinion on the panel was divided. We were uncertain about the impacts of the corrosive metabolite of zinc phosphide in possums. For PAPP (carnivores) and sodium nitrite (pigs), experiences of breathlessness before loss of consciousness are poorly understood. In general, there are insufficient data available to assess the impacts of VTAs on the welfare of herbivores such as wallabies and deer. For many VTAs, there is insufficient information to conduct comprehensive analyses of welfare impacts. In particular, data on the time between onset of symptoms and loss of consciousness (duration of negative experiences) are lacking, as is information on the level of consciousness during critical events, e.g. convulsions, respiratory compromise. The report should be used to identify specific directions and methods for generating such information in future research. Overall, the framework was considered suitable for the purpose, after minor modifications were made. This report evaluated only effects of VTAs delivered using ‘best practice’; welfare impacts associated with sub-optimal/lethal dosing should also be assessed. In addition, there remain a range of other VTAs and control methods that could be assessed using the Australian welfare assessment framework.

Keywords: 1080, animal welfare impacts, anticoagulants, cyanide, kill traps, PAPP, pest control, phosphorus, sodium nitrite, vertebrate toxic agents, zinc phosphide.

1.2. BACKGROUND

The welfare of wild animals subject to human interference is receiving increasing attention in New Zealand and overseas (Sherley 2007). This includes the welfare of the target pests and also of non-target species. The growing body of literature on the welfare impacts of tools used to control mammal pests in New Zealand represents an increasing knowledge base for gauging the acceptability of different methods on the basis of animal welfare.

However, gaps remain in the available information regarding the welfare impacts of pest control methods. In particular, research-based data relating to the impacts of vertebrate toxic agents (VTAs) are lacking. This lack of information means that it is currently difficult to determine policy directions for the use of VTAs, or for a pest control operator to choose an optimal tool on the basis of overall animal welfare impact.

To date, there has been no systematic evaluation of the animal welfare impacts associated with various pest control tools used in New Zealand. This is partly explained by the absence of a suitable framework to undertake this type of evaluation; however, recently, such a framework has been developed in Australia (Sharp & Saunders 2008). The Australian framework can be used to produce relative impact scores, and therefore rankings, for various lethal and non-lethal pest control methods such as shooting, trapping, mustering, fencing and poison-baiting (VTAs).

The purpose of the project was to apply the Australian framework to develop a ranking of the relative animal welfare impacts of a nominated selection of VTAs currently used (or pending registration for use) in New Zealand. Using the Australian framework, rankings were developed for agreed target pest species (possums, rodents, mustelids and feral cats) and also for a range of introduced mammal species known to be affected by different VTAs as non-target species. A range of kill trap types was also nominated for assessment. Relevant data from literature, pest control practitioners, animal welfare researchers and other current information were collated to provide reference material for assigning rankings and to identify where information gaps exist regarding specific pest control tools.

1.3. OBJECTIVES

- To collate relevant literature from New Zealand, Australia, and elsewhere, if available, and review its applicability and utility for assessing the animal welfare impacts on the agreed range of species and devices/tools, using the welfare assessment framework developed in Australia (Sharp & Saunders 2008);
- To apply the Australian framework to evaluate the relative animal welfare impacts of the nominated pest control tools used in New Zealand;
- To identify current gaps in the information necessary to assess relative welfare impacts of pest control tools to guide future research priorities.

1.3.1 Animal welfare and assessment of welfare impacts

Welfare is a state within an animal and most directly relates to what the animal experiences (Mellor et al. 2009). Briefly, welfare is considered here to be the integrated balance of all sensory inputs to the animal's brain that are cognitively processed and experienced as emotions or feelings (Mellor 2010). Sensory mechanisms continuously scan the internal functional state and external environment of the animal. These sensory inputs are then processed and interpreted within a context relevant to the species and individual animal. The

integrated cognitive and emotional outcomes of this process are reflected in the individual's welfare state, which can range from very bad to extremely good (Mellor 2010).

In accordance with this conception of animal welfare, current approaches to assessing the impacts of events, situations or procedures on welfare are based on the 'Five Domains of Potential Welfare Compromise' model developed by Mellor and Reid (1994). This model provides a means of clearly separating physical or functional impacts of the animal from the emotional or affective experiences, mental states or feelings that ultimately determine its welfare or well-being.

The five domains of potential compromise are summarized in Figure 1.1 below.

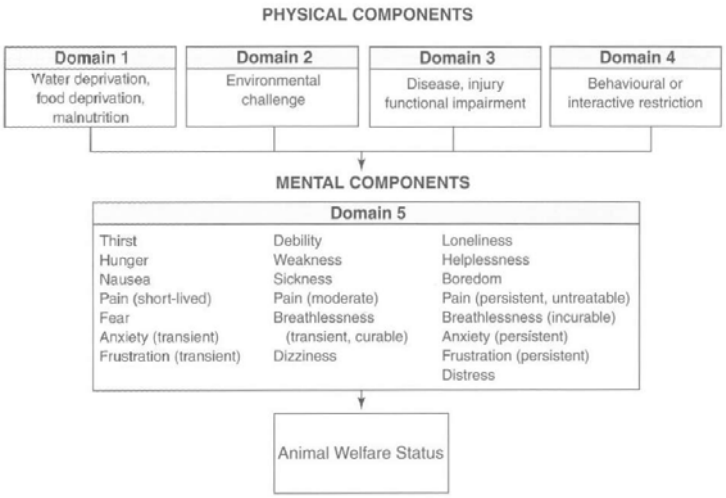

Figure 1.1 Domains of potential welfare compromise divided broadly into physical and mental components. From Mellor et al. (2009).

In assessing the welfare of individual humans, we rely similarly on behavioural, physiological and pathological indicators; however, most importantly, we have some access, although still indirect, to information about the affective experiences of people, i.e. they can *tell* us how they feel. For non-human animals, this latter information is unavailable. We are forced to extrapolate affective experiences from observable changes in behaviour, physiology and functional state. In addition, for certain groups of animals, we assume that it is appropriate to extrapolate their affective experiences from the reports of humans in similar situations. It is now generally accepted that adult mammals have the capacity to experience both positive and negative affective states relevant to welfare.

Finally, it is important to understand that, in order to experience any affective state, an animal must be conscious (Mellor et al. 2009). Consciousness, or awareness, is linked to wakefulness and is considered to be present when perception of, and response to, a stimulus involves the cerebral cortex (Mellor & Diesch 2007). It follows that an animal must be conscious to experience any welfare state, good or bad; there can be no compromise of animal welfare, no welfare impact, while an animal is unconscious. Events occurring during unconsciousness (e.g. during sleep or unconscious convulsions) are only relevant to welfare status if, and when, consciousness is recovered.

1.4. METHODS

1.4.1 Literature review (Step 1)

A selection of New Zealand pest control tools (vertebrate toxic agents and kill traps) and target and non-target species (mammals only) were agreed upon by MAFBNZ for initial consideration of relative welfare impacts using the Australian framework (Tables 1.1 and 1.2). The relevant references from a review of the literature were summarized as background reading and reference material for a panel of six people (see Step 2 below) to undertake the scoring process and produce a ranking of relative animal welfare impacts.

The Sharp & Saunders (2008) framework for assessing the relative welfare impacts of pest control tools involves two steps – Part A assesses overall welfare impact based on the five domains, and Part B (for lethal control methods) is an assessment of the killing technique (see below for Modifications of the Australian framework for evaluation of New Zealand pest control tools). The process requires the references and evidence used to 'score' parts A (number score) and B (letter score) are recorded. The scoring matrices described by Sharp & Saunders (2008) for Parts A and B are shown in Appendix 1.

The documents listed in section 6.1 of the original RFP and other relevant published papers and unpublished reports were reviewed and assessed for relevance to welfare impacts of the control tools. Articles relevant to each vertebrate toxic agent (Table 1.1 lists those covered by this review) were selected for inclusion in the following literature list where they addressed one of more of these areas:

1. Evaluation of welfare impacts or humaneness either by review or research on captive animals;

2. Description of the mode of toxic action or general toxic effects in mammals including information on behavioural, physiological, and pathological responses;

3. Studies of the VTA or kill trap in one of the nominated target or non-target mammal species that include description of effects, time to death, pathology or other information relevant to evaluating welfare effects;

4. Articles describing the toxic effects of a VTA on humans.

Internet searches and email contact with overseas wildlife research and management agencies (e.g. The Food and Environment Research Agency, FERA, York; National Wildlife Research Centre, Ft Collins, CO, USA) were undertaken to identify other procedures being developed or used for assessing animal welfare impacts of pest control tools, or other published assessments of welfare. Apart from some EU/UK focus on the humaneness of anticoagulant rodenticides (e.g. Guiding Principles for the Humane Control of Rats and Mice http://www.ufaw.org.uk/rodents.php), the majority of research relating to formal assessments of the relative humaneness of VTAs appears to have been undertaken in New Zealand and Australia.

The literature lists summarized for each VTA (see Results) comprise those articles considered most relevant and/or most recent (where similar information was available from several publications). Summaries of 'mode of toxic action', effects on particular mammal species, and reports of human poisoning have been provided in each section. The references reviewed

and selected include international literature, especially for human poisoning cases in general and the anticoagulant VTAs.

1.4.2 Assessment of animal welfare impacts

Panel members were provided with the summary of relevant literature on the agreed pest control tools and species generated in Step 1 (above) before the panel met. Members familiarized themselves with this information and then met as a group to undertake the assessment of welfare impacts according to the Australian framework. The panel members were:

- Professor David Mellor, BSc(Hons), PhD, HonAssocRCVS, ONZM, Professor of Applied Physiology, Professor of Animal Welfare Science and Bioethics, Co-Director of Animal Welfare Science and Bioethics Centre, Massey University
- Dr Ngaio Beausoleil, BSc, PGCertSci, PhD, Lecturer in Physiology, Associate of Animal Welfare Science and Bioethics Centre, Massey University
- Dr Kate Littin, BSc, MSc, PhD, Senior Advisor, MAF Animal Welfare Directorate
- Dr Penny Fisher, BSc, MApplSc, PhD, Research leader, Pest Control Technologies, Landcare Research
- Mr Bruce Warburton, BSc, DipSc, MSc, Research leader, Pest Control Technologies, Landcare, Past member of NAWAC
- Dr Rosalind Dalefield, BVSc, PhD, DABVT, DABT.
- (Absent) Dr Phil Cowan, BSc, PhD, Science leader, Pest Control Technologies, Landcare, Current member of NAWAC.

In applying the Australian framework, the panel agreed to follow its principles to avoid introducing personal bias due to experience and to ensure that rankings generated using the framework were broadly comparable between countries/groups. Scores were based on the assumption of 'best practice' in delivering the pest control tools. In other words, we assumed that every animal would receive the lethal dose of the VTA.

Before conducting the welfare assessment, the panel agreed on a number of modifications and qualifications to the Australian framework, largely to improve the suitability of the framework for assessing VTAs. These modifications and the rationale for the changes are briefly summarized below. Further details and suggestions for updating or clarifying the terminology of the original framework are presented in the Discussion section below.

1. Score impacts in domain 3 (functional impairment/pathology) only to point of loss of consciousness

Lethal doses of VTAs all have the same endpoint of the ultimate functional impairment: death. If each VTA was scored to this maximum level of impairment, the framework would provide no resolution between the different toxic compounds – all agents would receive a maximum score for part A (8). Therefore, to provide resolution between VTAs, and potentially between species for the same VTA, the panel scored impacts in domain 3 up to the point at which the animal lost consciousness.

2. Clear differentiation of functional impairment (domain 3) and negative affective experience (domain 5)

In the original framework, there is some mixing of terms reflecting functional impairment/injury/pathology (domain 3) and terms reflecting negative affective experiences associated with those impairments (domain 5). We consider terms such as sickness, lethargy and breathlessness, currently included in Box 3 (domain 3), to reflect the negative affective experiences, mental states, emotions or feelings associated with functional impairments/pathology rather than the impairments themselves. Therefore, for domain 3, the functional impairment, injury or pathology itself was described and the associated affective experience/feeling was described and scored separately for domain 5.

As we currently have no way to measure subjective mental states directly, we have based our scores for domain 5 on observable impacts in the other domains (particularly domain 3) and on the reports of affective experiences of human poisoning victims. Our confidence in each of these extrapolations is indicated in the Results tables as follows:

> *0 = no animal data available, possible negative affective experiences inferred from human poisoning reports;*
>
> *1 = low confidence, more specific/detailed animal data required;*
>
> *2 = moderate confidence, more specific/detailed animal data needed to clarify;*
>
> *3 = high confidence*

3. Expand range of negative affective states described in domain 5

In the Australian framework, domain 5 describes only anxiety, fear, pain, distress, sickness and greater than normal thirst/hunger. In our analysis, we also considered the potential for at least the following negative affective experiences: breathlessness, nausea, lethargy/weakness, and dizziness.

4. Assess VTAs using Part A of the Australian framework

Development of the framework saw the inclusion of Part A to assess the ‘impact on the animal prior to the action that causes death’, with Part B also assessed for lethal control methods to assess the ‘actual mode of death’ and the 'extent and duration of suffering caused'. This was to enable comparison of a wider range of pest control methods than VTAs alone e.g. mustering, aerial shooting , leg hold traps followed by shooting (T. Sharp, pers. comm.). In the case of bait-delivered VTAs as lethal control methods, Part A could be considered as the period before the bait was taken, where no impacts on Domains 1 to 5 would normally be expected,

The panel noted that for the VTAs, completion of Part B simply produced repetitions of Part A scores (except with letters rather than numbers), providing no additional information on welfare impacts. In the case of VTAs, the ‘control tool’ and the method that causes death were considered one and the same. For this reason, and because only VTAs were assessed in this report, the panel decided that Part A scoring applied to the actual mode of action of a VTA in a nominated species allowed a more detailed consideration of available information to describe of the relative welfare impacts of the VTAs. Consideration of the specific effects

of a VTA across mammal species, using the Part A domains, allowed a greater differentiation and more transparent explanation of how rankings were derived.

5. Defer assessment of the relative welfare impacts of kill traps to existing NZ guideline

Various 'live capture' traps are used in both Australia and New Zealand for pest control. When live traps are used, the trapped animal is restrained by the device until a human operator attends to release or euthanase the animal. In contrast, kill traps are designed to kill the animal without involvement of a human operator. There appears to be no (or very limited use) of kill traps for pest animal control in Australia; however, traps designed to kill a target pest animal as the direct result of capture are widely used New Zealand and there already exists a welfare-related guideline for such devices.

The National Animal Welfare Advisory Committee (NAWAC) developed a guideline to provide an objective basis for assessing the welfare-related performance of kill traps and to formalize how kill traps are to be tested, along with criteria for determining whether they have satisfactory performance (http://www.biosecurity.govt.nz/animal-welfare/nawac/policies/guideline09.htm). To assess whether a kill trap is acceptable in terms of its ability to kill quickly, the time to loss of brainstem reflex in the captured animal is measured. To meet the NAWAC guideline, 10 of 10, or 13 of 15 target animals must be rendered irreversibly unconscious within 3 min of capture. Consciousness is determined using the palpebral (blinking) reflex which is absent when the animal has lost consciousness.

The NAWAC guideline provides a standard for humaneness in the case of kill traps used in New Zealand, such that assessment of kill traps using the Australian framework was not considered useful, and may have even caused confusion. Because 'best practice' in this instance represents the use of kill-traps that have met NAWAC guidelines for a nominated target species, all such trap/species combinations were likely to have been allocated the same score by the panel using the Australian framework. In addition, providing new scores for kill traps already assessed using the NAWAC guideline may confuse policy makers considering the status of various trap types available in New Zealand, as well as pest control operators wanting to select traps recognized as acceptable on animal welfare grounds.

Information available from assessment against the NAWAC guideline was compiled (Appendix 1.1) for kill-trap types and nominated target pest species (Table 1.2). For kill traps that did not meet the NAWAC guideline (Table 1.2) or have not been assessed, there is very little information available to inform welfare impact scoring decisions, in terms of the duration and extent of impacts.

1.5. RESULTS

1.5.1 Literature Review

Tables 1.1 and 1.2 details the vertebrate toxic agents (VTAs), types of kill traps, and species assessed.

Table 1.1: List of vertebrate toxic agents (VTAs) used in New Zealand, bait formulations used, and the relevant target and non-target mammalian species for which literature was assembled to be used in the assessment of relative animal welfare impacts. Primary non-targets are those that may ingest baits containing VTAs. Secondary non-targets are those that may ingest VTAs by consuming other poisoned animals.

			Non-target species	
VTA	Bait formulations	Target species	Primary	Secondary
Sodium fluoroacetate (1080)	0.15% & 0.08% cereal & carrot baits 0.06% & 0.04% cereal baits 0.1% ACP cat	Possum Rabbit Rat Cat	Pig Deer Wallaby	Ferret Stoat
Cyanide (NaCN and KCN)	Trapper's paste ACP paste Cyanara paste Feratox pellets	Possum Wallaby		
Cholecalciferol	Feracol Decal Nopossum	Possum		
Brodifacoum	Talon Pestoff	Possum Rat Mouse		Ferret Stoat Cat
Diphacinone	Ratabate Ditrac Pestoff ferret paste	Rat Mouse Ferret		
Pindone	PMS pellets possums/rats PMS pellets rabbits Liquid for carrot/oats	Possum Rat Rabbit		
Phosphorus	ACP single strength paste ACP double strength paste	Possum Rabbit	Pig	
Zinc phosphide*		Possum		
p-aminopropriophenone (PAPP)*		Stoat Cat		
Sodium nitrite*		Pig		

* VTA not yet registered in NZ so formulation details and active concentrations are not currently specified

Table 1.2: Kill traps used in New Zealand, for which animal welfare impacts were assessed. Target species for assessment of kill traps were: possum, ferret, stoat, ship rat, Norway rat.

Trap type/model	Species	NAWAC Pass/Fail
DOC 150	Norway rat	Pass
DOC 150	Stoat	Pass
DOC 200	Stoat	Pass
DOC 200	Norway rat	Pass
DOC 250	Ship rat	Pass
DOC 250	Stoat	Pass
DOC 250	Norway rat	Pass
DOC 250	Ferret	Pass
Fenn Mk4 (new)	Stoat	Fail
Fenn Mk6 (new)	Stoat	Fail
Nooski	Norway rat	Pass
Possum Master	Possum	Fail
Possum Master	Ferret	Fail
Sentinel	Possum	Pass
SetnForget	Ferret	Fail
SetnForget	Possum	Pass
S&F	Ferret	Fail
Timms	Possum	Fail
Timms	Ferret	Fail
Timms Tunnel	Ferret	Fail
Victor Professional Snapback	Norway rat	Pass
Warrior	Possum	Pass
Warrior	Ferret	Fail

1.5.2 Assessment of animal welfare impacts

Vertebrate Toxic Agents (VTAs)

For ease of interpretation, the outcomes of the panel's assessments have been tabulated by single species or species groupings of mammals introduced to New Zealand:

- Possum
- Rodents (Norway rat, ship rat, house mouse)
- Carnivores (stoat, ferret, cat)
- Rabbit
- Pig
- Deer and wallabies

It should be noted that most species assessed are recognized pests, targeted for control in New Zealand by the nominated vertebrate toxic agent, e.g. possums and 1080. However in some instances, the assessment was undertaken because a mammal species was recognized as a significant non-target, potentially affected by use of a VTA to control other species, e.g. stoats and 1080, deer and 1080. Species/VTA combinations that are 'non target' are noted in the tables below.

In each table, the median score and range of scores from six panelists are given for overall impact and for each domain of potential animal welfare compromise:

- Domain 1 = Water/food deprivation, malnutrition
- Domain 2 = Environmental challenge
- Domain 3 = Injury, disease, functional impairment
- Domain 4 = Behavioural/interactive restriction
- Domain 5 = Negative affective experiences.

Within each domain, impact was scored as No, Mild, Moderate, Severe or Extreme. For each panelist, the Overall Impact reflected the highest scores assigned for a domain (usually domain 5). The Overall Grade was a number from 1 to 8, as determined by a matrix for Part A of the Sharp & Saunders (2008) framework. The Overall Grade integrated the Overall Impact score and the duration for which those impacts were experienced, with a higher number indicating a higher welfare impact. For each species/VTA combination, the median score and range of scores from the six panelists are provided for Overall Grade. NDD = No Direct Data, indicating that impacts within the domain (1, 2 and 4) have been inferred from other known effects. Confidence level (C) were also assigned to Overall Grades, to indicate the degree of confidence the panel had in the data on which the scores were based:

> *0 = no animal data available, possible negative affective experiences inferred from human poisoning reports;*
>
> *1 = low confidence, more specific/detailed animal data required;*
>
> *2 = moderate confidence, more specific/detailed animal data would clarify;*
>
> *3 = high confidence.*

Details of evidence and references used to decide on scores for each VTA by the panelists are presented after each table.

Table 1.3: Relative welfare impacts of VTAs affecting possums (*Trichosurus vulpecula*) in New Zealand. In all cases, possums are the species targeted for control by the VTA used. Zinc phosphide is undergoing registration as a VTA for possum control, but is currently not available for operational use. Within each domain, impact is scored as No, Mild, Moderate, Severe or Extreme.

NDD = No Direct Data, indicating that impacts within the domain (1, 2 and 4) have been inferred from other known effects.

	Domain of potential welfare compromise											
Agent	1	2	3	4	5	C	Overall impact	C	Duration	C	Overall grade	C
1080	Mild (*No – Mod*) NDD	Mild (*No – Mild*) NDD	Severe (*Mod – Ex*)	Mild (*No – Mod*) NDD	Severe (*Mod – Ex*) Nausea Lethargy/weakness Pain Sickness Breathlessness Dizziness Anxiety/fear	 3 3 2 1 0 0 0	Severe (*Mod – Ex*)	2	Hours	1	6 (*5 – 7*)	1
Cyanide	No	No	Mild (*No – Mod*)	No (*No – Mild*)	Mild – Moderate (*No – Mod*) Breathlessness Sickness Lethargy/weakness Anxiety Headache Dizziness Nausea	 3 0 0 0 0 0 0	Moderate (*Mild – Mod*)	2	Minutes	3	4 (*3.5 – 4*)	2
Cholecalciferol	Extreme (*Sev – Ex*)	No (*No – Mild*) NDD	Extreme (*Sev – Ex*)	Moderate (*No – Sev*) NDD	Extreme (*Sev – Ex*) Breathlessness Pain/discomfort Lethargy/weakness Sickness Nausea	 3 2 2 1 0	Extreme (*Sev – Ex*)	2	Days	3	8 (*7 – 8*)	2

					Hunger/thirst	0					
Brodifacoum	Mild (*Mild – Mod*)	Mild (*No – Mod*)	Severe (*Mod – Ex*)	Mild (*No – Sev*) NDD	Severe – Extreme (*Mod – Ex*) Lethargy/weakness Pain Sickness Breathlessness Nausea Hunger/thirst Dizziness	 3 3 2 1 0 0 0	Severe – Extreme (*Mod – Ex*)	3	Days – Weeks	3	8 (*7 – 8*)	3
Pindone	Moderate (*Mild – Mod*) NDD	Mild (*No – Mild*) NDD	Severe (*Mod – Sev*)	No (*No – Mod*) NDD	Severe (*Mod – Ex*) Sickness Lethargy/weakness Nausea Hunger/thirst	 0 0 0 0	Severe (*Mod – Ex*)	1	Days - Weeks	1	7.5 (*7 – 8*)	1
Phosphorus	Mild (*No – Mod*) NDD	Mild (*No – mild*) NDD	Moderate – Severe (*Mild – Sev*)	Mild (*Mild – Mod*) NDD	Severe (*Mod – Sev*) Pain Nausea Sickness Lethargy/weakness Anxiety	 3 2 1 0 0	Severe (*Mod – Sev*)	1	Hours (*Hours – Days*)	2	6 (*5 – 7*)	1
Zinc phosphide	Mild (*No – Mild*)	Mild (*No – Mild*) NDD	Severe (*Sev – Ex*)	Mild (*No – Mild*) NDD	Severe (*Sev – Ex*) Nausea Pain Breathlessness Sickness Lethargy/weakness Thirst Anxiety	 3 3 3 2 2 0 0	Severe (*Sev – Ex*)	3	Hours	3	6 (*6 – 7*)	3

1.5.2.1. Sodium fluoroacetate (1080) for possums

Basis of welfare assessment and scoring (summary)

No direct data are available for assessing impacts in domains 1, 2 or 4. Scores for food/water deprivation are based on the duration of sickness behaviour and lethargy/weakness observed as well as likely nausea, although it is unknown whether poisoned possums experience hunger/thirst. Likewise, potential environmental challenge and behavioural/interactive alterations are based on the quality and duration of altered behaviour (9.5 hrs). Scores for domain 3 (functional impairment) are based on observations of retching/vomiting, abnormal postures (which may be indicative of sickness or pain), incoordination and spasms/tremors/seizures. Whether or not animals are conscious during or after these neural events is unknown. Animals conscious during such events or those that recover consciousness afterwards may experience pain, breathlessness and anxiety/fear.

Conscious experience of nausea, lethargy/weakness, sickness and pain seem likely, although it has been suggested (P. Fisher, unpubl. data) that there is a progressive reduction in awareness which could reduce the duration of such negative affective experiences. Humans poisoned with 1080 report epigastric/abdominal pain, respiratory distress (breathlessness), nausea, dizziness and anxiety, but there are no available data with which to assess these experiences in animals.

Information required

Information on level of consciousness at various times/events after lethal exposure of possums to 1080 is required to determine the quality and duration of negative affective experiences. Information on time to loss of behavioural reflexes, normal EEG and evoked potentials after dosing is also needed. Such measures should be used to evaluate level of consciousness immediately before and after spasms/tremors/seizures. Studies involving administration of analgesic and anti-anxiety drugs may shed light on animals' experiences of pain and anxiety during 1080 toxicosis. More specific information on respiratory compromise is required to assess potential breathlessness. Direct information on feeding/drinking and social behaviour, and potential for environmental exposure, is required to assess welfare impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

Cooper et al. (2007) stated that 1080-poisoned animals die in considerable pain. A recent review of the speed and mode of toxic action, appearance and behaviour of affected animals, experiences of human victims, long-term effect on survivors, and welfare risk to non-target animals concluded that sodium fluoroacetate should not be considered a humane poison (Sherley 2007). This conclusion has met rebuttal from Twigg & Parker (2010) who suggest that the time from ingestion of 1080 to death is not a reliable indicator of its humaneness. Further, as 1080 impairs neurological function and his impairment includes some pain receptors, they suggest it is difficult to interpret the behaviour of 1080-affected animals, or to assess their ability to experience discomfort and pain.

Mode of toxic action:

In an organism, fluoroacetate (the toxic principle of 1080) undergoes a series of metabolic conversions resulting in the synthesis of fluorocitrate – this process was named 'lethal synthesis'. There is a latent period between the time fluoroacetate is ingested and signs of poisoning first appearing (at least 30 min to 3 hrs in mammals), which presumably is the time required for fluoroacetate to be absorbed, to penetrate cells, be converted into fluorocitrate, and then to begin to disrupt cellular processes. Synthesis of fluorocitrate in the mitochondria competitively inhibits the tricarboxylic acid cycle (TCA) enzyme aconitase and also inhibits mitochondrial citrate transport. The resulting block in the TCA cycle, and the inhibition of citrate transport mechanisms, ultimately result in the accumulation of citrate in the tissues and plasma, energy deprivation, and death. In addition, high levels of citrate inhibit the glycolytic enzyme, phosphofructokinase and can also binds to serum calcium, resulting in hypocalcemia and ultimate heart failure.

In addition to its known catalytic function, mitochondrial aconitase also appears essential for maintaining the integrity of mitochondrial DNA – required for cells to maintain their respiratory competency. Thus binding of fluorocitrate to aconitase may further compromise the production of energy (ATP) in 1080-poisoned animals. The formation of fluorocitrate during fluoroacetate toxicity results in two main consequences: 1) the inhibition of aconitase within the mitochondria, and 2) the inhibition of citrate flux into and out of mitochondria. These appear to have their earliest effects on cells or organs with high energy demands, e.g. cardiac muscle.

Death from fluoroacetate poisoning is likely to be a multifactorial event, including the occurrence of neurotoxic effects in highly sensitive species (e.g. canids). The neurotoxic effects of fluorocitrate may result from citrate as an inhibitor of the production of acetylcholine, the main neurotransmitter involved in the communication between muscles and their associated nerve junctions.

Toxic effects on possums:

McIlroy (1983): In captive possums administered a range of 1080 doses, first signs of poisoning were observed 1.4–38.2 hrs after dosing. Deaths occurred from 5.0–126.6 hrs after dosing and survivors began recovering 11.6–38.9 hrs after being dosed. Almost 64 percent of possums first exhibited visible symptoms within 12 hrs and 89 percent showed visible symptoms within 24 hrs. Approximately 66 percent of deaths occurred within 24 hrs of dosing, and 91 percent within 48 hrs. Six per cent of deaths occurred. 97–126.6 hrs after dosing. The length of time before symptoms appeared was related to amount of 1080 administered, becoming shorter as the dose increased, but the time to death did not show a similar relationship..

Littin et al. (2009): Captive wild-caught possums were observed after they ingested 1080 carrot baits. Eight lethally dosed possums were not handled and another nine lethally dosed possums were handled to determine responses to various stimuli, indicating level of consciousness and time to loss of consciousness. Unhandled lethally dosed possums died 11 hrs 26 min (± 1 hr 55 min; mean ± SEM) after dosing. Half had abnormal appearances and postures 1 hr 50 min (± 9 min) after consuming baits. Seven showed retching, and three vomited over a period of 27 ± 12 min, starting from 2 hrs 53 min (± 13 min). Lack of coordination began 3 hrs 37 min (± 32 min) after dosing, then possums spent most of the time until death lying, showing spasms and tremors. Five showed seizures while lying prostrate. Possums ingesting a lethal dose of 1080 experienced ~9.5 hrs of changed behaviour.

Nine lethally dosed possums were repeatedly handled and tested from the time they began lying (approx. 3.5 hrs after dosing) till just before death (6 hrs 37 min ± 1 hr 7 min). Loss of responsiveness to different stimuli varied greatly between individuals and some responses were lost and then regained in subsequent tests. Response to a threatening stimulus was lost first, approximately 3 hrs before death. Response to handling, indicating substantial loss of consciousness, was lost in only two possums before death. It was concluded that consciousness was lost only close to death in lethally dosed possums, although awareness was likely reduced for sometime beforehand.

Another eight possums that consumed a dose intended to be sublethal were observed until death or recovery but not handled. All showed signs of poisoning, including abnormal postures, lethargy, lack of coordination, retching, spasms or tremors, and a cessation of grooming, feeding and activity. One died 18 hrs 15 m after dosing, experiencing two seizures within 30 mins of death.

Yockney & Anderson (2009): In a field experiment, time to death in possums after poisoning varied considerably between 5.2 hrs and a maximum recorded 93.9 hrs (mean 30 ± 4.8 hrs).

Toxic effects on humans:

Brockmann et al. (1955): Patient reported to hospital within 45–60 min of ingesting a sodium fluoroacetate solution. Apparently he had dissolved a large amount of the poison in water and swallowed the solution, after which he promptly vomited. He stated that he had noted almost immediate epigastric pain. At the time of admission (4 am) patient was alert and responsive but complained of epigastric pain….the patient gradually became more and more unresponsive and by 5:20 am he was comatose. A half hour later he had a grand mal convulsion associated with fecal incontinence (treatment and physical findings described)…patient in a deep coma…during the next 12 hrs the patient became very restless…frequent episodes of severe carpopedal spasm…periods of neuromuscular hyperactivity…at 11:30 pm of the second day examination showed no change in the physical findings, except that the pupils once again reacted to light. (Treatments and further observations)…patient died on fifth day, apparently without regaining consciousness.

Cai et al. (1997, article in Chinese with English abstract): 'The symptoms of the poisoned persons included dizziness, headache, nausea, vomiting and tic'.

Chi et al. (1996): Retrospective study of 38 consecutive cases of 1080 poisoning in China. Seven of 38 patients (18 percent) died – death occurred within 72 hrs with most deaths soon after ingestion. The clinical manifestations of poisoning are extraordinarily variable – nausea, vomiting and abdominal pain occur initially, followed by anxiety, agitation, muscle spasm, stupor, seizure and coma. Extreme anxiety, verbosity, irritability and hyperactivity were noted. Subjective complaints of respiratory distress were more prevalent in the fatalities. The most common symptom was nausea or vomiting (74 percent). The most frequent ECG finding was nonspecific ST-T and T wave abnormalities (72 percent). Hypocalcemia (42 percent) and hypokalemia (65 percent) were the common electrolyte abnormalities. Discriminant analysis identified hypotension, increased serum creatinine, and decreased pH as the most important predictors of mortality, with sensitivity of 86 percent and specificity of 96 percent. Hypotension and the early onset of metabolic acidosis and increased serum creatinine are associated with poor short-term survival.

Goncharov et al. (2006): In humans, the most characteristic intoxication signs involve generalised tonic–clonic convulsions alternating with deep depression, sudden loss of

consciousness and coma may occur. These were associated with metabolic acidosis, hypotension as well as cardiac rhythm disturbances. Death occurs in 3 hrs to 5 days of heart block, arrhythmia or respiratory failure.

Robinson et al. (2002): First reported case of intentional ingestion of 1080 in the USA in over 15 years. A 47-y-old male was brought to the emergency room status post tonic–clonic seizure. At 34 hrs post ingestion, he responded only to noxious stimuli and at 48 h, he was unresponsive to painful stimuli, was intubated and placed on a ventilator. Over the following 3 days, he became minimally responsive to external stimuli with bouts of agitation and hypertension. Two days later he was discharged with no evidence of neurologic sequelae.

Trabes et al. (1983): Attempted suicide by 1080 ingestion: thirty minutes after ingestion she complained of nausea, vomiting and abdominal pain. One hour later a grand mal seizure occurred. Physical examination revealed tachycardia and profuse sweating, disorientation to time and place and psychomotor agitation. During the next four hrs a progressive deterioration of consciousness with three additional grand mal seizures, then became comatose. Slow improvement by the third day and during the following two weeks patient became progressively more alert. Further evidence of brain atrophy in surviving patient.

Williams (1948): During the weighing [of 1080 powder], a small quantity of poison was blown into the writer's face and some of it was inhaled. A tart, sourish taste was shortly thereafter noted, followed almost immediately by a tingling sensation around the corners of the mouth and in the nasal passages. Becoming alarmed, medical assistance was sought. Soon the entire face had become numb, and the tingling sensation was rapidly entering the arms and legs. This was followed by spasmodic contractions of the voluntary muscles, gradual loss of speech, and within 2.5 hrs after inhaling the powder as noted above, unconsciousness. No actual pain was noted during the entire onset.

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1.5.2.2. Cyanide for possums

Basis of welfare assessment and scoring (summary)

Due to the very quick time to loss of consciousness and death, it is unlikely that possums experience any welfare compromise in domains 1, 2 or 4. Scores for functional impairment (domain 3) are based on observations of respiratory compromise, loss of co-ordination, abnormal movements, followed by prostration with muscle spasms and convulsions. Convulsions are reported to occur after the time reactivity to external stimuli begins to decline, suggesting that consciousness is reduced or absent at this time. In support of this, cortical EEG activity is lost rapidly. The dominant negative affective experience before the loss of consciousness appears to be mild to moderate breathlessness. Increased drive to breathe (and conscious perception of increased drive) is likely related to metabolic acidosis (falling blood pH) as hypoxic tissues switch to anaerobic metabolism.

Humans poisoned with cyanide report respiratory distress, generalized weakness, headache and nausea. Vomiting is common in human victims but is not observed in possums. There are currently no available data upon which to judge these experiences in animals.

Review of literature

Evaluations of welfare impacts / humaneness:

Cyanide is a potent and rapid-acting asphyxiant. At lethal doses inhalation or ingestion of cyanide produces adverse reactions within seconds and death within minutes. In animals, clinical effects occur in rapid succession. Initially there can be excitement and generalised muscle tremor. Animals may salivate, void faeces and urine, and gasp for breath. Convulsions will follow due to anoxia. Note that Mason & Littin (2003) evaluated cyanide gas used as a burrow fumigant for rodent control, rather than ingestion of cyanide bait. It is assumed that the difference in exposure route does not change the overall evaluation of welfare impacts made by these authors: 'For rodents cyanide gas is said to be a quick and relatively untraumatic cause of death although there are few data on its clinical signs and speed of action. Gaseous concentrations of 1 mg/L will kill rabbits in under 1 min (mice reported as being more sensitive and rats less so), while 0.22 mg/L kills rabbits in 18 min on average. In this study, animals generally 'collapsed' in about a third of the time taken to die, while in another cited study, death occurred a minute or less after onset of symptoms, even at concentrations that took 29 min to kill'.

Mode of toxic action:

Cyanide disrupts energy metabolism by preventing the use of oxygen in the production of energy. Cyanide's toxic effect is due to its affinity for the ferric haem form of cytochrome a3 (also known as cytochrome c oxidase), the terminal oxidase of the mitochondrial respiratory chain. Formation of a stable cytochrome c oxidase – CN complex in the mitochondria produces a blockage of electron transfer from cytochrome oxidase to molecular oxygen and cessation of cellular respiration, causing cytotoxic hypoxia in the presence of normal haemoglobin oxygenation.

Tissue anoxia induced by the inactivation of cytochrome oxidase causes a shift from aerobic to anaerobic metabolism, resulting in the depletion of energy-rich compounds such as glycogen, phosphocreatine, and adenosine triphosphate, and the accumulation of lactic acid

with decreased blood pH. The combination of cytotoxic hypoxia with lactate acidosis depresses the central nervous system, the most sensitive site to anoxia, resulting in respiratory arrest and death. Cyanide is known to produce a range of biochemical changes in the brain associated with poisoning. Some of these changes will be associated with acute toxicity, anoxia, and death. On necropsy, carcasses show signs of terminal anoxia with agonal haemorrhages on the myocardium, congestion and haemorrhage in the lungs, trachea and the mucous membranes of the stomach and small intestine.

Toxic effects on possums:

Gregory et al. (1998): Cyanide causes some signs of discomfort but only briefly, these being rapidly followed by unconsciousness. Signs of poisoning include short episodes of hyperpnoea or dyspnoea, uncoordinated and abnormal body movements for about 1 min, and prostration with spasms and a growing lack of responsiveness to external stimuli for a further 3–4 min. No retching, vomiting or evidence of pain was observed. Convulsions occur, but as cyanide causes a rapid loss of cortical EEG activity and as the convulsions occur after the start of the progressive loss of reactivity to external stimuli, they are believed not to be distressing. In this study, ataxia occurred in possums on average by 3 min, convulsions occurred in 73 percent of possums on average at 3 min and 40 s, loss of consciousness at 6.5 min and cessation of breathing (death) at a mean of 18 min.

O'Connor et al. (2003): Based on data from Gregory et al. (1998), cyanide caused mild abnormal breathing in 52 percent of the poisoned possums and convulsions occurred in all animals after they had become unconscious. Potentially little more than 3 min of welfare compromise, based on time from incoordination to loss of response to handling.

Eason & Wickstrom (2001): Death from lower doses of cyanide can in some cases take from 1 to 4 hrs, hence the importance of using high-quality baits and baiting practices to ensure maximum efficacy.

Toxic effects on humans:

Signs of acute poisoning in humans are hyperventilation, headache, nausea and vomiting, generalised weakness and coma, followed by respiratory depression and death (Hayes 1994). Others, such as the depletion of dopamine may be associated with chronic toxicity, such as the development of delayed progressive Parkinsonism and dystonia in humans following sub-lethal cyanide intoxication (Kanthasamy et al. 1994). However, healthy survivors of cyanide exposure have also been reported, both in human and in animal studies.

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1.5.2.3. Cholecalciferol for possums

Basis of welfare assessment and scoring (summary)

The extreme impact score in domain 1 is based on the observation of prolonged anorexia and significant weight loss in possums. No direct information is available to assess environmental challenge or behavioural/interactive alterations. However, the observation of prolonged lethargy suggests that normal behaviour and social interactions may be disrupted. Extreme functional impairment is considered to occur based on observations of altered behaviour and pathology. In possums, mineralization of lungs, heart and kidneys were most commonly reported. Both lung and cardiac pathology may contribute to the observed respiratory compromise.

There is no evidence that consciousness is reduced or lost before death. Prolonged experience of breathlessness, pain (headaches, muscle pain), lethargy/weakness and sickness is likely. Sublethal poisoning of humans with cholecalciferol results in anorexia, vomiting and weight loss, and nausea, neural effects (irritability and depression), headaches and pain in other body parts are reported. Vomiting is not observed in possums suggesting that nausea may not be experienced in this species; however, there are no available data upon which to judge this and other negative experiences in possums.

Information required

The physiological cause of anorexia and whether possums experience significant hunger/thirst after cholecalciferol poisoning is unknown. Studies involving administration of analgesic and

anti-nauseant drugs would shed light on animals' experiences of pain and nausea during progressive mineralization of tissues. More detailed information on respiratory compromise is required to assess intensity and duration of breathlessness.

Review of literature

Evaluations of welfare impacts / humaneness:

Mason & Littin (2003): Dose can affect symptomology (especially progressive calcification) and hence the humaneness implications of such data for lethally poisoned rodents. Poisoned rodents display similar lesions and signs of pain and dysfunction as other mammals and humans. In mice given intra-peritoneal cholecalciferol these included ocular squinting, a reluctance to move, lethargy, weakness, anorexia, hunched posture, rough coat, and dehydration, followed at larger doses by tremors and coma. In another study, high doses led to appetite loss, listlessness, piloerection, hunched posture, lack of reaction to external stimuli, weight loss, priapism, and frequent micturition. Anorexia is also described in much of the rodent control literature.

Internally, blood vessel calcification can be seen in poisoned rodents along with calcification of internal organs. Some workers state that rodents usually experience acute symptoms within 14 hrs; however, others put the onset period at a little later, most rodents becoming ill and ceasing to eat after 24–48 h. In a study of mice the time to the onset of illness after a lethal calciferol injection was two to four days. Another study states that tissue calcification can be seen from two days onward. Death, however, usually takes a few days longer. For example, in laboratory bait studies, times until death range from three to eleven days in mice, two to ten days in black rats, and one to thirteen days in Norway rats with four to five days an approximate average.

In field conditions, typical times to death are also three to five days, sometimes longer. Thus the period for which animals show clinical signs is several days long. For instance, in one study of house mice, the symptomatic period for lethally poisoned animals averaged four to six days depending on dose and in another it ranged from two to seven days. More recently, the mean symptomatic period has been put at two days for rats and three for mice.

Mode of toxic action:

Eason &Wickstrom (2001): Cholecalciferol undergoes metabolic conversion to 25-hydroxycholecalciferol. At toxic doses, this active metabolite mobilizes calcium stores from bones into the bloodstream, and decreases calcium excretion by the kidneys. This results in very high concentrations of blood calcium (hypercalcaemia) and tissue calcification, which can occur in the cardiovascular system, kidneys, stomach, lungs, and muscles. Mineralization and blockage of blood vessels, with death probably from heart failure, appears to be the mode of action of cholecalciferol in the *possum*, as in rodents. In other species, including cats and dogs, renal failure (caused by vessel blockage and nephrocalcinosis) and gastrointestinal haemorrhage appear more prominent.

Toxic effects on possums:

Jolly et al. (1993): The occurrence, speed of onset, and severity of signs is dose-dependent. There appears to be some species variation in the clinical signs of poisoning and target organs affected by cholecalciferol. The clinical signs reported in cats and dogs include nausea,

vomiting, and diarrhoea, but these do not occur in possums. Possums that receive a lethal dose of cholecalciferol bait died within 4–7 days, probably from heart failure. Clinical signs commonly expressed include loss of appetite, constipation, lethargy, tachypnea (rapid and shallow breathing), and death. Autopsy revealed pale mottled hearts and lung congestion in all animals. Histological examination revealed widespread mineralization of cardiac muscle fibres and calcification of blood vessel walls in the heart and kidney. Congestion and alveolar haemorrhage were visible in the lungs. Sublethally affected possums stopped eating and became constipated three or four days after dosing.

Morgan & Milne (2002): Mean time (± SEM) to death in possums by cholecalciferol reported as 6.9 ± 0.5 days (LD15 dose) or 13.3 ± 2.3 days (LD40 dose).

Morgan & Rhodes (2000): Most possums died between 3 and 10 days after eating FeraCol paste bait. One possum died 14.5 days afterwards. No significant difference in time to death between male and female possums, and no possums lost more than 25 percent bodyweight before death.

O'Connor et al. (2003): Cholecalciferol caused mineralization in the organs of 67 percent of possums and lung damage in 59 percent of the animals (in which lung failure was considered the primary cause of death). Seventy-one percent of possums had abnormal breathing for 1.5 days before death. They did not eat for 7 days, on average, and 21 percent lost more than 30 percent of their bodyweight. Mineralization is likely to be associated with pain and distress if it occurs in active muscles or certain organs. In poisoned possums mineralization largely occurred in the heart and kidneys.

Toxic effects on humans:

Mason & Littin (2003): 'Human data tend to come from chronic low-dose poisoning, such as side-effects from medicinal uses, rather than acute high-dose poisoning. However, they are presented here because chronic low-dose human poisoning may illustrate the welfare impact of sublethal poisoning. The primary cause of illness or death in these cases is kidney failure, a secondary outcome being haemorrhage following the rupture of calcified blood vessels. For example, a woman who took calciferol every day for two months developed mental and renal impairment, and another patient, permanent renal damage. Fatal cases have also involved the calcification of heart and lung tissue, as well as the arteries and renal tubules. Victims typically show vomiting, anorexia, weight loss, irritability and depression, and experience severe, frequent (if transient) headaches, nausea, and pain and intense discomfort in other parts of the body.'

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1.5.2.4. Brodifacoum for possums

Basis of welfare assessment and scoring (summary)

The mild impact score in domain 1 is based on the reduced feed intake and moderate weight loss occurring over days to weeks after poisoning. Although there is little or no direct information to assess welfare impacts in domains 2 and 4, progressive lethargy and inactivity suggest that possums could experience environmental challenge and disrupted behaviour and social interactions. There is also one report of possums experiencing hypothermia following brodifacoum poisoning (personal communication, Kate Littin).

Functional impairment is considered to be severe based on altered behaviour, abnormal postures, and severe haemorrhaging in various locations. In possums, haemorrhaging commonly occurred in locations likely to cause severe pain and other negative experiences such as breathlessness e.g. abdomen, lungs, heart, muscles, gut and reproductive organs. There is no evidence of reduced consciousness before death and severe to extreme negative affective states are likely to be experienced. These include: pain due to haemorrhaging; lethargy and weakness due to blood loss; and breathlessness if haemorrhaging occurs in respiratory structures. Reduced feeding may relate to gastrointestinal pain, general sickness, dizziness or nausea; although there are currently no data on which to judge such experiences. Poisoned humans report pain and/or breathing difficulties depending on the site of haemorrhage, as well as dizziness, and reduced motor capabilities.

Information required

It is unknown whether possums experience hunger/thirst after poisoning. Studies involving administration of analgesic and anti-nauseant drugs would shed light on animals' experiences of pain and nausea during the days following brodifacoum exposure.

Review of literature

Evaluations of welfare impacts / humaneness:

Mason & Littin (2003): The latent period between the time of ingestion and the onset of clinical signs varies considerably, but usually occurs within a week of lethal exposure. The symptomatic period ranges – depending on the individual, the particular anticoagulant and, to

some extent, the dose – from just a few hours (in some difenacoum and brodifacoum studies) to, more commonly, an average of one to three days, with a maximum of four to five days of clinical signs. Onset of signs may occur suddenly; this is especially true when haemorrhage of the cerebral vasculature or pericardial sac occurs. Clinical signs commonly include anaemia and weakness. Haemorrhaging may be visible around the nose, mouth, eyes, and anus of mammals. When pulmonary haemorrhage has occurred, blood-tinged froth may be visible around the nose and mouth. Swollen, tender joints are common and if haemorrhage involves the brain or central nervous system, ataxia or convulsions can occur.

Poisoned animals die of multiple causes related to anaemia or hypovolemic shock. The nature, degree and duration of any suffering caused by anticoagulants depend on the site and severity of haemorrhages. This is influenced by the dose received and the exact nature of the anticoagulant compound (see also pindone and diphacinone), but individual predispositions also play a major role.

Mode of toxic action:

Eason & Wickstrom (2001): Brodifacoum, like other anticoagulant toxicants, acts by interfering with the normal synthesis of vitamin K-dependent clotting factors in the liver of vertebrates. In the liver cells the biologically inactive vitamin K1-2,3 epoxide is reduced by a microsomal enzyme into biologically active vitamin K, which is essential for the synthesis of prothrombin and other clotting factors (VII, IX, and X). Brodifacoum antagonism of the enzyme vitamin K1-epoxide reductase in the liver causes a gradual depletion of the active form of the vitamin, and consequently of vitamin K-dependent clotting factors, which results in an increase in blood-clotting time until the point where no clotting occurs. The greater potency of second-generation anticoagulants such as brodifacoum compared to first-generation anticoagulants such as warfarin and pindone is likely to be related to their greater affinity for vitamin K-epoxide reductase and subsequent accumulation and persistence in the liver and kidneys after absorption. Anticoagulants share this common binding site, but the second-generation anticoagulants have a greater binding affinity than the first-generation compounds. All tissues that contain vitamin K-epoxide reductase (e.g. liver, kidney, and pancreas) are target organs for accumulating these toxicants.

Toxic effects on possums:

Littin et al. (2002): Blood-clotting time was prolonged 8 days after possums first began ingesting brodifacoum and time to death was 20.7 ± 1.7 days. Clinical signs of poisoning, including changed appearance, pale noses and external bleeding, appeared from 14 days after initial poisoning (7 days before death). Possums gradually became inactive and lethargic, typically crouching and lying in abnormal postures for 6 days before death. Feed intake reduced concurrently, resulting in significant body weight loss of 5.9 ± 2.1 percent. All possums had widespread, usually severe, haemorrhaging. Internal haemorrhages first appeared in all possums 8 days after initial ingestion. These haemorrhages, and consequent blood loss, may cause distress, pain, weakness or sickness, and this is supported by evidence from humans and other animals. Reduced feed intake, inactivity, lethargy and the display of abnormal postures suggest that possums do experience distress for at least 6 days before death.

Littin et al. (2000): Feed intake declined over the thirteen days after brodifacoum ingestion, and was significantly reduced at day fifteen. Crouching and lying postures in possums

increased markedly 15 days after initial exposure to brodifacoum (5 days before death) with a corresponding drop in the amount of time spent in a resting posture. Throughout prolonged inactivity, clinical signs included external bleeding and pale extremities. Possums had most haemorrhaging in subcutaneous or deep tissues in the lumbar region (9/12), abdomen (6/12), lungs (8/12), heart (7/12), limbs (6/12), gut (6/12) and testes (4/12). One possum had free blood in the hip joints. Possums took nearly three times longer to die than rats – the onset and duration of sickness also occurred later/longer in possums than rats (up to 5 days vs. up to 3 days).

O'Connor et al. (2003): Based on data from Littin et al. (2002), mean time to death was 21 days. Behaviour and appearance of clinical signs suggest possums can experience welfare compromise for 6–7 days before death.

Toxic effects on humans:

Mason & Littin (2003): Mildly poisoned humans show increased bruising rates and bleeding from cuts, occasional nose and gum bleeds, blood in the faeces or urine, a pale mouth and cold gums, and general weakness. More severe cases involve widespread haemorrhaging, usually internal autopsies reveal, for example, pulmonary and sub-dural haemorrhages, ovarian haematomas, multiple bleeding sites on the skin, and sub-mucosal bleeding into the lips. Medical case reports further describe bleeding from the urethra, intra-abdominal haemorrhaging, mesenteric haematomas, pleural effusions, acute renal failure, pericardial haemorrhages, haemoarthrosis, blood in the gastrointestinal tract, intra-cerebral haemorrhages, and other lesions.

Bleeding *per se* is not painful, but the accumulation of blood in enclosed spaces generally is. Thus, poisoned humans can experience localized muscle pain, joint pain and potentially severe abdominal pain caused by intra-peritoneal, mesenteric or ovarian bleeding. Haemorrhages within the lungs, kidneys, spinal cord, orbits of eyes and gonads are also painful. Bleeding into lungs or airways can cause further distress by making breathing difficult, and poisoned humans may also experience dizziness, localized reduced motor strength, the inability to urinate, and sometimes even paraplegia.

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1.5.2.5. Pindone for possums

Basis of welfare assessment and scoring (summary)

Possums appear resistant to the anticoagulant effects of pindone. Welfare impacts relate primarily to liver failure resulting from chronic administration of relatively high doses of pindone. Functional impairment of the liver after such chronic dosing is severe enough to eventually result in death. Although no data are available, progressive liver failure is likely to cause negative affective states such as sickness, lethargy/weakness, and possibly nausea. No direct data are available for assessment of welfare impacts in domains 1, 2 and 4. However, the prolonged period over which liver failure occurred and the assumed sickness/malaise and lethargy suggest that feed/water intake may be moderately compromised and animals may be exposed to environmental challenge.

Information required

It is unknown whether possums experience hunger or thirst after pindone poisoning. More specific information on the time to onset of sickness behaviours after chronic pindone dosing and the quality/intensity of those behaviours is required to definitively evaluate the intensity and duration of negative welfare impacts. Direct information on feeding/drinking behaviour, potential for environmental exposure, and alterations in behaviour is required to assess impacts in domains 1, 2 and 4. Studies involving administration of analgesic and anti-nauseant drugs would shed light on animals' experiences of pain and nausea during the days and weeks following chronic pindone poisoning.

Review of literature:

Evaluations of welfare impacts / humaneness:

As for brodifacoum, noting that toxic effects on possums appear distinctive from those reported in some other mammal species.

Mode of toxic action:

As for brodifacoum.

Toxic effects on possums:

Eason & Jolly (1993): Six possums were dosed orally with 25 mg/kg pindone, and another five possums were dosed with 100 mg/kg pindone. Mean haematocrit and one-stage prothrombin time were unchanged in possums that received 25 mg/kg. In possum that received 100 mg/kg, the one-stage prothrombin time increased from baseline (41.9 to 80.0 s) six days after dosing and the activated partial prothrombin time increased from 30.2 to 103.9 s

six days after dosing. The haematocrit was unchanged. There was no mortality in possums at any dose.

Jolly et al. (1994): Possums were administered a range of pindone doses. None of 12 possums dosed with 8 or 16 mg/kg/day for 5 days died. One of 12 possums died when dosed with 32 mg/kg/day for 5 days and 9 of 14 possums died when dosed with 64 mg/kg/day for 5 days. Most possums died between 1 and 2 weeks after dosing ceased, and liver damage was the only apparent pathology. Necropsy showed that all possums killed by the poison had pale, mottled and hardened livers. Histological examination of liver tissue revealed severe centri-acinar changes, with hepatocytes showing necrotic changes characterised by fine vacuolation of the cytoplasm and areas of fibrinoid necrosis. Cell nuclei showed pyknosis and karyorrhexis. In some animals, up to 20 percent of each lobule was affected. In 3 possums which died 15 to 18 days after dosing small areas of subcutaneous haemorrhage, as well as liver damage were observed. The possum appears resistant to the anticoagulant effects of pindone and death from high doses is the result of liver failure.

Toxic effects on humans:

As for brodifacoum.

References

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1.5.2.6. Phosphorus for possums

Basis of welfare assessment and scoring (summary)

There are no direct data available on which to assess welfare impacts in domains 1, 2 and 4. Mild impact scores in these domains were based on mild to moderate gastrointestinal pathology and altered behaviour after phosphorus ingestion. Observations of mild to moderate gastrointestinal pathology, vomiting and retching, abnormal postures and reduced activity were the basis of the moderate to severe impact score for domain 3 (functional impairment). As animals remained conscious until shortly before death, they were likely to experience severe negative affective states for a number of hours, including gastrointestinal pain, nausea and sickness.

Information required

Direct information on feeding/drinking behaviour, potential for environmental exposure, and alterations in behaviour is required to assess impacts in domains 1, 2 and 4. Studies involving administration of analgesic, anti-nauseant and anti-anxiety drugs would shed light on animals'

experiences of pain, nausea and anxiety after ingestion of phosphorus paste. More detailed information on level of consciousness after animals become prostrate would help clarify the duration of negative affective experiences. Time to loss of normal EEG and evoked potentials after dosing may be useful.

Review of literature

Evaluations of welfare impacts / humaneness:

Eason & Wickstrom (2001): In the veterinary literature, phosphorus poisoning is usually categorized in three phases: (1) An acute initial phase occurring within hrs of ingestion characterized by gastrointestinal, abdominal, and circulatory signs. Initial signs generally involve vomiting and diarrhoea. If the dosage is sufficiently large, shock, cyanosis, incoordination and coma may develop, with death occurring before the second and third phases appear; (2) An interim or latent phase with apparent recovery occurs at lower doses approximately 48 hrs to several days after initial clinical signs; (3) The third stage is characterized by recurrence of marked clinical signs involving the gastrointestinal tract. Liver failure then occurs. These literature reports suggest that death may occur in 1–2 days, or there may be improvement for 1–2 days before vomiting, diarrhoea, and other signs return. Death is usually due to liver necrosis and heart failure.

There may be a delay of up to 3 weeks after ingestion before convulsions, coma, and death. Pathological changes include gross evidence of fatty degeneration and swollen livers, as well as gastrointestinal irritation, necrosis, and haemorrhage. If death is sufficiently prompt, there is no pathology except irritation of the oesophagus and stomach. Perforation may occur. Following survival for several days, fatty degeneration is striking in the liver, heart, and kidney but may be found in all organs, including the brain. We were unable to locate any material relating to genotoxicity or teratogenicity, or data from other regulatory toxicology studies on phosphorus.

Mode of toxic action:

Eason & Wickstrom (2001): The mode of action is unknown. It has not been possible to associate the main clinical or pathological features of intoxication with inhibition of any particular enzyme or class of enzymes. Phosphorus is sometimes referred to as a protoplasmic poison, but it is difficult to distinguish its possible direct effects on the liver, kidney, brain, and heart from the effects of anoxia on those organs. The peripheral vascular dilatation, which is the first and most pervasive systemic effect of phosphorus, contributes to all the disorders that may be seen in various organs. However, the mechanism of this dilatation is not clear. Phosphorus not only leads to structural damage of vital organs, but also produces serious disruption of their metabolic function, as evidenced by hypoglycemia, azotemia, inhibition of glycogen formation in the liver, and many other disorders.

Toxic effects on possums:

O'Connor et al. (2007): After ingestion of phosphorus paste by wild-caught possums (18 high dose, nine low dose, and 12 non-poisoned controls), behavioural observations were made at 15-min intervals for 24 hrs or until death. Serum biochemistry, and gross and microscopic pathology were assessed at 3-hourly intervals in a further 21 possums. Possums that ingested phosphorus paste developed an abnormal posture (high incidence of crouching after 4–8 h), mild congestion of the gastric mucosa, and elevated levels of creatine kinase in serum after 3–

6 h. Retching was observed in 67 percent possums, and 44 percent vomited at least once. Possums were prostrate from about 18 hrs after eating the poison, and the response to handling, an indicator of consciousness, was lost at about 24 hrs, followed by death at 25 hrs.

The main welfare concern was the possibility of discomfort or pain caused by the congestion of the gastric mucosa, as indicated by the crouched posture adopted by poisoned possums. Retching and vomiting may also have caused pain and distress. The degree of pain or discomfort would depend on the degree of congestion of the gastric mucosa, which was typically mild, and on the duration and severity of retching and vomiting, which were typically short and mild. Possums remained conscious until 1 hr before death, implying that they were able to experience pain and distress from the effects of ingestion of phosphorus for almost the entire period of illness, which lasted for approximately one day.

Toxic effects on humans:

Duerksen-Hughes et al. (1997): There are numerous reports of death following oral exposure to white phosphorus, including a report in which 56 individuals consumed 0.19–6.3 g of white phosphorus. The mortality rate was 48.2 percent, with 90 percent mortality in those ingesting ≥1.57 g of phosphorus. However, due to rapid vomiting induced by oral ingestion, the doses resulting in death are difficult to estimate.

Fahim et al. (1990): Thirty patients with acute phosphorus poisoning were chosen from the poison control centre, Cairo, (Ain Shams University), and classified into three groups according to the amount of ingested inorganic phosphorus. Group I recorded high mortality rate (90 percent) with liver failure as a main cause of death. This group also showed hepatorenal failure, hypoglycemia and severe effect on the heart function. Groups II and III recorded no mortality, but their patients showed an effect on the liver, which was severe in group II and mild in group III.

McCarron et al. (1981): Ten cases of ingestion of yellow phosphorus rat poison, including four cases that occurred during the past 3 years, are reported. Comparison of these cases with 82 others from the literature showed that ingestion of yellow phosphorus paste often results in clinical findings that are different from those described for acute yellow phosphorus poisoning in current toxicology texts. The time lag between swallowing the poison and onset of symptoms varied from a few minutes to 24 h. Garlic odor, mucosal burns, and phosphorescent vomitus or faeces occurred in only a small percentage of cases. Diarrhoea was not a presenting complaint. Initial symptoms were referable to the gastrointestinal tract, central nervous system, or both. Mortality rates were 23 percent for patients who had early symptoms of vomiting or abdominal pain; 73 percent for those where the first manifestation of intoxication was restlessness, irritability, drowsiness, stupor, or coma; and 47 percent for patients who had a combination of these GI and CNS symptoms initially.

Rao et al. (1974): There appear to be three stages in phosphorus poisoning. Stage 1 usually begins shortly after ingestion and symptoms include nausea, vomiting, abdominal pain and shock. This can last from 6–8 hrs and, if the patient does not die, can be followed by a symptom-free period (Stage 2) of 1–3 days (in some cases up to 10 days). During the third stage, there is systemic toxemia due to the action of the absorbed phosphorus. This can affect the liver, kidneys, and heart and usually results in death.

Simon & Pickering (1976): Classical descriptions of phosphorus poisoning show that symptoms can occur within a few minutes to hrs following exposure. Time from onset of symptoms to death varies, depending on dose, from minutes to up to 3 weeks.

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1.5.2.7. Zinc phosphide for possums

Basis of welfare assessment and scoring (summary)

The basis for the mild impact score for domain 1 was observation of reduced appetite followed by retching and vomiting. Although no direct data are available for scoring compromise in domains 2 and 4, the marked sickness behaviour and pathology suggest that altered behavioural interactions and environmental challenge are likely. Severe functional impairment relates to retching and vomiting, abnormal postures, gastric and lung pathology, respiratory compromise, and progressive neural effects such as incoordination and convulsions. Level of consciousness appears to be progressively reduced over the minutes preceding death; however, convulsions followed by recovery are reported by Fisher et al. (2004). Possums are likely to experience hours of severe negative affective states before consciousness is reduced or lost. Nausea, gastrointestinal pain and breathlessness are likely experienced, as well as sickness and lethargy/weakness. Thirst, anxiety are also reported by human victims; however, no data are currently available on which to assess such experiences in animals.

Information required

Most importantly, more specific information on the level of consciousness during and after convulsions is required. This could be achieved through evaluation of EEG, evoked potentials and behavioural reflexes immediately before and after convulsions. Studies involving administration of analgesic, anti-nauseant and anti-anxiety drugs would shed light on animals' experiences of pain, nausea and anxiety. More specific information on respiratory compromise is required to assess potential breathlessness. Direct information on feeding/drinking behaviour, potential for environmental exposure, and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

Mason & Littin (2003): Clinical signs generally appear rapidly. For example, a reduction in feeding by rodents may be apparent in 15 min or less, reduced activity within 1 hr and behaviours such as abdomen-kicking 3–6 hrs after ingestion. Most studies indicate a symptomatic period of several hours with intoxication occurring over several days in those rodents that do not die overnight. Necropsies of poisoned rodents have shown acute catarrhal enteritis in the duodenum and gastric ulcers consistent with chemical corrosion, along with blood in the trachea and lungs, and coronary and hepatic congestion.

Signs of poisoning in animal studies are similar to those reported for humans (except that rodents cannot vomit), and include respiratory distress, diarrhoea, excitation and lassitude and depression. Poisoned rodents may kick at their abdomens with their hind feet and show postural changes indicative of pain. Final symptoms can include convulsions and paralysis. Times taken to die vary between studies, with an almost bimodal distribution of death times that is presumably dose-related and perhaps reflects the poison's two main actions. Deaths seem to be either rapid (i.e. well under 24 hrs) or more prolonged (e.g. 24–72 hrs). Various rodent studies report that most die within 24 hrs or even 'overnight', but that some deaths can be delayed for several days, in which case liver damage occurs. Time to death in Zinc phosphide poisoning appears to be dose-dependent.

Mode of toxic action:

Zinc phosphide reacts with stomach acid to produce phosphine gas which, upon absorption, is a potent inhibitor of cytochrome oxidase with additional direct cytotoxic effects. Organs with the greatest oxygen requirements, such as the heart and brain, are thus particularly sensitive to damage. Effects on blood vessels and erythrocyte membranes lead to cardiovascular collapse and death usually occurs as a result of cardiac and respiratory failure and in some cases haemolytic effects on liver and kidney, preceded by pulmonary oedema and hypotension. Corrosive phosphine can also damage tissue in other organs such as the liver and kidneys.

Toxic effects on possums:

Fisher et al. (2004): Captive possums that voluntarily consumed a lethal dose (50 mg/kg) of ZP were observed throughout the progression of toxicosis until death, and behavioural observations were used to make an assessment of the signs and timing of ZP poisoning in this

species. The first illness event observed in poisoned possums was retching, which generally coincided with observations of an abnormal posture. Retching was closely associated with vomiting in most possums. Possums were generally conscious until just before (6–30 min) death (n = 6), or did not appear to become unconscious before death (n = 2). The mean time to death in possums that voluntarily consumed 50 mg ZP/kg was 4 hrs 41 min (range 2:49 to 6:54). The total duration of abnormal behaviours (potential welfare compromise) observed in these possums ranged from 49 min 38 s to 4 hrs 8 min 56 s. There was potential welfare compromise for 3.25 hrs during ZP poisoning in possums.

The main clinical signs seen that could be associated with welfare compromise were abnormal postures (suggestive of gastric distress and/or pain), vomiting and/or retching, convulsions followed by recovery. Possums were necropsied soon after death. The most common observations were mild inflammation in the stomach or small intestine (five possums), congestion of patches of lung (five possums), moderate haemorrhage in the spleen (four possums) and slight congestion of the liver (three possums). Samples for histology were taken from the parts of the lungs of two possums that showed extensive dark areas. In the lungs of the first possum, there was severe congestion of alveolar septal capillaries and venules, with small numbers of red blood cells in a few alveoli. The lungs of the second possum were very similar, with moderate numbers of red blood cells in many alveoli. This histology corresponds to severe vascular congestion in both cases, with haemorrhage also present in the second possum.

The histopathology seen in possums poisoned by ZP was attributed to phosphine being absorbed in the blood from the stomach and intestines, and circulating. Phosphine is recognised as a severe pulmonary irritant, and was likely to have also caused the gastric pathology seen. Most of the gross pathology observed in possums is consistent with that reported for other mammals poisoned with ZP. However, the occurrence of spleen lesions in possums seemed to be more common than reported in other mammals.

Henderson et al. (2002): After possums ate baits containing ZP (> 20 mg/kg) there was a latent period of 1.3–1.5 hrs before clinical signs; the latent period was dose-dependent. Frequent urination was noted during the early stages of toxicosis, followed by sickness behaviour that lasted average 1.5–2.4 hrs, including appetite suppression at 1.0–2.25 hrs after eating bait followed by vomiting, with most possums that consumed larger toxic doses vomiting. Retching was observed in 2 of 33 possums. Shortly after vomiting most possums assumed a slightly hunched posture that characterises epigastric pain. The duration of this posture varied but was rarely evident by the time possums started to lose muscular co-ordination.

For possums ingesting >20 mg/kg, ataxia was evident 1.4–5.0 hrs after eating bait. Shortly after losing muscle co-ordination most possums became recumbent and died. Dyspnoea was apparent in most possums 1.5–4 hrs after eating bait and for some this continued until death. Possums remained conscious until a few minutes before death. At various stages during the 30 min before death possums became unresponsive to noise. Most possums remained responsive to pain until 1–12 min before they died. Except for 2 possums that lost consciousness 10–12 min before death, the remainder retained the blink reflex and/or attempted to roll onto their stomach almost until the time of death. For possums ingesting <20 mg/kg ZP, the average period of sickness behaviour was 16.9 hrs and time to death 19.9 hrs. For possums ingesting >20 mg/kg the period of sickness behaviour ranged from 0.3–2.8 hrs, with average times to death 2.9–4.0 hrs.

O'Connor et al. (2006): In possums that voluntarily consumed ~ 100 mg zinc phosphide, two of ten showed retching, six of ten vomiting and two of ten had convulsions. Mean time to death was 7.38 ± 0.94 hrs.

Toxic effects on humans:

Zinc phosphide ingestion causes fatty acid degeneration and necrosis of the liver and pulmonary hyperemia and edema and cardiac damage. Clinical symptoms include nausea, abdominal pain, chest tightness, excitement and agitation and a feeling of being 'cold all over'. Vomiting is constant and jaundice has been noted in some cases. Shock, early dyspnoea, thirst, oliguria, convulsions or coma have been features of fatal outcomes.

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Table 1.4: Relative welfare impacts of VTAs affecting rodents in New Zealand: Mouse (*Mus musculus*); Norway rat (*Rattus norvegicus*), Ship or Black rat (*Rattus rattus*). Compound 1080 is used for control of rat species, brodifacoum and diphacinone is used to target all three species, and pindone is registered for control of rats (not mice).

	Domain of potential welfare compromise											
Agent	1	2	3	4	5	C	Overall impact	C	Duration	C	Overall grade	C
1080	Mild (*No – Mod*) NDD	No – Mild NDD	Mod – Sev	Mild (*No – Mod*) NDD	Severe (*Mod – Ex*) Pain Nausea Sickness Breathlessness Anxiety	 2 1 1 1 1	Severe (*Mod – Ex*)	1	Hours	1	6 (*5 – 7)*	1
Brodifacoum*	Mod (*No – Sev*) NDD	Mild – Moderate (*No – Sev*) NDD	Severe – Extreme	Moderate (*No – Sev*)	Severe – Extreme Pain Breathlessness Lethargy/weakness Sickness Dizziness Anxiety	 3 1 0 0 0 0	Severe – Extreme	3	Days	3	7.5 (*7 – 8*)	3
Pindone†	See text											
Diphacinone†	See text											

* No direct data for mice, assume impacts same as for rats

† Insufficient published evidence for assessment of pindone or diphacinone.

1.5.2.8. Sodium fluoroacetate (1080) for rodents

Basis of welfare assessment and scoring (summary)

No direct data are available for assessing impacts in domains 1, 2 or 4. Scores for food/water deprivation are based on the duration of sickness behaviour observed as well as likely nausea, although it is unknown whether poisoned rats would experience hunger/thirst during this time. Likewise, potential environmental challenge and behavioural/interactive alterations are based on the quality and duration of altered behaviour. Scores for domain 3 (functional impairment) are based on observations of abnormal postures and pain-related behaviours (increased grooming/scratching of abdomen), altered respiration, and neural effects such as incoordination, hypersensitivity and convulsions. Whether animals are conscious during or after these convulsions is unknown. Animals conscious during such events or those that recover consciousness afterwards may experience pain, breathlessness and anxiety/fear.

Based on these impairments, it seems likely that rats poisoned with 1080 experience abdominal pain, nausea, sickness, breathlessness and anxiety. Experiences of nausea and anxiety are supported by the ameliorating effects of anti-nauseant and anxiolytic drugs on relevant behavioural symptoms. Hyperpnoea and breathlessness are likely stimulated by the metabolic acidosis caused by citrate accumulation (Peters, 1952). Humans poisoned with 1080 report epigastric/abdominal pain, respiratory distress (breathlessness), nausea, dizziness and anxiety.

Information required

Information on the duration of symptoms (i.e. onset to loss of consciousness) is lacking for rats. Evaluations of level of consciousness at various times/events after dosing is required to determine the quality and duration of negative affective experiences. In contrast to possums, there is no published information for rats suggesting a progressive reduction in awareness after poisoning which could reduce the duration of negative affective experiences. Information on time to loss of behavioural reflexes, normal EEG and evoked potentials after dosing is required. In addition, such measures should be used to evaluate level of consciousness immediately before and after convulsions.

Studies involving administration of analgesic drugs would shed light on animals' experiences of pain. More specific information on respiratory compromise (i.e. time of onset, progression relative to alterations in consciousness) is required to assess potential breathlessness. Direct information on feeding/drinking behaviour, potential for environmental exposure, and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to 1080 section above (under possums).

Mode of toxic action:

Refer to 1080 section above (under possums).

Toxic effects on rats:

Buffa et al. (1971): In the fed rat, a lethal dose of fluoroacetate caused marked accumulation of citrate, rapid fall in the amount of glycogen and decrease of tissue ATP in liver cells.

Cook (1998): In comparison to saline-dosed laboratory rats, rats orally dosed with 2.5 mg/kg 1080 showed hypersensitivity to light and sound, and increased incidence of grooming or scratching of abdomen, increased cage pacing and increased curled-but-awake posture. Five of ten 1080-dosed rats showed convulsive behaviour between 4 and 10 hrs of 1080 administration. The author concluded 'there is no clear evidence yet to suggest that the behaviours observed following 1080 administration have discomfort or welfare relevance. However, the observation that an anti-nausea and emetic agent and an anxiolytic agent reduced these behaviours, by extrapolation, suggests speculatively that they may respectively represent some form of gastric upset or anxiety'.

Gorniak et al. (1994): Per oral dosing of six laboratory rats with 1.09 mg/kg 1080 produced convulsions in all animals.

McIlroy (1982): Black rats (Rattus rattus) orally dosed with 1080 showed a 0.8–27.8 hrs latent period and 2.4–36.5 hrs time to death and laboratory rats (Rattus norvegicus) orally dosed with 1080 showed a 0.4–2.3 hrs latent period and 2.5–112.0 hrs time to death…'all affected individuals initially appeared depressed, often sitting quietly hunched in a corner or lying on their side, back or stomach, with their eyes partly closed…Most animals were hypersensitive to external touch or sounds, but were generally reluctant to move and if forced to, did so in an uncoordinated manner, with unsteady balance. Respiration was initially very rapid but gradually became slower, shallower and more irregular, until death occurred….convulsions were commonly observed amongst Rattus species…with affected animals suddenly squealing, sometimes rapidly circling the cage or gripping the cage wire with their front feet or teeth, and, while lying on their sides or backs, making rapid paddling motions'.

Toxic effects on humans:

Refer to 1080 section above (under possums).

References

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1.5.2.9. Brodifacoum for rodents

Basis of welfare assessment and scoring (summary)

Although brodifacoum is used to poison mice in New Zealand, there are currently insufficient published data to assess potential welfare impacts in this species. In our current evaluation of welfare impacts on mice, we have assumed similar effects and responses to those observed in rats. Rats are anorexic for several days before death and are reported to experience significant weight loss over this time (personal communication with Kate Littin). The altered behaviour of rats (and mice) poisoned with brodifacoum is likely to make them more susceptible to predation, resulting in a Moderate impact score for domain 4, and suggesting they may experience mild–moderate environmental challenge (domain 2).

The degree of functional impairment depends primarily on the site(s) of haemorrhage. Haemorrhages have been reported in locations likely to result in severe impairment and pain (muscles, joints, gastrointestinal tract, abdominal cavity, reproductive organs). In addition, bleeding into the lungs may result in respiratory compromise and breathlessness. Observations of reduced activity, abnormal postures and patterns of behaviour, paresis and paralysis support these impairments and experiences. Rats are reported to remain conscious until death and therefore likely experience pain, lethargy/weakness, sickness, breathlessness and possibly anxiety/fear.

Information required

Comprehensive studies supporting assessment of welfare impacts of brodifacoum on mice are required, particularly those providing more detailed information on quality, intensity and duration of symptoms (i.e. time to onset, time from onset to loss of consciousness/death). For rats and mice, studies involving administration of analgesic and anxiolytic drugs would shed light on animals' experiences of pain and anxiety/fear following brodifacoum poisoning.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to Brodifacoum section above (under possums).

Mode of toxic action:

Refer to Brodifacoum section above (under possums).

Toxic effects on rats:

Cox (1991): Behaviour of rats poisoned by brodifacoum was studied on time-lapse video in individual cages and group enclosures. All rats readily took the bait and nine out of ten individuals died, with females surviving longer than males. In the group enclosures, when

outside the nest box, the rats spent most time at the edges. But four days after dosing, time spent in the open areas increased five-fold and, rats spent more of the light phase outside the nest box. It was concluded that the behaviour of anticoagulant-treated rats could make them more vulnerable to predation.

Littin et al. (2000): In Norway rats, there was a mean of three days between the onset of clinical signs and death (which occurred at a mean of 7.2 days). Four days after ingesting brodifacoum, rats showed reduced activity levels, anorexia, and less use of their normal curled sleeping posture. Instead, they were frequently seen lying, or standing in a hunched posture with the abdomen tucked up and head lowered. Animals typically remained conscious during this time. One third of the rats developed paresis and then paralysis two days before death. Those rats that underwent partial paralysis lay prostrate for a mean of 11.4 hrs prior to death, but stayed conscious and occasionally pushed or pulled themselves along the floor. Necropsies of rats revealed the following spectrum of haemorrhage sites: subcutaneous and deep tissues of the thorax (10/12 animals), limb musculature (7/12), testes (5/6 males), and stifle joints (2/12).

Mason & Littin (2003): Poisoned rats show external bleeding and pale extremities along with bloody diarrhoea. Internally, there can be multiple haemorrhages throughout the muscles and intestinal tract, bleeding into the body cavities and epididymis and haemorrhages into the joints, lungs, other viscera and skeletal muscle and subcutaneous haematomas are common. A range of rat studies report similar findings, plus gastrointestinal, orbital, intra-cranial and a variety of other haemorrhages judged 'capable of producing severe pain'. The symptomatic period is presumably reduced when times to death are shorter. In one study, for example, most poisoned rats took just one to three days to die, and some, less than 24 hrs. However, times to death are typically in the region of four to eight days. Thus, overall, although there can be variation, the norm is for clinical symptoms to last for several days.

Toxic effects on mice:

Brown & Singleton (1998): Sixteen radio-tracked mice in a field study were found dead within 6 days of brodifacoum bait application.

Mason & Littin (2003): Mice show evidence of altered behaviour: in the wild, brodifacoum-poisoned mice have been observed above ground during the day and, in one study, around 25 percent died above ground or half submerged in their burrows indicating abnormal activity patterns consistent with illness. Mice can take up to eleven days to die.

Morriss (2007): Wild-caught mice offered brodifacoum bait had a mean time to death of 9.0 ± 0.6 days. Three of forty mice took longer to die (18–21 days) in the controlled captive conditions of the trial. The rigors of living in the wild would probably reduce this time to death, as poisoned mice would be exposed to movements and minor injuries that would probably exacerbate the likelihood of fatal haemorrhage caused by poisoning.

O'Connor & Booth (2001): Mean time to death of 9.9 days (range: 6–18 days) in wild-caught mice offered brodifacoum bait.

Toxic effects on humans:

Refer to Brodifacoum section above (under possums).

References

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1.5.2.10. Pindone for rodents

Basis of welfare assessment and scoring (summary)

Overall, there is insufficient evidence to assess welfare impacts of pindone poisoning on rats. Considering the identical mode of action and similar symptoms and pathology reported (anaemia, abnormal postures, external and internal haemorrhages), the welfare impacts for rats are likely to be similar to those of brodifacoum. Time to death may be slightly longer for pindone; however, there is no published information available on duration of symptoms (i.e. time from onset of symptoms to loss of consciousness) for pindone.

Information required

Comprehensive studies supporting assessment of welfare impacts of pindone on rats are required if use of this first-generation anti-coagulant is to continue for rat species. In particular, studies should provide more detailed information on quality, intensity and duration of symptoms (i.e. time to onset, time from onset to loss of consciousness/death).

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to Brodifacoum section above (under possums).

Mode of toxic action:
Refer to Brodifacoum section above (under possums).

Toxic effects on rats:

Bentley & Rowe (1956): Range of days to death in Norway rats (*Rattus norvegicus*) was 4–10 days. Range of days to death in Black rats (*Rattus rattus*) was 4–13 days.

Fisher et al. (2003): Mean time to death in two groups of laboratory rats (*R. norvegicus*) after ingestion of pindone bait was 2.0 ± 0.45 days and 8.39 ± 0.54 days. All rats that died in the treatment groups showed behaviour and post-mortem pathology indicative of anticoagulant poisoning, e.g. anaemia and un-groomed appearance, hunched posture, visible bleeding from nose, large internal haemorrhages.

Toxic effects on humans:
Refer to Brodifacoum section above (under possums).

References

Bentley, E. W. and Rowe, M. (1956). Pival, an anticoagulant rodenticide. *Journal of Hygiene* 54, 20–27.

Fisher, P., O'Connor, C., Wright, G., and Eason, C. (2003). Persistence of four anticoagulant rodenticides in the liver of laboratory rats. DOC Science Internal Series 139. New Zealand Department of Conservation, Wellington, 19p.

1.5.2.11. Diphacinone for rodents

Basis of welfare assessment and scoring (summary)

Overall, there is insufficient evidence to assess the welfare impacts of diphacinone on rats and no information available for mice. Considering the identical mode of action and similar symptoms (reduced activity) and pathology reported (haemorrhage in joints, limbs, gastrointestinal tract and other internal organs and subcutaneously), the welfare impacts for rats are likely to be similar to those of brodifacoum. Time to death may be slightly longer for diphacinone; however, there is no published information available on duration of symptoms (i.e. time from onset of symptoms to loss of consciousness).

Information required
Comprehensive studies supporting assessment of welfare impacts of diphacinone on rats and mice are required. In particular, studies should provide more detailed information on quality, intensity and duration of symptoms (i.e. time to onset, time from onset to loss of consciousness/death).

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to Brodifacoum section above (under possums).

Mode of toxic action:

Refer to Brodifacoum section above (under possums).

Toxic effects on rats:

Elias & Johns (1981): In a 90-day feeding study with laboratory rats, times to death following ingestion of diphacinone ranged from 3 to 20 days. Necropsy generally revealed massive haemorrhage primarily in the thoracic or abdominal areas.

Fisher et al. (2004): Mean times to death in two groups of laboratory rats ingesting diphacinone bait were 69.21 ± 4.43 hrs and 8.24 ± 0.38 days. No significant differences from times to death from other anticoagulants (including brodifacoum and pindone) were found.

Swift (1998): Days to death for wild-caught *R. rattus* ingesting diphacinone bait ranged from 5.5 to 10.5 days, with most mortalities occurring between six and nine days. Signs of poisoning observed during necropsy included haematoma in the joints and extremities, massive haemorrhaging under the skin, especially around the neck and on the skull, and extremely pale flesh and internal organs. Stools were black, indicative of internal bleeding. Many animals had a pinkish discharge from the eyes (also noted by Elias and Johns, 1981) that was not observed in control rats. One rat that was euthanased at the end of a ten-day post-treatment period, as it had lost 31 percent of its pre-test bodyweight and had been lying on its side for five days.

Toxic effects on mice:

Kusano (1974): Laboratory mice poisoned in diphacinone toxicity trials died in 3 to 12 days.

Toxic effects on humans:

Refer to Brodifacoum section above (under possums).

Katz & Metz (1969): Ingestion of diphacinone ('Didandin', used at the time as a human anticoagulant therapeutic) by a 5 year old boy. Two days after admission, severe epistaxis, profuse haematuria and melena occurred. General condition deteriorated, he became confused and increasingly pale. Multiple bruises were still present with fresh ecchymotic haemorrhages on trunk and legs. Decreased haemoglobin concentration and haematocrit. Successfully treated with transfusions and Vitamin K.

Spiller et al. (2003): An 18-year-old male worker at a pest exterminating company spilled a concentrated liquid preparation of 0.16 percent diphacinone in his boot. He did not remove the boot or wash the area for 6 to 8 h. Seven days later he presented to the emergency department with flank pain, hematuria and epistaxis. Urinalysis reported gross hematuria with RBCs too numerous to count. Prolonged bleeding was noted at intravenous puncture sites. Initial therapy included intramuscular injection of vitamin K and nasal packing. Patient successfully treated.

References

Elias, D. J. and Johns, B. E. (1981). Response of rats to chronic ingestion of diphacinone. *Bulletin of Environmental Contamination and Toxicology* 27, 559–567.

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Spiller, H. A., Gallenstein, G. L., and Murphy, M. J. (2003). Dermal absorption of a liquid diphacinone rodenticide causing coagulaopathy. *Veterinary and Human Toxicology* 45, 313–314.

Swift, C. E. (1998). Laboratory bioassays with wild-caught black (*Rattus rattus*) and Polynesian (*R. exulans*) rats to determine minimum amounts of Ramik® Green (0.005 percent diphacinone) and exposure times for field broadcast applications in Hawaii. Masters thesis, University of Hawaii, Honolulu, USA.

Table 1.5: Relative welfare impacts of VTAs affecting carnivores in New Zealand: Ferret (*Mustela furo*); Stoat (*Mustela erminea*); Cat (*Felis catus*). Compound1080 is used in bait formulations for the control of feral cats, and can also affect stoats and ferrets as 'non-target' species through secondary poisoning. Brodifacoum is not used for direct control of any of the three carnivore species; all are 'non-targets' potentially affected by secondary poisoning. Diphacinone is registered for ferret control, but not for feral cats and stoats. Note that PAPP is undergoing registration as a VTA for stoat and feral cat control, but is currently not available for operational use.

	Domain of potential welfare compromise											
Agent	1	2	3	4	5	C	Overall impact	C	Duration	C	Overall grade	C
1080	Mild *(No – Mild)* NDD	No – Mild NDD	Severe *(Mod – Ex)*	Mild *(No – Mild)*	Severe – Extreme *(Mod – Ex)* Nausea Lethargy/weakness Sickness Breathlessness Pain Anxiety	 3 2 1 1 0 0	Severe – Extreme *(Mod – Ex)*	1	Hours	1	6.5 (5.5 – 7.5)	1
Brodifacoum*	Mild – Moderate NDD	Mild *(No – Mod)* NDD	Severe – Extreme	Mild *(No – Mod)* NDD	Severe – Extreme Pain Lethargy/weakness Breathlessness	 3 2 1	Severe – Extreme	2	Days – Weeks	1	7.5 *(6.5 – 8)*	1
Diphacinone	See text											
PAPP	No – Mild NDD	No – Mild NDD	Moderate – Severe *(Mod – Ex)*	Mild *(No – Mod)* NDD	Severe *(Mild – Ex)* Lethargy/weakness Nausea Breathlessness	 3 2 1	Severe *(Mild – Ex)*	1	Minutes – Hours	1	5.5 *(4 – 6.5)*	1

* No direct data for stoats, assume impacts same as for ferrets

1.5.2.12. Sodium fluoroacetate (1080) for carnivores

Basis of welfare assessment and scoring (summary)

Little or no direct data are available for assessing welfare impacts in domains 1, 2 and 4. Ferrets exposed to sublethal doses of 1080 showed food avoidance, while the behaviour patterns of stoats were altered several hours after dosing. As death usually occurred within 12 hours of ingesting poison, impacts in these domains are expected to be mild for all three species. Functional impairment (domain 3) is considered to be severe for all three species. Although symptoms are reported to be highly variable in carnivores (within and between species), neural effects predominated for stoats, ferrets and cats. Poisoned animals progressed from states of hyper-activity and unco-ordinated movement to lethargy, recumbency (paralysis in ferrets), tremors/spasms and tonic/clonic convulsions. Vomiting occurred in all ferrets and was noted occasionally in stoats but not in cats. Progressive alteration in respiration was also noted in cats and stoats.

Given the severe functional impairments observed, carnivores are likely to experience lethargy/weakness, sickness and probably some breathlessness before consciousness is lost. Ferrets and probably stoats likely also experience nausea. Humans poisoned with 1080 report epigastric/abdominal pain, respiratory distress (breathlessness), nausea, dizziness and anxiety; more detailed data are required to assess these experiences in carnivores. Whether carnivores are conscious during or after spasms or convulsions is uncertain, although there is some evidence of a progressive reduction in responsiveness in stoats. Animals conscious during such events or those that recover consciousness afterwards may experience pain, breathlessness and anxiety/fear.

Information required

Most importantly, information on level of consciousness at various times/events after dosing is required to determine the quality and duration of negative affective experiences. Information on time to loss of behavioural reflexes, normal EEG and evoked potentials after dosing is required. In addition, such measures should be used to evaluation level of consciousness immediately before and after spasms/tremors/convulsions. Studies involving administration of analgesic, anti-nauseant and anti-anxiety drugs would shed light on carnivores' experience of pain, nausea and anxiety. More specific information on respiratory compromise, particularly relative to level of consciousness, is required to assess potential experience of breathlessness. Direct information on feeding/drinking behaviour, potential for environmental exposure and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to 1080 section above (under possums).

Mode of toxic action:

Refer to 1080 section above (under possums).

Toxic effects on cats (target species):

Eason & Frampton (1991): Feral cats were observed after they had ingested 1080 bait, equivalent to doses of 0.1–1.3 mg/kg. Deaths occurred with 0.6 mg of 1080 in each bait and all higher doses. Symptoms included…'disorientation, uncoordinated movements and occasional vocalization….the cats became subdued and lethargic before death' The onset of symptoms and time to death was dose-dependent and at doses of 1 mg and above, death occurred within 12 hrs.

McIlroy (1981): Latent period of 1.0–5.6 hrs and times to death 20.7–21.0 hrs. It was noted in general for 1080 poisoning in carnivores there was no constancy in the pattern of symptoms, with as much variation in response between species as between individuals. 'Convulsions were usually preceded by a variety of symptoms….briefly and in rough order, these included restlessness, increasing hyper-excitability or response to stimuli; bouts of trembling; rapid, shallow breathing; incontinence or diarrhoea; excessive salivation; twitching of the facial muscles; nystagmus or bulging eyes with dilated pupils and rapid blinking plus in cats discharge of mucus from the eyes; slight lack of coordination or balance; abrupt bouts of vocalization; and finally sudden bursts of activity…..all affected animals then fall to the ground in a tetanic seizure, with hind limbs or all four limbs and sometime the tail extended rigidly….this tonic phase is followed by a clonic phase in which the animals lie and kick….with the front legs….during this phase the tongue and penis may be extruded and the eyes rolled back….and the teeth ground together. Breathing is rapid but laboured, with some animals partly choking on their saliva. Finally, such individuals begin to relax, breathing more slowly and shallowly and lying quietly with the hind legs still extended by apparently semi-paralyzed. From then on individuals either ….die shortly afterwards; after a delay experience another series of convulsions and then die shortly afterwards….(or) remain lying quietly, scarcely breathing until death…'.

Toxic effects on stoats (secondary non-target species):

Dilks & Lawrence (2000): From a field study of radio-tracked stoats that took egg baits containing 1080, the majority of stoats died within 12 hrs. One stoat was confirmed to have died within 3.5 hrs.

Potter et al. (2006): Stoats were offered 1-g baits containing 0.1 percent 1080 and their subsequent behaviour was monitored by video and direct observation. Twelve of 14 stoats offered the baits ate them voluntarily, and a 13th licked bait off its fur; all 13 died between 1 hr 15 min and 4 hrs 7 min (mean 2 hrs 38 min) later. At first (range 29 min – 2 hrs min, mean 1 hr1 min), their behaviour appeared to be normal. Ataxia and hyperactivity were the first behavioural signs of poisoning, and lasted for 2 min – 1 hr 40 min (mean 26 min). This was followed by recumbency with convulsions and rapid breathing (range 16 min to 2 hrs, mean 58 min), then recumbency with limited activity and progressively shallower breathing prior to death (range 1–51 min, mean 33 min). Stoats became non-responsive to a light being turned on, or to touch once recumbency became sustained.

Spurr (2000): Of the two captive stoats that ate 0.5 mg of 1080 per kg body weight, one (a male) survived and the other (a female) died within 12 hrs. All seven captive stoats that ate 0.75 mg/kg or more died within 12 hrs.

Unpublished data, Landcare Research: Stoats died between 3.5 and 32 hrs (mean of 12 hr 50 min) after eating a 1080 egg bait. The first signs of illness behaviours were observed between 2 and 4 hrs after eating the toxic egg (mean 2 hr 41 min). Stoats were then observed outside

the nest box, often lying or standing in an abnormal stance. Following this there would be periods of tremors throughout the bodies leading to tonic–clonic convulsions and paddling of the limbs while lying on their side. These periods of convulsive activity were first seen 2.75 hrs after eating 1080 eggs and continued for 1–2 hrs and then less frequently until death. Over all observations, these nervous system responses were observed 39 percent of the time, with abnormal lying, sternal recumbency or lying prostrate a further 32 percent of the time. Only one stoat vomited (of four), 1 hr after eating the 1080 egg and two of the four stoats vocalized on more than one occasion during the convulsive events.

Toxic effects on ferrets (secondary non-target species):

Hornshaw et al. (1986): Note this was LC50 testing with captive ferrets, so represents some intentional sublethal and lethal exposures. Subacute dietary exposure to 1080 resulted in dose dependent decreases in body weights and feed consumption. Clinical signs of 1080 poisoning in adult ferrets included food avoidance, incoordination or paralysis (especially of the hindquarters) and occasional unconsciousness. No gross lesions were noted at necropsy in ferrets that had died of poisoning.

Unpublished data, Landcare Research: Ferrets which ate 1080-laced chicks died on average 8 hr 29 min later (range 6 hr 46 min – 9 hr 34 min). Although far more active than stoats, the first signs of poisoning were similar (i.e. lying) and occurred 30–100 min after eating the chick. All ferrets vomited, a mean of 54 ± 6 min after eating the chicks. The first convulsions occurred 99 ± 15 min after eating the chicks and were closely followed by periods of complete body rigidity (tonic seizure). A typical convulsive event would include a seizure, often with head thrashing and legs paddling, lasting 1–10 s, interspersed with lying stiff on the side with the head arched fully back and the legs stiff, sometimes then interspersed by tremors. This pattern of convulsive events lasted for 2 hrs and then ferrets tended to lie until death, some 5 hrs later. All ferrets vocalized; this usually occurred during a convulsive event.

Toxic effects on humans:

Refer to 1080 section above (under possums).

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1.5.2.13. Brodifacoum for carnivores

Basis of welfare assessment and scoring (summary)

No direct data are available on which to base a welfare assessment for domains 1, 2 or 4, except that ferrets showed reduced feed intake several days before death. There is no published information on the effects of brodifacoum on stoats, except that they may take longer to die than other small carnivores. For the purpose of this evaluation we have assumed that the effects would be similar to those observed in ferrets. Functional impairments are consistent with the anti-coagulant actions of brodifacoum and include internal and external haemorrhages in both cats and ferrets. Ferrets also showed laboured breathing and tremors, while cats appeared reluctant to move and moved abnormally.

There is no evidence of reduced consciousness before death and severe to extreme negative affective states are likely to be experienced. These include: pain due to haemorrhaging; lethargy/weakness due to blood loss; and breathlessness if haemorrhaging occurs in respiratory structures. Poisoned humans report pain and/or breathing difficulties depending on the site of haemorrhage, as well as dizziness and reduced motor capabilities.

Information required

Comprehensive studies supporting assessment of welfare impacts of brodifacoum on stoats are required. For all three species, more detailed information on time to onset of symptoms would allow more accurate assessment of the duration of negative affective experiences. In addition, studies involving administration of analgesic drugs would provide information on animals' experience of pain during the days after dosing. Direct information on feeding/drinking behaviour, potential for environmental exposure and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:
Refer to Brodifacoum section above (under possums).

Mode of toxic action:
Refer to Brodifacoum section above (under possums).

Toxic effects on cats:

Alterio (1996): A field study showed that feral cats in NZ can be secondarily poisoned (confirmed by detection of brodifacoum in liver) after brodifacoum baiting for rabbit control.

Morgan & Miekle (1995): Cats that ate lethal doses of brodifacoum took 7–12 days to die and those that ate sublethal doses were affected by the toxin for up to 20 days before recovering. Affected cats haemorrhaged around the mouth, ears and anus, and their eye-whites turned red.

Dead cats had extensive haemorrhaging under the skin and around the kidneys and heart. Cats typically became reluctant to move, and some appeared uncomfortable while moving.

Toxic effects on stoats:

Alterio (1996): A field study showed that stoats in NZ can be secondarily poisoned (confirmed by detection of brodifacoum in liver) after brodifacoum baiting for rabbit control.

Brown et al. (1998): From a field study of radio-tracked stoats, it was estimated that they took 13–29 days to die after application of brodifacoum bait for possum and rat control.
Toxic effects on ferrets:

Alterio (1996): A field study showed that ferrets in NZ can be secondarily poisoned (confirmed by detection of brodifacoum in liver) after brodifacoum baiting for rabbit control.

Ogilvie et al. (1996): Ferrets fed brodifacoum in food behaved normally until 1–2 days before death, when they showed a loss of appetite, laboured breathing, tremors, occasional bleeding from the anus and pale nasal mucous membranes consistent with anaemia. Average time to death in females was 8 days, and in males ranged from 9–11 days. Necropsy showed consistent internal haemorrhaging.

Toxic effects on humans:
Refer to Brodifacoum section above (under possums).

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Ogilvie, S. C., Spurr, E. B., Young, N., and Eason, C. T. (1996) A field baiting strategy for the control of ferrets. Landcare Research contract report LC9596/129. Prepared for the Animal Health Board, New Zealand, 15p.

1.5.2.14. Diphacinone for carnivores

Basis of welfare assessment and scoring (summary)

Overall, there is insufficient evidence to assess the welfare impacts of diphacinone on ferrets. Considering the identical mode of action and similar symptoms (anorexia for several days prior to death) and pathology reported (internal and external haemorrhage), the welfare

impacts are likely to be similar to those of brodifacoum. The duration of symptoms (1–2 days before death) is reported to be similar for both brodifacoum and diphacinone.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to Brodifacoum section above (under possums).

Mode of toxic action:

Refer to Brodifacoum section above (under possums).

Toxic effects on ferrets:

Ogilvie et al. (1996): Average time to death in male ferrets was 10.5 days (range 7–12 days). All ferrets that died had internal haemorrhaging consistent with anticoagulant poisoning.

Spurr et al. (2005): The average time to death in ferrets that ingested diphacinone bait was 9.3 days (range 5–17 days, n=25). The ferrets that died appeared to behave normally until 1–2 days before death when they showed loss of appetite and external bleeding. Necropsy showed all ferrets had subcutaneous or internal bleeding.

Toxic effects on humans:

Refer to Diphacinone rodents and Brodifacoum possums.

References

Ogilvie, S. C., Spurr, E. B., Young, N., and Eason, C. T. (1996). A field baiting strategy for the control of ferrets. Landcare Research contract report LC9596/129. Prepared for the Animal Health Board, New Zealand, 15p.

Spurr, E. B., Ogilvie, S. C., Morse, C. W., and Young, J. B. (2005). Development of a toxic bait and baiting strategy for control of ferrets (*Mustela furo*) in *New Zealand. New Zealand Journal of Zoology* 32, 127–136.

1.5.2.15. p-aminopropiophenone (PAPP) for carnivores

Basis of welfare assessment and scoring (summary)

No direct data are available on which to base a welfare assessment for domains 1, 2 or 4; however, based on the relatively short times to death (less than one hour for stoats; 30 mins to 4 hrs, depending on dose, for cats), only mild impacts are expected in these domains. Functional impairments may include incoordination, inability to behave normally (e.g. stand), retching/vomiting (appear to be more common in cats), respiratory compromise and convulsions (stoats). Overall, there is currently insufficient evidence to assess the welfare impacts of PAPP on stoats and cats. In particular, it is not clear whether the animals experience significant breathlessness prior to loss of consciousness. This uncertainty is

reflected in the very wide range of scores assigned for domains 3 and 5 and overall for PAPP (mild to extreme).

Increased respiration (and associated negative sensations of breathlessness) is partly stimulated by reduced oxygen content in arterial blood, sensed by peripheral chemoreceptors (Nunn, 1994). A progressive increase in percent MetHb results in a concomitant decrease in O2 saturation of haemoglobin but does not significantly decrease total blood oxygen content, at least in the early stages of methaemoglobinemia (Nunn, 1994). Therefore, it may be possible that animals poisoned by PAPP fall unconscious and die without experiencing breathlessness. Rising percent MetHb will however, reduce oxygen delivery to tissues. At some point, hypoxic tissues will switch to anaerobic metabolism, producing lactate and causing a drop in blood pH which will also stimulate peripheral chemoreceptors and increase the drive to breathe. The point at which these events occur, relative to cerebral hypoxia and loss of consciousness in cats and stoats, is unknown.

At least one study has reported altered respiration in conscious stoats after dosing with PAPP; breathing became rapid and shallow approximately 30 mins after dosing, thereafter becoming increasingly laboured (Fisher et al., 2005). In addition, dyspnoea (breathing discomfort) is reported by humans in the early stages of methaemoglobinemia (30 percent MetHb) (Coleman and Coleman, 1996). In contrast, other studies make no mention of respiratory symptoms in PAPP-poisoned animals (e.g. foxes: Marks et al., 2004).

Of greatest importance is the timing of breathlessness relative to reduction or loss of consciousness. As noted, humans report dyspnoea, along with nausea and tachycardia when MetHb reaches 30 percent. At around 55 percent MetHb, lethargy and stupor set in and consciousness is reduced. Fisher et al. (2005) report that PAPP-poisoned stoats became less responsive to disturbance (noise and touch) around the time that altered respiration was first observed. Stoats became completely unresponsive just before death, when breathing was slow and irregular or gasping. These findings suggest that consciousness may be reduced when respiratory stimulation (and breathlessness) is increasing.

Information required

Most importantly, more detailed observations of respiratory symptoms and level of consciousness are required to accurately assess welfare impacts of PAPP. Information on level of consciousness at various times/events after dosing is required to determine the quality and duration of negative affective experiences such as breathlessness and nausea. More detailed information on time to loss of behavioural reflexes, normal EEG and evoked potentials after dosing would improve our understanding of the welfare impacts of progressive methaemoglobinemia in PAPP-poisoned animals. In addition, percent MetHb, blood pH and other physiological indicators relating to respiratory stimulation should be measured and correlated with behavioural changes and indicators of consciousness in cats and stoats poisoned with PAPP. Direct information on feeding/drinking behaviour, potential for environmental exposure and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

In general, assessments of PAPP poisoning in target pest species (foxes in Australia, stoats and cats in NZ) indicate that it is relatively more humane than currently-used pesticides such as 1080, through both speed of action and the toxic effects produced.

Mode of toxic action:

Oxidisation of the haem iron in red blood cells from the ferrous salt (Fe2+) to the ferric state (Fe3+) to form methaemoglobin (MetHb). MetHb is incapable of carrying oxygen and elevated levels cause anaemia, respiratory distress with resultant cyanosis, with death occurring if the elevation of MetHb is high enough (>70 percent required in humans).

Toxic effects on cats:

Cats are highly susceptible to orally administered PAPP. Symptoms of toxicosis include vomiting, salivation and lethargy occurring between 16–204 mins and death between 37– 246 mins, dependent upon dose. Cats exposed to lethal doses of PAPP tended to lose consciousness without spasms or convulsions.

Toxic effects on stoats:

Fisher et al. (2005): Within approximately 20 min of dosing with PAPP, movements of stoats appeared slower and less coordinated, with cyanosis (a bluish discoloration of the skin resulting from inadequate oxygenation of the blood) around the lips and nose also first observed within this time. By 30 min, stoats were less responsive to disturbance, (e.g., noise or touch), often lying curled with head down and displaying rapid, shallow respiration. After this, in comparison with control stoats, the behaviour of PAPP-dosed stoats was visibly affected. They showed little activity, often lying in a curled position, with cyanosis evident in exposed skin. Some stoats first displayed excessive salivation at this stage, with respiration remaining rapid and increasingly laboured. Any movement attempted appeared very uncoordinated, often with legs splayed sideways and an inability to raise their heads. Excessive salivation was observed in 10 of 24 PAPP-dosed stoats, and vomiting or retching was observed in two of 24 stoats. Stoats that died appeared to lose consciousness just prior to death (unresponsive to noise or touch), with breathing slow, irregular and 'gasping'. Just prior to death, some stoats showed writhing or paddling movements, which appeared involuntary. Some animals exhibited clonic convulsions just before death. Death usually occurred within 40 min of dosing, except for the lowest-dose group (9.38 mg/kg), where the mean time to death (n = 4) was just over 1 hr after dosing.

Toxic effects on humans:

At sublethal levels, other than bluish lips, no physical, intellectual, or psychological problems were observed. In all subjects, appetite was good and there were no renal problems (urine flow and diuresis were normal). Two showed slight changes in AP, AQRS, and AT axes in the ECG trace. At very high doses, PAPP was mildly haemolyzing.

Coleman & Coleman (1996): Dyspnoea, nausea and tachycardia occur at MetHb levels of greater than or equal to 30 percent, while lethargy, stupor and deteriorating consciousness occur as methaemoglobin levels approach 55 percent. Higher levels may cause cardiac

arrhythmias, circulatory failure and neurological depression, while levels of 70 percent are usually fatal.

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Table 1.6: Relative welfare impacts of VTAs affecting rabbits (*Oryctolagus cuniculus*) in New Zealand. Compound 1080, pindone and phosphorus are all registered for rabbit control.

	Domain of potential welfare compromise											
Agent	1	2	3	4	5	C	Overall impact	C	Duration	C	Overall grade	C
1080	Mild *(No – Mod)*	No – Mild NDD	Severe – Extreme *(Mod – Ex)*	Mild *(No – Mod)* NDD	Severe *(Mod – Ex)* Lethargy/weakness Breathlessness Pain Anxiety/fear	 3 1 0 0	Severe *(Mod – Ex)*	1	Hours	1	6 *(6 – 7)*	1
Pindone	Moderate *(No – Sev)* NDD	No – Mild *(No – Sev)* NDD	Extreme *(Sev – Ex)*	Moderate *(No – Sev)* NDD	Extreme *(Sev – Ex)* Pain Breathlessness Lethargy/weakness Sickness Dizziness Anxiety/fear	 3 0 0 0 0 0	Extreme *(Sev – Ex)*	2	Days	1	8 *(7.5 – 8)*	1
Phosphorus	See text											

1.5.2.16. Sodium fluoroacetate (1080) for rabbits

Basis of welfare assessment and scoring (summary)

Little or no direct data are available for assessing welfare impacts in domains 1, 2 and 4, except that there is a reduction in feeding for several hours. Based on the time to death in lethally dosed rabbits (hours), any effects in these domains are likely to be mild. Functional impairments are judged to be severe to extreme, based on observations of respiratory distress relating to pulmonary oedema and haemorrhage, the generalized effects of cardiac dysfunction, and clonic/tonic convulsions. Whether rabbits are conscious during or after convulsions is unknown. Animals conscious during such events or those that recover consciousness afterwards may experience pain, breathlessness and anxiety/fear.
Based on observations of behaviour and pathology, rabbits are likely to experience lethargy/weakness and breathlessness due to respiratory compromise. Humans poisoned with 1080 report epigastric/abdominal pain, respiratory distress (breathlessness), nausea, dizziness and anxiety; however, there is currently insufficient information to assess these experiences in rabbits.

Information required

Most importantly, information on level of consciousness at various times/events after dosing is required to determine the quality and duration of negative affective experiences. Information on time to loss of behavioural reflexes, normal EEG and evoked potentials after dosing is required. In addition, such measures should be used to evaluate level of consciousness immediately before and after spasms/tremors/convulsions. Studies involving administration of analgesic, anti-nauseant and anti-anxiety drugs would shed light on rabbits' experience of pain, nausea and anxiety. More specific information on respiratory compromise, particularly relative to level of consciousness, is required to assess potential experience of breathlessness. Direct information on feeding/drinking behaviour, potential for environmental exposure and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to 1080 section above (under possums).

Mode of toxic action:

Refer to 1080 section above (under possums).

Toxic effects on rabbits:

Gooneratne et al. (1994): In rabbits administered a sublethal dose of 1080 (0.1 mg/kg), clinical signs were not observed but five of thirty six rabbits showed abnormal electrocardiogram readings – four showed sinus tachycardia and one ventricular fibrillation. Times to death in twelve lethally-dosed (0.8 mg/kg) rabbits ranged from 1 to 7.5 hrs, with 9 of these dying within 3 hrs.

Quin & Clark (1947): Rabbits administered 1080 by injection '…. usually showed no signs for three or four hours when sudden spasms of respiratory distress occurred, these were often violent in character and accompanied by the emanation of froth from the nose and mouth'. Post-mortem lesions were indicative of heart failure – there was a generalised cyanosis and venous congestion, especially severe in the heart and lungs. In addition the lungs showed marked oedema and in some cases pulmonary haemorrhage.

Williams (1996): provides a summary of the progression of 1080 poisoning in rabbits: 'Extracardiac effects, if present at all, are masked by the magnitude of the cardiac effects which characterize fluoroacetate poisoning in the rabbit…..No clinical change in the condition of a poisoned rabbit is discernable for 0.5 –2 hrs….The rabbit may then stop feeding if it is doing so and develop obvious weakness. The rabbit remains fully conscious at this time but may lie with its head to one side between extended forepaws and can be roused. The lethargic state lasts for about two hours when an LD100 dose of fluoroacetate is administered, but the period is dose-dependent and may vary from one to twelve hours. There is no hypermotility of the gut or tooth grinding which is considered to be a typical expression of pain in the rabbit. A sudden violent episode of clonic convulsion sometimes associated with laryngeal stridor and a sharp cry invariably follows. Opisthotonus, mydriasis and retinal blanching develop rapidly, followed by progressive relaxation a few gasping respiratory movements and death. Twenty percent of rabbits will convulse and die immediately after the latent period without showing lethargy'. Williams' (1996) review further discussed cardiac effects of 1080 poisoning in rabbits and concluded that convulsions in rabbits occur while they are unconscious.

McIlroy (1982): Latent periods of 1.1 to 10.1 hrs and times to death of 3.0 to 44.3 hrs were reported in rabbits dosed with 1080 in a trial to calculate lethal dose values. McIlroy observed ' all affected rabbits exhibited increased sensitivity to noise or disturbance – thereafter some recovered while the remainder experienced one or more convulsions, during which they coughed and squeaked, kicked with their hind legs and struggled for breath. Most convulsing rabbits died but a few recovered. All rabbits that survived began recovering 5.0–23.2 hrs after being dosed'.

Toxic effects on humans:
Refer to 1080 section above (under possums).

References

Gooneratne, R., Dickson, C., Wallace, D., Eason, C. T., Fitzgerald, H., and Wright, G. (1994). Plasma and tissue 1080 in rabbits after lethal and sub-lethal doses. In: Seawright, A.A. and Eason, C.T. (Eds). Proceedings of the Science Workshop on 1080, 12–14 December 1993, Christchurch, New Zealand. Royal Society of New Zealand, Wellington, 67–73.

McIlroy, J. C. (1982). The sensitivity of Australian animals to 1080 poison III. Marsupial and eutherian herbivores. *Wildlife Research* 9, 487–503.

Quin, J. I. and Clark, R. (1947). Studies on the action of potassium monofluoroacetate (CH2FCOOK) [*Dichapetalum cymosum* (Hook) Engl.] toxin on animals. *Onderstepoort Journal of Veterinary Science and Animal Industry* 22(1), 77–82.

Williams, D. (1996). Animal welfare aspects of the use of sodium fluoroacetate to poison wild rabbits. In: Fisher, P. and Marks, C. A. (Eds). Humaneness and vertebrate pest control: Proceedings of the seminar held on March 27, 1996, Melbourne, Agriculture Victoria, Dept.

of Natural Resources and Environment. Report series / Victorian Institute of Animal Science. Vertebrate Pest Research Unit, 37–41.

1.5.2.17. Pindone for rabbits

Basis of welfare assessment and scoring (summary)

Overall there is insufficient published information to assess the welfare impacts of pindone on rabbits. As there are no data available on behavioural symptoms, the current evaluation is based on reports of pathology and extrapolation from human poisoning reports. No direct data are available on which to base a welfare assessment for domains 1, 2 or 4. Scores for these domains are based on the reported times to death (days) and behavioural symptoms noted in other anti-coagulant poisoned species (anorexia, lethargy); however, the uncertainty about impacts is reflected in the wide range of scores in each domain (No – Severe). Functional impairments are consistent with the anti-coagulant actions of pindone and include external and internal haemorrhages in various locations likely to cause severe pain and lethargy/weakness due to blood loss (anaemia). There is no evidence of reduced consciousness before death and severe to extreme negative affective states are likely to be experienced. Poisoned humans report pain and/or breathing difficulties depending on the site of haemorrhage, as well as dizziness and reduced motor capabilities; however, there is currently insufficient information to assess these effects in rabbits.

Information required

Comprehensive studies supporting assessment of welfare impacts of pindone on rabbits are required. In particular, more detailed information on behavioural responses to poisoning and time to onset of symptoms would allow more accurate assessment of the quality and duration of negative affective experiences. Specific information on respiratory compromise (reported in other species due to pulmonary haemorrhage) is required to assess potential breathlessness. In addition, studies involving administration of analgesic drugs would provide information on animals' experience of pain during the days after dosing. Direct information on feeding/drinking behaviour, potential for environmental exposure and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to Brodifacoum section above (under possums).

Mode of toxic action:

Refer to Brodifacoum section above (under possums).

Toxic effects on rabbits:

Eason & Jolly (1993): Six rabbits were dosed orally with 25 mg/kg pindone. Six days after dosing, mean haematocrit was reduced from 0.41 to 0.14 percent, while the one-stage

prothrombin time increased from 7.1 s to more than 120 s and activated partial prothrombin time increased from baseline (26.1 s) to more than 180 s. Half the rabbits died within 6 days with extensive haemorrhaging in the abdomen and thorax. Affected rabbits appeared weak, with colourless eyes and pale mucous membranes indicating profound anaemia.

Oliver & Wheeler (1978): In rabbits which died following chromic administration of pindone, the most characteristic symptom was widespread haemorrhage throughout the muscles on the posterior aspect of both hind legs. Other common symptoms were massive leakage of blood into the abdominal cavity, haemorrhage in muscle around the rib cage and in the submandibular region and numerous smaller subcutaneous over the body. Less common were leakage of blood into the pericardium and cerebral haemorrhage. Small focal haemorrhages were found in most internal organs. Skin and externally visible mucous membranes were almost colourless, and bleeding from external orifices was often apparent. Days to death ranged from 5 to 14 days.

Robinson & Wheeler (1983): In a field study of radio-tracked rabbits during pindone baiting, the first death occurred on day 6. Losses followed at a' fairly steady' rate over the next 7 days and the last of the 22 rabbits with transmitters died on day 16.

Toxic effects on humans:

Refer to Brodifacoum section above (under possums).

References

Eason, C. T. and Jolly, S. E. (1993). Anticoagulant effects of pindone in the rabbit and Australian brushtailed possum. *Wildlife Research* 20, 371–374.

Oliver, A. J. and Wheeler, S. H. (1978). The toxicity of the anticoagulant pindone to the European rabbits *Oryctolagus cuniculus* and the sheep *Ovis aries*. *Australian Wildlife Research 5*, 135–142.

Robinson, M. H. and Wheeler, S. H. (1983). A radio tracking study of four poisoning techniques for control of the European rabbit *Oryctolagus cuniculus*. *Australian Wildlife Research* 10, 513–520.

1.5.2.18. Phosphorus for rabbits

Basis of welfare assessment and scoring (summary)

There is currently insufficient information available to assess the welfare impacts of phosphorus on rabbits.

Information required

Comprehensive studies supporting assessment of welfare impacts of phosphorus on rabbits are required. In particular, detailed information on behavioural responses to poisoning,

pathology/functional impairment, and time to loss of consciousness and death would allow assessment of the quality and duration of negative affective experiences.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to Phosphorus section above (under possums).

Mode of toxic action:
Refer to Phosphorus section above (under possums).

Toxic effects on rabbits:

In rabbits orally exposed to 7 mg/kg phosphorus, the latent period before signs of poisoning was 2–4 days (Hone and Mulligan, 1982).

Toxic effects on humans:

Refer to Phosphorus section above (under possums).

References

Hone, J. and Mulligan, H. (1982). Vertebrate pesticides. Department of Agriculture, New South Wales: Sydney.

Table 1.7: Relative welfare impacts of VTAs affecting pigs (*Sus scrofa*) in New Zealand. There are currently no VTAs registered for feral pig control in New Zealand, however secondary 1080 poisoning and non-target phosphorus poisoning of pigs is known to occur. Note that sodium nitrite is undergoing development as a VTA for feral pig control in Australia and New Zealand, but is not currently available operationally.

	Domain of potential welfare compromise											
Agent	1	2	3	4	5	C	Overall impact	C	Duration	C	Overall grade	C
1080	Mild *(No – Mild)* NDD	Mild *(No – Mild)* NDD	Severe – Extreme	Mild *(No – Mod)* NDD	Severe – Extreme Sickness Nausea Lethargy/weakness Breathlessness Pain Dizziness Anxiety	 3 3 3 2 1 0 0	Severe – Extreme	3	Hours – Days	2	6.5 *(6 – 7.5)*	2
Phosphorus	Mild *(No – Mod)*	Mild *(No – Mild)* NDD	Severe *(Mild – Ex)*	Mild – Moderate	Severe *(Sev – Ex)* Lethargy/weakness Nausea Sickness Pain	 3 1 1 1	Severe *(Sev – Ex)*	1	Days	1	7 *(7 – 8)*	1
Sodium nitrite	No *(No – Mild)* NDD	No *(No – Mild)* NDD	Severe *(Mild – Ex)*	No *(No – Mild)* NDD	Severe *(Mild – Ex)* Nausea Breathlessness Lethargy/weakness Pain	 2 1 1 0	Severe *(Mild – Ex)*	1	Minutes *(Mins – Hrs)*	1	5.5 *(3 – 7)*	1

1.5.2.19. Sodium fluoroacetate (1080) for pigs

Basis of welfare assessment and scoring (summary)

No direct data are available for assessing welfare impacts in domains 1, 2 and 4. Scores for food/water deprivation are based on the duration of sickness behaviour and lethargy/weakness observed as well as likely nausea, although it is unknown whether poisoned pigs experience hunger/thirst. Likewise, potential environmental challenge and behavioural/interactive alterations are based on the quality and duration of altered behaviour (hours to days). Functional impairments are judged to be severe to extreme, based on reports of frequent, intense retching and vomiting in pigs, as well as lethargy/weakness, cardiac and respiratory effects. Pathological effects include gastritis and degeneration of heart, liver and kidneys. In the later stages of 1080 poisoning pigs show severe neural effects including spasms/tremors and convulsions. Whether animals are conscious during or after these events is unknown. Animals conscious during such events or those that recover consciousness afterwards may experience pain, breathlessness and anxiety/fear.

Conscious experience of nausea, lethargy/weakness, sickness and breathlessness (due to metabolic acidosis from accumulating citrate) seem likely. Humans poisoned with 1080 also report epigastric/abdominal pain, dizziness and anxiety; however, there is currently insufficient information to assess these experiences in pigs. Nor is there any published information upon which to evaluate the possibility of diminishing awareness which could reduce the duration of such negative affective experiences in pigs.

Information required

Most importantly, information on level of consciousness at various times/events after dosing is required to determine the quality and duration of negative affective experiences. Information on time to loss of behavioural reflexes, normal EEG and evoked potentials after dosing is required. In addition, such measures should be used to evaluate level of consciousness immediately before and after spasms/tremors/convulsions. Studies involving administration of anti-nauseant and anti-anxiety and analgesic drugs would shed light on pigs' experience of nausea, anxiety and pain. More specific information on respiratory compromise, particularly relative to level of consciousness, is required to assess potential experience of breathlessness. Direct information on feeding/drinking behaviour, potential for environmental exposure and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to 1080 section above (under possums).

Mode of toxic action:
Refer to 1080 section above (under possums).

Toxic effects on pigs:

Hone & Kleba (1984): Seventeen of 19 pigs dosed with 1080 (sublethal or lethal exposures) vomited.

McIlroy (1983): 'First signs of poisoning appeared within 1.9–47.3 hrs of dosing (mean 10.9 hrs, median 6.2 hrs), and were either vomiting or increasing lethargy and laboured respiration, often with a white froth appearing around the mouth and nostrils…Deaths occurred 2.8–80.0 hrs (mean 23.4 hrs, median 16.1 hrs) after pigs were dosed with 1080. Usually affected animals simply lay quietly, breathing slowly and laboriously until death…only the lower time limits and median times before signs of poisoning appeared, and the lower time limits to death…decreased markedly as the dose of 1080 increased'.

O'Brien (1988): '1080 exerted a potent emetic action on feral pigs, eliciting vomiting in 98 percent (78/80) of animals dosed. Vomiting was the most consistent sign of intoxication, commencing a median 49 min (range 10–350 min) after 1080 ingestion…the frequency of vomiting was high, averaging 13.6 episodes per pig over all doses tested…Median time from 1080 ingestion until death…was 244 min (range 131–7200 min)…Most pigs (89 percent) died between 131 and 390 min…however four animals died between 1 and 5 days after poisoning. Time until death had a significant negative correlation with log10 1080 dose.'.

Schwarte (1947): 'Shortly after the administration the swine appeared restless and hypersensitive. Increased respirations and heart action were observed. One-half hour to two hours following administration…there was nausea and vomiting…weakness and depression with an ever-increasing heart beat were evident. Convulsions and spasms in some animals occurred while others soon became comatose. Those subjects showing severe nervous reactions moved about the pens squealing, then fell down on their sides with rapid leg motions simulating running movements. This action was followed by convulsions and death…those animals showing a comatose condition would maintain a sterna recumbent position or lie on one side shivering. Prior to death, heart action was so rapid and weak that the pulse rate could not be determined…The outstanding characteristic pathology changes…were the haemorrhages on the heart, cardiac degeneration and the dark tar-like colour of the blood. The animals which received the higher doses had the most severe lesions. They frequently developed a mild gastritis with degenerative changes in the liver and kidneys. In several cases, the bladder was greatly distended with dark brownish coloured urine. Petechial haemorrhages appeared on the pericardial sac of two animals'.

Toxic effects on humans:

Refer to 1080 section above (under possums).

References

Hone, J. and Kleba, R. (1984). The toxicity and acceptability of warfarin and 1080 poison to penned feral pigs. *Australian Wildlife Research* 11, 103–111.

McIlroy, J. C. (1983). The sensitivity of Australian animals to 1080 poison V. The sensitivity of feral pigs, Sus scrofa, to 1080 and its implications for poisoning campaigns. *Australian Wildlife Research 10*, 139–148.

O'Brien, P. H. (1988). The toxicity of sodium monofluoroacetate (compound 1080) to captive feral pigs, Sus scrofa. *Australian Wildlife Research* 15, 163–170.

Schwarte, L. H. (1947). The toxicity of sodium monofluoroacetate (1080) for swine and chickens. *Journal of the American Veterinary Medical Association* 114, 301–303.

1.5.2.20. Phosphorus for pigs

Basis of welfare assessment and scoring (summary)

Little direct data is available for assessing impacts in domains 1, 2 and 4: feed intake declines suddenly after poisoning and poisoned pigs do not withdraw from an approaching human, suggesting mild to moderate impacts in domain 4. Functional impairment is considered to be severe, based on prostration, reduced activity and the occurrence of clonic convulsions. Whether pigs are conscious during or after these convulsions is unknown. Animals conscious during such events or those that recover consciousness afterwards may experience pain, breathlessness and anxiety/fear. Pathological effects include haemorrhage and degeneration of the gastrointestinal tract and liver and external haemorrhages. Pigs are likely to experience lethargy/weakness, and probably nausea and sickness as liver failure progresses. In addition, pigs may experience epigastric/abdominal pain which has been reported by human victims.

Information required

More specific information on the time to onset of sickness behaviours after ingestion of phosphorus and the quality/intensity of those behaviours is required. In addition, data on level of consciousness at various times/events after dosing are required to determine the quality and duration of negative affective experiences. These data might include time to loss of behavioural reflexes, normal EEG and evoked potentials. In addition, such measures should be used to evaluate level of consciousness immediately before and after convulsions. More detailed information on feeding/drinking behaviour, potential for environmental exposure and alterations in behaviour is required to more accurately assess impacts in domains 1, 2 and 4. Studies involving administration of analgesic and anti-nauseant drugs would shed light on pigs' experiences of pain and nausea after ingestion of phosphorus paste.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to Phosphorus section above (under possums).

Mode of toxic action:

Refer to Phosphorus section above (under possums).

Toxic effects on pigs (primary non-target species):

O'Brien & Lukins (1990): After phosphorus poisoning, most pigs appeared lethargic and depressed. Pigs typically died 2–4 days after intoxication. Vomiting occurred rarely (3/24). Most pigs moved and ate little, reflected in a sudden decline of food consumption after

poisoning. Moribund animals lay on their sides, did not withdraw when approached, paddled while lying and vocalized occasionally. External evidence of poisoning was not common at post mortem but included per rectum haemorrhage (7/21) and haemorrhagic mucus in the nares (1/21). Gross pathology was restricted to the liver and gastrointestinal tract. Livers were characteristically rigid, granular and friable with extensive petechial haemorrhage (4/21). The gall bladder wall of two animals was abnormally thickened. Livers showed large areas of coagulative necrosis and replacement haemorrhage. There was minimal fatty change and no significant inflammatory response. Gross gastrointestinal pathology consisted of areas of haemorrhage in the stomach (5/21), small intestine (7/21) and rectum (7/21).

Toxic effects on humans:
Refer to Phosphorus section above (under possums).

References

O'Brien, P. H. and Lukins, B. (1990). Comparative dose-response relationships and acceptability of warfarin, brodifacoum and phosphorus to feral pigs. *Australian Wildlife Research* 17, 101–112.

1.5.2.21. Sodium nitrite for pigs

Basis of welfare assessment and scoring (summary)

No direct data are available on which to base a welfare assessment for domains 1, 2 or 4; however, based on the relatively short times to death (min to hrs), only mild impacts are expected in these domains. Functional impairments include frequent vomiting, progressively more laboured or difficult breathing, and neural effects including incoordination and convulsions near the time of death. Overall, there is currently insufficient information to accurately assess the welfare impacts of sodium nitrite on pigs. In particular, it is not clear whether the animals experience significant breathlessness prior to loss of consciousness. This uncertainty is reflected in the very wide range of scores assigned for domains 3 and 5 and overall for sodium nitrite (mild to extreme).

Increased respiration (and associated negative sensations of breathlessness) is partly stimulated by reduced oxygen content in arterial blood, sensed by peripheral chemoreceptors (Nunn 1994). A progressive increase in percent MetHb results in a concomitant decrease in O2 saturation of haemoglobin but does not significantly decrease total blood oxygen content, at least in the early stages of methaemoglobinemia (Nunn 1994). Therefore, it may be possible that animals poisoned by sodium nitrite fall unconscious and die without experiencing breathlessness. Rising percent MetHb will however, reduce oxygen delivery to tissues. At some point, hypoxic tissues will switch to anaerobic metabolism, producing lactate and causing a drop in blood pH which will also stimulate peripheral chemoreceptors and increase the drive to breathe. The point at which these events occur, relative to cerebral hypoxia and loss of consciousness in pigs, is unknown.

Of greatest importance is the timing of breathlessness relative to reduction or loss of consciousness. Humans report dyspnoea, along with nausea and tachycardia when MetHb reaches 30 percent. At around 55 percent MetHb, lethargy and stupor set in and consciousness is reduced (Coleman & Coleman 1996). One study on pigs reports difficult breathing or

apnoea (not breathing at all) 30–60 min after oral gavage with sodium nitrite, and this difficulty is reported to worsen until the time of death. However, no information is given on the level of consciousness at the onset of breathing difficulty or after this point, so it is impossible to tell for how long, if at all, pigs experience negative affect (breathlessness).

Information required

Most importantly, more detailed observations of respiratory symptoms and level of consciousness are required to accurately assess welfare impacts of sodium nitrite. Information on level of consciousness at various times/events after dosing is required to determine the quality and duration of negative affective experiences such as breathlessness and nausea. More detailed information on time to loss of behavioural reflexes, normal EEG and evoked potentials after dosing would improve our understanding of the welfare impacts of progressive methaemoglobinemia in sodium nitrite-poisoned pigs. In addition, % MetHb, blood pH and other physiological indicators relating to respiratory stimulation should be measured and correlated with behavioural changes and indicators of consciousness in pigs poisoned with sodium nitrite. Direct information on feeding/drinking behaviour, potential for environmental exposure and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

None found.

Mode of toxic action:

Sodium nitrite has essentially the same mode of action as PAPP, but has less selective and relatively lower oral toxicity. Ingestion of sufficient sodium nitrite causes oxidization of the haem iron in red blood cells from the ferrous salt (Fe2+) to the ferric state (Fe3+) to form methaemoglobin (MetHb). MetHb is incapable of carrying oxygen and respiration distress and cyanosis results, with death occurring if the elevation of MetHb is high enough.

Toxic effects on pigs:

Cowled et al. (2008): Pigs that received sodium nitrite doses of 135 mg/kg or more died. Mean time to death was 106 ± 75 min (range 42–130 min). The mean peak MetHb concentration in pigs that died was 82 percent. Pigs given a lethal dose become dyspnoeic (not breathing or able to breathe except with difficulty) 30–60 min after oral gavage and this gradually worsened until marked gasping just before death. Incoordination, paddling and short convulsive seizures occurred in two of nine pigs. Seizures occurred close to the time of death and were caused by terminal hypoxia.

Pigs given a lower dose (90 mg/kg) generally show a prolonged period of lethargy that lessened over 4–6 hrs. Vomiting occurred in two of three pigs at around 3 hrs after dosing. One pig died after approximately 5 hrs after dosing. The two surviving pigs had near-zero MetHb levels 14 hrs after dosing. All pigs administered sodium nitrite showed brown cyanotic mucous membranes typical of MetHb poisoning, along with dark coloured blood and discolouration of organs and tissues that are well vascularized (tongue, gums).

Winks et al. (1950): Two pigs given a sublethal dose of sodium nitrite (0.04 g/kg) showed signs of methaemoglobinemia. Two hrs after dosing, the skin over the whole body was 'blanched' and pigs were docile and lethargic but ran normally when chased. The conjunctival vessels were a nut-brown colour. About five hrs after dosing, these symptoms had disappeared and the animals appeared normal. Individual pigs dosed with 0.05, 0.06, 0.07 or 0.08 g/kg (sublethal doses) developed signs of methaemoglobinemia that peaked three to four hrs after dosing. Blanching of the skin was the first visible symptom….signs of methaemoglobinemia were more severe with increased dose, with distinctly coffee-coloured blood visible in the conjunctival vessels. Respiration became rapid and shallow and the pigs were disinclined to exert themselves. Two pigs dosed with 0.09 g/kg developed severe methaemoglobinemia and died two hrs after treatment. There was no 'struggling' until about five min before death.

Toxic effects on humans:

Boink & Speijers (2001): Human symptoms include nausea, headaches and a lowering of blood pressure (hypotension) that can be fatal. Oxygen transport becomes impaired with high levels of methaemoglobin being formed.

References

Boink, A., and Speijers, G. (2001). Health effects of nitrates and nitrites, a review. *Acta Horticulturae* 563, 29–36.

Cowled, B.D., Elsworth, P. and Lapidge, S.J. (2008). Additional toxins for feral pig (Sus scrofa) control: identifying and testing Achilles' heels. *Wildlife Research*, 35, 651–662.

Nunn, J.F. (1994). Nunn's Applied Respiratory Physiology (4th ed). The University Press, Cambridge.

Winks, W. R., Sutherland, A. K., and Salisbury, R. M. (1950). Nitrite poisoning of pigs. *The Queensland Journal of Agricultural Science* 7, 1–14.

Table 1.8: Relative welfare impacts of VTAs affecting deer (*Cervus elaphus; C. nippon*;) and wallabies (*Macropus rufogriseus, Dama dama*) in New Zealand. There are currently no VTAs registered for deer control in New Zealand, however non-target poisoning of deer by 1080 occurs. Wallabies are currently not targeted for control using 1080, but can be poisoned as non-targets. Note that cyanide is undergoing development as a VTA for wallaby control in New Zealand, but is not currently available operationally for this species.

	Domain of potential welfare compromise											
Agent	1	2	3	4	5	C	Overall impact	C	Duration	C	Overall grade	C
1080*	No – Mild NDD	No – Mild NDD	Moderate – Severe *(Mod – Ex)*	Mild – Moderate *(No – Mod)* NDD	Moderate – Severe *(Mod – Ex)* Lethargy/weakness Sickness Pain Breathlessness	 2 1 0 0	Moderate – Severe *(Mod – Ex)*	1	Hours	1	5.5 *(5.5 – 7)*	1
Cyanide	See text											

* Evaluation for wallabies only, insufficient information to assess impacts for deer

1.5.2.22. Sodium fluoroacetate (1080) for deer and wallabies

Basis of welfare assessment and scoring (summary)

Deer in New Zealand are an acknowledged non-target species of 1080 poisoning operations aimed at possums and rodents. There are currently insufficient published data to assess potential welfare impacts of 1080 in deer species. Behavioural responses to poisoning must be interpreted carefully as cervids are notoriously non-expressive when in pain (Wilson & Stafford 2002). Indeed, the absence of activity in poisoned deer may, in itself, be indicative of pain and/or sickness. Judgments about negative affective experiences should be made on the basis of careful assessment of behaviour supported by physiological and pathological changes.

For wallabies, there are no direct data on which to base a welfare assessment for domains 1, 2 or 4. Based on the reported duration of symptoms (hours) for red-necked wallabies, only mild impacts are expected in these domains. Moderate to severe functional impairment relates to observations of incoordination, sickness behaviour and convulsions, along with post-mortem evidence of gradual cardiac failure. Whether animals are conscious during or after convulsions is unknown. Animals conscious during such events or those that recover consciousness afterwards may experience pain, breathlessness and anxiety/fear.

Conscious experience of lethargy/weakness and sickness are likely in wallabies. Based on the mode of toxic action and observations of other species, including humans, breathlessness (due to metabolic acidosis from accumulating citrate) and pain may also occur; however, there is currently insufficient information to assess these experiences in macropodids. Nor is there any published information upon which to evaluate the possibility of diminishing awareness which could reduce the duration of such negative affective experiences in wallabies.

Information required

For both wallabies and deer, comprehensive studies supporting assessment of welfare impacts of 1080 are required, particularly those providing more detailed information on the quality, intensity and duration of symptoms (i.e. time to onset, time from onset to loss of consciousness/death). Most importantly, information on level of consciousness at various times/events after dosing is required to determine the quality and duration of negative affective experiences. Information on time to loss of behavioural reflexes, normal EEG and evoked potentials after dosing is required. In addition, such measures should be used to evaluate level of consciousness immediately before and after spasms/tremors/convulsions. Studies involving administration of anti-nauseant and anti-anxiety and analgesic drugs would shed light on animals' experience of nausea, anxiety and pain. More specific information on respiratory compromise, particularly relative to level of consciousness, is required to assess potential experience of breathlessness. Direct information on feeding/drinking behaviour, potential for environmental exposure and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:
Refer to 1080 section above (under possums).

Mode of toxic action:

Refer to 1080 section above (under possums).

Toxic effects on deer:

Daniel (1966): '…the deer became lethargic and lay down quietly without any of the convulsions or leg-thrashing commonly reported in Canidae (e.g. dogs). The time lapse from ingestion of poisoned bait until death varied from about two hours in deer that had ingested 20–50 lethal doses…to about 30 hours in those that had consumed one lethal dose.'.

Toxic effects on wallabies:

(McIlroy 1982): For Bennett's wallaby (red-necked wallaby, Macropus rufogriseus), the range of latent periods was <16.9–23.2 hrs (from 7 wallabies observed) and the range of times to death was 8.9–38.9 hrs (from 23 wallabies observed). For dama wallaby (M. eugenii), time to death was 13.8–37.1 hrs. 'Most of the affected macropodids were observed simply lying quietly or found dead on their sides, without signs of convulsions having occurred. Affected Bennett's wallabies were sometimes observed sitting hunched up, with heads held shakily just above the ground. Generally they appeared non-alert and 'sick', with shivering or shaking forelimbs and unsteady balance. Most individuals then experienced convulsions, falling to the ground and lying on their backs and sides, kicking or making running motions with their hind legs before dying. Many individuals also ejaculated shortly before death and others exuded a white froth from their nostrils and mouth.' Post-mortem examinations indicated that both species died from gradual heart failure.

Elgie (1961): Time to death in Bennett's wallabies was estimated as 12–24 hrs.

Toxic effects on humans:

Refer to 1080 section above (under possums).

References

Daniel, M.J. (1966). Early trials with sodium monofluoroacetate (Compound 1080) for the control of introduced deer in New Zealand. Technical Paper No. 51, O.D.C. 414.11. Forest Research Institute, New Zealand Forest Service, Wellington, 27p.

Elgie, H.J. (1961). Wallaby eradication by aerial poisoning. *New Zealand Journal of Agriculture* 102(1), 26–31.

McIlroy, J.C. (1982). The sensitivity of Australian animals to 1080 poison III. Marsupial and eutherian herbivores. *Wildlife Research* 9, 487–503.

Wilson, P., and Stafford, K.J. (2002). Welfare of farmed deer in New Zealand. 2. Velvet antler removal. *New Zealand Veterinary Journal* 50, 221–227.

1.5.2.23. Cyanide for deer and wallabies

Basis of welfare assessment and scoring (summary)

Overall, there is insufficient published information to assess welfare impacts of cyanide poisoning on wallabies.

Information required

Comprehensive studies supporting assessment of welfare impacts of cyanide are required, particularly those providing detailed information on the quality, intensity and duration of symptoms (i.e. time to onset, time from onset to loss of consciousness/death). Importantly, information on level of consciousness at various times/events after dosing is required to determine the quality and duration of negative affective experiences. Specific information on respiratory compromise, particularly relative to level of consciousness, is required to assess potential experience of breathlessness. Direct information on feeding/drinking behaviour, potential for environmental exposure and alterations in social behaviour is required to assess impacts in domains 1, 2 and 4.

Review of literature

Evaluations of welfare impacts / humaneness:

Refer to Cyanide section above (under possums).

Mode of toxic action:

Refer to Cyanide section above (under possums).

Toxic effects on wallabies:

Eason et al. (2009): Pen trials with Bennett's and dama (tammar) wallabies have established that the encapsulated cyanide formulation Feratox® has a similarly rapid progression of poisoning as seen in possums, indicating similar humaneness. However, data regarding times to loss of consciousness and death in wallabies are not currently published.

Toxic effects on humans:

Refer to Cyanide section above (under possums).

References

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Table 1.9: Summary of overall grades (median of panelist scores) and associated confidence levels (C) assigned for all VTA/species combinations. ND = insufficient data available to assign an overall grade.

	Possums		Rodents		Carnivores		Rabbits		Pigs		Deer/wallabies	
	Grade	*C*	Grade	*C*	Grade	*C*	Grade	*C*	*Grade*	*C*	Grade	*C*
1080	6	*1*	6	*1*	6.5	*1*	6	*1*	6.5	*2*	5.5	*1*
Cyanide	4	*2*									ND	
Cholecalciferol	8	*2*										
Brodifacoum	8	*3*	7.5	*3*	7.5	*1*						
Pindone	7.5	*1*	ND				8	*1*				
Diphacinone			ND		ND							
Phosphorus	6	*1*					ND		7	*1*		
Zinc phosphide	6	*3*										
PAPP					5.5	*1*						
Sodium nitrite									5.5	*1*		

1.6. DISCUSSION

The objectives of this project were:

1. To collate relevant current literature from New Zealand, Australia, and elsewhere, and review it for its applicability and utility for assessing the animal welfare impacts on the agreed range of species and devices/tools, using the framework developed in Australia (Sharp and Saunders, 2008);

2. To apply the Australian framework to evaluate the relative animal welfare impacts of the nominated pest control tools used in New Zealand.

3. To identify current gaps in the information necessary to assess relative welfare impacts of pest control tools to guide future scientific research.

The pest control tools identified and agreed upon for this project were vertebrate toxic agents (VTAs) and kill traps currently used to control mammals, as well as three VTAs presently undergoing registration or development for use in New Zealand. Information forming the basis of the welfare assessment was primarily sourced from articles and reports published in the scientific literature, supplemented with some unpublished reports and occasional personal communications with pest control experts. For VTAs, assessments of welfare impacts were based on the mode of toxic action, specific effects on the nominated species/group, and reports from human victims.

As noted in the Introduction, overall grades of animal welfare impacts generally reflect the impact score assigned for domain 5, along with the duration for which those impacts were experienced by the animal. The reason for this is two-fold. Firstly, negative affective experiences relate, at least in part, to impacts in the other 4 domains, particularly to functional impairments and pathology in domain 3. Secondly, according to our current conception of animal welfare, negative affective experiences are most directly relevant to the welfare state of the animal (Mellor et al. 2009).

1.6.1 Relative welfare impacts of vertebrate toxic agents

Based on the considered opinions of a panel of scientists expert in pest control technology, physiology, toxicology, veterinary science, animal welfare science, and bioethical analyses, who had familiarized themselves with the relevant literature, and following the Australian framework, the following conclusions were drawn regarding the relative welfare impacts of lethal doses of VTAs used to control mammalian pests in New Zealand (Table 1.9).

Possums

Cyanide was judged to have the least overall impact on possum welfare (median score 4). Moderate breathlessness lasting for minutes was considered the primary negative experience, with potential for sickness, lethargy, headache and nausea. Our confidence in the grade was relatively high.

In contrast, cholecalciferol and the anticoagulant poisons (brodifacoum, pindone) were considered to have the highest overall impact on possum welfare. Severe or extreme impacts lasting days to weeks resulted in median scores of 7.5 to 8 for these agents. We consider that

possums poisoned with cholecalciferol experience breathlessness, pain, lethargy and sickness, and possibly nausea. Brodifacoum poisoning also results in lethargy, pain and sickness, and breathlessness may occur if haemorrhages occur in respiratory structures. While we had high confidence in the grades assigned for cholecalciferol and brodifacoum, there was insufficient evidence to reliably evaluate the impacts of pindone on possums. However, based on the identical mode of toxic action of anticoagulant VTAs in general, we considered the impacts to be comparable to brodifacoum. Further information on respiratory compromise associated with cholecalciferol poisoning in possums would be useful. For pindone, the time to onset of symptoms, and more detailed information on the quality and intensity of those symptoms in possums is required.

Intermediate impact grades (6) were assigned to 1080, phosphorus and zinc phosphide when applied to possums, representing severe impacts lasting for hours. For 1080, nausea, lethargy, pain and sickness are the most likely experiences, followed by breathlessness, dizziness and anxiety. We had relatively low confidence in the overall grading based on currently available information, mainly due to uncertainty about the duration of negative experiences. It has been suggested that 1080's neural effects may progressively diminish awareness, potentially reducing the duration of negative experiences. However, more information on the level of consciousness during and after key events (e.g. convulsions) is required to support this proposition.

Possums poisoned with phosphorus likely experience pain and nausea and possibly sickness, lethargy and anxiety for hours. We were moderately confident in this grading, however, further animal data on the character and intensity of negative impacts and the level of consciousness after animals become prostrate are needed for clarification.
Zinc phosphide poisoning is likely to result in severe nausea, pain, breathlessness, sickness and lethargy. Although the median score was intermediate (6), a wide range of scores was assigned by the panel (4–7), possibly reflecting our uncertainty about the negative impacts produced by phosphine, the corrosive metabolic product of zinc phosphide. Further information on the level of consciousness during and after convulsions and more detailed data on respiratory compromise are required to clarify potential impacts of zinc phosphide on possums.

Rodents

Rats lethally poisoned with 1080 were considered to experience severe pain, nausea, sickness, breathlessness and anxiety for hours (median grade 6). The experience of nausea and anxiety in rats is indirectly supported by experimental evidence. Nonetheless, our confidence in the overall grade was low and more detailed information regarding the intensity and duration of negative experiences is required.

In contrast, we were highly confident about the impacts of brodifacoum on rat welfare. Brodifacoum-poisoned rats likely experience severe to extreme pain for days, as well as other negative states, depending on the site of haemorrhage (median grade 7.5). There is currently no relevant information available to assess welfare impacts of brodifacoum for mice. Likewise, there is insufficient information to evaluate the impacts of the other anticoagulant poisons on rodent welfare. Based on the identical mode of toxic action and similarity in observed symptoms, the impacts of diphacinone and pindone are likely to be comparable to brodifacoum for rodents.

Carnivores

As for possums and rodents, lethal brodifacoum-poisoning had the highest welfare impacts of the VTAs assessed for carnivores (median grade 7.5). Ferrets and cats likely experience severe to extreme pain and lethargy for days to weeks, as well as breathlessness if haemorrhages involve respiratory structures. Our confidence in this grading was low, primarily due to uncertainty about the duration of negative experiences. More detailed information on the time to onset of symptoms is required. No information is available to assess impacts on stoats, and comprehensive studies are required to provide data on the quality, intensity and duration of welfare impacts for this species.

There is currently insufficient information to evaluate the impacts of diphacinone on ferret welfare. Based on the identical mode of toxic action and similarity in observed symptoms, the impacts are likely to be comparable to brodifacoum for rodents.

In accordance with results for other species/groups, carnivores appear to experience intermediate welfare impacts due to 1080-poisoning (median grade 6.5). Carnivores likely experience severe to extreme nausea, lethargy, sickness and breathlessness. However, due to uncertainty about the progressive effects of 1080 on level of consciousness, our confidence in this grading is low. More information on the level of consciousness during and after key events (e.g. convulsions) is required.

P-aminopropriophenone (PAPP) has recently been proposed as a 'more humane' vertebrate toxic agent for controlling cats and stoats in New Zealand. In accordance with this notion, the median overall grade (5) was lower than those assigned to VTAs currently used to control these species. We were relatively confident that PAPP-poisoned carnivores experience severe lethargy and nausea for minutes to hours. However, we had very low confidence in the overall grading and panel members assigned a wide range of overall scores for PAPP. This range reflects our uncertainty regarding experiences of breathlessness and anoxic headaches prior to reduction or loss of consciousness (cerebral anoxia). Future research should provide more detailed data on the intensity and onset of respiratory effects relative to level of consciousness. In addition, real-time correlations between physiological variables (e.g. % MetHb) and behavioural symptoms would aid our understanding of the duration of negative experiences.

Rabbits

Pindone was judged to have the highest impact on welfare (median score 8), with rabbits likely experiencing extreme pain for days. They may also experience lethargy, sickness and breathlessness, depending on the site of haemorrhage. As there are no data available on behavioural symptoms, the evaluation was based on reports of pathology and extrapolation from human poisoning reports. Consequently, our confidence in this grading was low. Comprehensive studies are required to provide data on the time to onset and quality of symptoms, particularly the respiratory effects, shown by rabbits poisoned with pindone.

Consistent with results for other species, 1080-poisoning was considered to have intermediate impacts on rabbit welfare (median grade 6). Based on the information available, rabbits are likely to experience severe lethargy for hours, as well as possibly breathlessness, pain and anxiety. Our confidence in this grading is low due to uncertainty regarding the intensity and duration of negative impacts. More information on the level of consciousness during and after

key events (e.g. convulsions) and the onset and intensity of respiratory compromise relative to any reduction in consciousness in rabbits is required.

There is currently insufficient information to evaluate the impacts of phosphorus on rabbit welfare. Comprehensive studies supporting assessment of welfare impacts of phosphorus on rabbits are required. In particular, detailed information on behavioural responses to poisoning, pathology/functional impairment, and time to loss of consciousness and death would allow assessment of the quality and duration of negative affective experiences.

Pigs

The overall impact grade for pigs poisoned with phosphorus was higher than for possums (median score of 7 vs 6). This higher grade primarily reflects the longer duration of symptoms for pigs (days vs. hours) but also the perceived greater severity of functional impairment (gastrointestinal and liver degeneration) and associated lethargy, nausea, sickness and pain (severe vs moderate–severe). Our confidence in this grading was low due to uncertainty about the intensity and duration of negative experiences. More detailed data on the onset and intensity of symptoms and the level of consciousness during and after key events (e.g. clonic convulsions) is required.

As for all other species currently examined, the overall impact grade for 1080 was intermediate (median 6.5). Based on the observed behaviour and pathology, pigs are likely to experience severe to extreme sickness nausea, lethargy, breathlessness and pain for hours to days after 1080 poisoning. We had moderate confidence in this grading. Future studies should provide information to clarify the onset and intensity of respiratory effects and level of consciousness during and after neural events (e.g. convulsions).

Sodium nitrite, like PAPP, is currently being considered for registration in New Zealand. Sodium nitrite has the same mode of toxic action as PAPP (reducing the effective oxygen-carrying capacity of the blood) and was assigned the same overall grade when applied to pigs (median grade 5). Pigs poisoned with sodium nitrite likely experience moderate to severe nausea for minutes to hours. They may also experience breathlessness, anoxic headache and lethargy. However, our confidence in the overall grading is low and the panel assigned a wide range of scores for this combination (3 – 7). This range reflects our uncertainty regarding experiences of breathlessness and anoxic headaches prior to reduction or loss of consciousness (cerebral anoxia). As for PAPP, future research should provide more detailed data on the intensity and onset of respiratory effects relative to level of consciousness. In addition, real-time correlations between physiological variables (e.g. % MetHb) and behavioural symptoms would aid our understanding of the duration of negative experiences.

Wallabies and deer

The overall impact grade for 1080 affecting various species of wallabies and deer as non-targets was lower than for the other species assessed (median 5.5 vs 6 – 6.5). These groups were judged to experience moderate to severe impacts for hours, primarily lethargy and perhaps sickness. However, there is very little information available for either group and herbivores are known to be relatively undemonstrative of pain and sickness. We had low confidence in this grading and comprehensive studies supporting assessment of welfare impacts of 1080 are required, particularly those providing more detailed information on the quality, intensity and duration of symptoms (i.e. time to onset, time from onset to loss of

consciousness/death). Judgments about negative affective experiences in these groups should be made on the basis of careful assessment of species-specific behaviour supported by physiological and pathological changes.

There is currently insufficient evidence available to evaluate the impacts of cyanide on wallaby welfare. Comprehensive studies supporting assessment of welfare impacts of cyanide are required, particularly those providing detailed information on the intensity and duration of respiratory effects.

For those agents used for multiple targets, and/or inadvertently consumed by several non-target species, the impact scores were relatively consistent across species/groups (Table 1.9). For example, 1080 was assigned intermediate impact scores (5.5 – 6.5) for all species considered. Similarly, the anticoagulant agents were assigned very high scores (7.5 – 8) for possums, rodents and carnivores. The other agents were used only for a single species/group (e.g. cholecalciferol and zinc phosphide for possums, PAPP for carnivores, sodium nitrite for pigs) or there was insufficient information available to assess welfare impacts, meaning that a comparison of the agent's impacts across species was not possible.

For most of the agents used to control mammalian pests in New Zealand, studies involving administration of analgesic, anti-nauseant and anti-anxiety drugs would improve our understanding of the animals' experiences of pain, nausea and anxiety. In most cases, direct information on feeding/drinking behaviour, potential for environmental exposure, and alterations in social behaviour is also required to more accurately assess impacts in domains 1, 2 and 4.

1.6.2 Applicability and modifications of Australian framework

Overall, the Australian framework was found to be highly applicable to the evaluation of relative welfare impacts of VTAs, with the minor modifications noted in the Methods section. We also suggest some amendments to the terminology of the original framework to better reflect the most current conception of animal welfare (Mellor et al. 2009). The modifications and suggested amendments to the Australian framework are as follows:

Score impacts in domain 3 only to point of loss of consciousness

Lethal doses of VTAs all have the same endpoint, the ultimate functional impairment: death. If each VTA was scored to this maximum level of impairment, the framework would provide no resolution between tools – all agents would receive a maximum score for part A (8). Therefore, to provide resolution between lethal methods, the panel scored impacts in domain 3 up to the point at which the animal lost consciousness.

Only assess VTAs using Part A of the Australian framework

All VTAs and traps assessed in the current report are designed to be lethal. The initial control tool and the method that causes death are one and the same. Therefore, completion of Part B simply produced repetitions of Part A scores (except with letters rather than numbers), providing no additional information on welfare impacts. For this reason the panel decided that Part A scores provided sufficient description of the relative welfare impacts of the control tools assessed.

Examples given for domains 4 and 5 have low applicability for VTAs

Most of the examples given for impacts in domain 4 describe degrees of physical restraint resulting in behavioural or interactive restriction. As physical restraint is not a feature of pest control by administration of VTAs, these examples have low applicability for our present evaluation. For VTA control, impacts in domain 4 are more likely to relate to functional impairment. For example, neural effects (e.g. neuromuscular dysfunction, spasms, convulsions) and pathological conditions resulting in feelings of sickness and lethargy may incapacitate animals so that they are unable to withdraw from conspecifics, escape/defend themselves from predators, or fulfill other behavioural 'needs' e.g. feeding, grooming, mating, caring for young.

The inability to express these normal behaviours may result in feelings (domain 5) of anxiety, fear or frustration. However, it is also possible that sickness, lethargy and altered consciousness affect motivation to perform normal behaviours and therefore feelings resulting from an inability to do so. For example, do animals experiencing nausea or sickness also experience hunger/thirst if they are unable to feed/drink or exhibit anorexia for prolonged periods? Our current understanding of such effects on motivation and affective states is poor.

As for domain 4, the examples given for domain 5 (Appendix p vii) have low applicability for assessment of VTAs because human contact and handling are not features of these control methods. Examples for domain 5 should reflect likely affective states relating to functional impairments (domain 3) and reports from human victims of the same agent.

Replace 'humaneness' with 'animal welfare impact' throughout

'Humaneness' is not defined in Sharp and Saunders' framework, likely because there is currently no objective definition of this term. The panel noted that it would be rare for any control tool to be 'humane', in other words, for it to cause no negative impact on the animal. In reality, most, if not all, control tools impact negatively on animal welfare to some degree, meaning that rankings would most accurately range from 'most to least inhumane' rather than any being humane per se. In addition, to many people, the concept of 'humaneness' also involves consideration of whether it is 'cruel' to kill pest animals in the first place. Such issues make assessment of the relative welfare impacts of various tools, necessary on conservation and economic bases, less practicable.

To minimize such semantic problems, the panel decided to use the term 'relative animal welfare impact' in assessing pest control tools used in New Zealand. This term implicitly acknowledges that some degree of negative impact is likely with all methods and provides a more realistic objective for assessment and for practical decision-making.

Clear differentiation of functional impairment (domain 3) and negative affective experience (domain 5)

In the original framework, there is some mixing of terms reflecting functional impairment, injury or pathology (domain 3) and terms reflecting negative affective experiences associated with those impairments (domain 5). In particular, in Box 3 of the Appendix (Assessment of humaneness worksheet), examples include the following terms: sickness, lethargy, breathlessness. We consider these terms to reflect the negative affective experiences, mental states, emotions or feelings associated with functional impairments/pathology, rather than the

impairments themselves. For domain 3, the functional impairment, injury or pathology itself should be described and the associated affective experience/feeling should be described and scored separately for domain 5.

For example, the feeling of sickness may relate to the actions of pro-inflammatory cytokines in the brain and periphery (Gregory 1998) and is extrapolated by the appearance of abnormal postures, appearance and behaviours and reduced activity. Likewise, the experience of breathlessness (breathing discomfort) relates to unsatisfied chemical drive to breathe (air hunger), increased work of breathing (work) or bronchoconstriction (tightness) (Lansing et al. 2009). Feelings of lethargy are probably associated with neuromuscular dysfunction as well as sickness pathology, and are manifested as impairments of movement (weakness, incoordination, reduced activity, prostration). Finally, nausea is the feeling most commonly associated with retching and vomiting events, although human reports indicate that nausea can also occur in the absence of such observable symptoms.

As we currently have no way to directly measure these subjective mental states (although functional brain imaging studies are rapidly expanding our understanding), we have based our scores for domain 5 on observable impacts in the other domains (particularly domain 3) and on the reports of affective experiences of human poisoning victims. For each affective state, we have indicated our confidence in the relevance and suitability of the data on which our extrapolation was based.

Expand range of negative affective states described in domain 5 and clarify meaning of 'distress'

In the Australian framework, domain 5 describes only anxiety, fear, pain, distress, sickness and greater than normal thirst/hunger. We propose adding at least the following negative affective experiences for the purpose of comprehensively assessing potential welfare impacts: breathlessness, nausea, lethargy/weakness, and dizziness. For a full list of possible negative affective experiences see Figure 1.1 and Mellor et al. (2009).

In addition, clarification of the term 'distress', as independent from other specific negative affective experiences, is required. As currently written, distress appears to be used as a 'catch-all' phrase to accommodate 'other negative experiences that aren't clearly identifiable'.
To more accurately assess the experiences of negative affective states, we require a better understanding of the level of consciousness or awareness of the animal during various events after poisoning. This is particularly important for VTAs with neural effects (e.g. 1080); if consciousness is progressively reduced after poisoning, the negative affective experiences associated with functional impairment/pathology may also be reduced. As noted above, more information about neural dysfunction and likely levels of consciousness during such events is required.

Clarification of impacts associated with CNS effects such as convulsions

The panel felt that minor clarification was required in the description of impacts of Central Nervous System dysfunction on welfare. In particular, in Box 3 of the Appendix (Assessment of humaneness worksheet, Domain 3), one example given under Moderate Impact is '*convulsions whilst unconscious'*. There can be no welfare impact while the animal is unconscious, and events occurring during unconsciousness are only relevant if and when consciousness is recovered. Therefore, we suggest that this statement should read

'*convulsions whilst unconscious if the animal subsequently recovers consciousness*'. Alternatively, the statement should read 'convulsions' to simply describe the manifestations of CNS dysfunction (domain 3). Negative affective experiences relating to conscious experience during or after such events should be scored in domain 5 e.g. pain, breathlessness, anxiety/fear. Similar comments apply for the examples listed under Severe Impact (*intermittent convulsions*) and Extreme Impact (*convulsions whilst conscious*).

As more information about neural dysfunction and likely levels of consciousness during such events becomes available (e.g. from studies including EEG, evoked potentials etc), neural impairments should be more specifically defined and described e.g. localized spasms, tremors, paralysis, tonic convulsions, tonic–clonic convulsions. Each may have different welfare impacts if animals are conscious during the event or recover consciousness afterwards.

Acknowledgment that duration of impact is implicit in determining intensity scores for domains 1, 2 and 4

The panel wishes to highlight the fact that scores for impacts in domains 1, 2 and 4 implicitly relate to the duration over which those impacts are experienced. Psycho-physiological drives such as hunger and thirst do not impact upon welfare until they become severe – the severity of hunger and thirst are inherently related to the duration of food/water deprivation. Likewise, the degree of environmental challenge depends, in part, on how long the animal is exposed to environmental variables. Therefore, duration influences the grade of overall welfare compromise twice. However, as impacts in domains 3 and 5 were generally found to be more intense than impacts in the other three domains, this problem likely had little bearing on the overall grades assigned.

For evaluations that have a mode of death different from initial impacts (Completion of Part B), replace 'suffering' with 'negative impact' in Box 7

In accordance with comments made above, the panel proposes to replace the word 'suffering' with 'negative impact' throughout Box 7 of the Assessment worksheet Part B (Appendix). Changes in terminology consistent with above comments regarding the range of experiences listed for domain 5 are also suggested for Box 7 (i.e. increased specificity in describing negative affective experiences). We also note that dyspnoea (breathlessness) is not included as an example until 'Extreme suffering'. We suggest that mild to severe breathlessness can also occur and impact animal welfare to lesser degrees (particularly with inhaled vapors/gases).

As discussed above, use of the framework to assess kill-traps for which NAWAC-testing data were available was not considered appropriate, due to the expected uniformity of scores for passed traps and the need to retain reference to the NAWAC guideline as the current defining welfare criterion for assessing kill-traps in particular.

1.7. CONCLUSIONS

For all species/groups exposed (possums, rodents, carnivores, rabbits), lethal anticoagulant poisoning has the highest relative impact on animal welfare. These gradings reflect the severe or extreme pain associated with wide-spread haemorrhaging and the prolonged duration of such experiences (days to weeks). In contrast, cyanide has the lowest relative impact for possums, with animals losing consciousness within minutes of dosing.

In general, sodium fluoroacetate (1080) and phosphorus produce intermediate impacts relative to the other VTAs assessed. Although it has been suggested that the neural effects of 1080 result in a progressive decline in the level of consciousness and therefore reduced durations of negative experiences, at least in possums, further research is required to support this supposition. In addition, whether animals are conscious during, or recover consciousness after, neural events such as localized spasms/tremors and convulsions is currently poorly understood. Animals conscious during such events or those that recover consciousness afterwards may experience pain, breathlessness and anxiety/fear, and more detailed information on these issues could significantly alter the relative impacts of 1080 on animal welfare.

For those VTAs currently unregistered for use in New Zealand, opinion on the panel was divided. For Zinc phosphide applied to possums, most panel members assigned relatively high impact scores (6–7), while one considered the impacts to be mild (4). This disparity may relate to different understandings of the effects of bait formulation. For PAPP (carnivores) and sodium nitrite (pigs), the range of grades reflected the panel's uncertainty as to the animals' experiences of breathlessness and anoxic headaches before cerebral anoxia caused reduction or loss of consciousness. Again, future research will help clarify such issues.

In general, there is insufficient information available to assess the impacts of VTAs on the welfare of herbivores such as wallabies and deer. Herbivores are known to be relatively undemonstrative of pain and sickness, and judgments about their experiences should be made on the basis of careful assessment of species-relevant behaviour, supported by physiological and pathological changes.

Overall, the Australian framework developed by Sharp and Saunders (2008) is highly applicable to the evaluation of New Zealand pest control tools. Several key modifications made the original framework better suited to assessment of the welfare impacts of vertebrate toxic agents. These include: scoring functional impairments (domain 3) only to the point of loss of consciousness to allow resolution of different lethal methods; scoring only Part A of the framework because initial impacts and mode of death are the same for VTAs; and for domains 4 and 5, providing more general examples, that apply to VTAs as well as other control methods.

In addition, the panel suggest several amendments to the terminology of the original framework to better reflect our current conception of animal welfare. These include: replacing 'humaneness' with 'animal welfare impact' as truly humane control methods are rare; clearly differentiating impacts in domain 3 (functional impairment/pathology) from impacts in domain 5 (negative affective states associated with impairment/pathology); the addition of at least breathlessness, nausea, lethargy, and dizziness to the list of possible negative affective experiences, and clarification of the term 'distress'; and clarification of impacts associated with CNS dysfunction and level of consciousness in domain 3.

1.8. RECOMMENDATIONS

Studies should be undertaken to specifically provide data to address the current lack of information available to conduct comprehensive analyses of animal welfare impacts. In particular, dataare needed on the time between onset of symptoms and loss of consciousness (duration of negative affective experiences) are lacking, as is information on the level of consciousness during various critical events after poisoning, e.g. spasms/tremors, convulsions, respiratory compromise.

For most agents used to control mammalian pests in New Zealand, studies involving administration of analgesic, anti-nausea and anti-anxiety drugs would improve our understanding of the animals' experiences of pain, nausea and anxiety after poisoning. In most cases, direct information on feeding/drinking behaviour, potential for environmental exposure, and alterations in social behaviour is also required to more accurately assess impacts in domains 1, 2 and 4.

The current report evaluated only effects of VTAs delivered using 'best practice', i.e. assuming each animal received the lethal dose delivered using the most effective bait formulation. In reality, a range of outcomes are possible, and the frequency with which pest animals are actually poisoned according to 'best practice' is unknown. Therefore, although beyond the scope of the current report, it is critical that the welfare impacts associated with sub-optimal or sublethal dosing also be comprehensively studied and evaluated.

In addition, there remain a range of VTAs (e.g. avicides, piscicides) and other New Zealand pest control methods (e.g. leg-hold and cage traps, shooting) that could, in future, be assessed for relative welfare impacts using the Australian framework. It would also be worthwhile to compare the scores derived and reported here by a New Zealand panel, with scores produced by Australian panels using the framework for the common combinations of VTAs/ species, e.g. 1080/rabbits/pigs.

The panel has suggested several amendments to the terminology of the original framework to better reflect our current conception of animal welfare and to improve its applicability to a wider range of pest control methods.

1.9. ACKNOWLEDGEMENTS

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1.11. APPENDIX – REFERENCES FOR TESTING KILL-TRAPS USED IN NEW ZEALAND AGAINST THE NAWAC GUIDELINE

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2. In-burrow rabbit control methods

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August 2010

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2.1. ABSTRACT

Project Code

- 11326: Original RFP title: How humane are our pest control tools?

- Title of Part 1 report prepared by Beausoleil et al. (2010): Assessment of animal welfare impacts of vertebrate toxic agents and kill traps used for controlling mammalian pests in New Zealand. This report comprises Part 2 and covers in-burrow rabbit control tools.

Business/Institution: Landcare Research, Pest Control Technologies team, Lincoln

Programme Leader: Penny Fisher

Goal

- Review literature and current information about New Zealand use of 'in burrow' control methods for rabbits, including chloropicrin, magnesium phosphide, cyanide and methods based on flammable gas mixtures.

- Using information from the literature review regarding mode of action and effects on rabbits, apply a framework for assessment of humaneness to evaluate the relative animal welfare impacts of in-burrow rabbit control methods used in New Zealand.

Context of the project

- Increasingly, information on the animal welfare impacts of pest control methods is required to inform the decisions of pest management operators and policymakers. To date, there has been no cohesive evaluation of welfare impacts associated with pest control methods used in New Zealand. The purpose of this project was to apply a recently developed Australian model to produce a ranking of the relative welfare impacts of 'in-burrow' rabbit control methods used in New Zealand.

Approach

- The 'in burrow' rabbit control methods nominated for review by MAFBNZ were chloropicrin, magnesium phosphide, cyanide, and methods based on flammable gas mixtures. Registration in New Zealand of products used for rabbit control based on these methods was reviewed, and the availability and operational use of these methods assessed by phone surveys and Internet searches. Literature for each of the active ingredients in rabbit fumigant products was reviewed to summarise mode of toxic action and reported effects in rabbits (the target pest species), other mammals, and humans. Information forming the basis of the welfare assessment was primarily sourced from articles and reports published in the scientific literature, supplemented with some unpublished reports and communication with pest control experts.

- This information was used by the authors to assess the welfare impacts of each in-burrow rabbit control method using both Parts A and B of the Australian model (Sharp & Saunders 2008), with some modifications as described by Beausoleil et al. (2010). Assessment assumed 'best practice' application of each control tool in killing rabbits (where this was defined), so welfare impacts associated with sublethal effects were not assessed.

Outcomes

- The in-burrow rabbit control methods currently registered and available for use in New Zealand are chloropicrin and magnesium phosphide (fumigants) and 'The Rodenator', a device designed to create an explosion in rabbit burrows through the ignition of flammable gas. Of these, magnesium phosphide appears to be recommended as 'best practice' and most commonly used. The extent of chloropicrin and 'Rodenator' use for rabbit control in New Zealand was difficult to determine.

- The overall score assigned to choloropicrin using the model was 5F, based on a combination of moderate to severe negative welfare impacts (inhalation of toxic vapour causing extreme irritation and respiratory distress in rabbits) over a duration of minutes. The availability of recently published, formal evaluations of the humaneness of chloropicrin relative to other rabbit fumigants gave a high certainty to this assessment.

- The overall score assigned to phosphine (the gas generated by magnesium phosphide) using the model was 3D, based on a combination of mild to moderate negative welfare impacts (moderate respiratory irritation in rabbits) over a duration of minutes. The availability of recently published, formal evaluations of the humaneness of phosphine relative to other rabbit fumigants gave a high certainty to this assessment.

- The overall score assigned to the 'Rodenator' using the model was 5E, based on a combination of welfare impacts ranging from none to extreme, with potentially extreme negative welfare impacts over a duration of immediate to seconds (consistent with close proximity of rabbits to a large explosion and very high temperatures). However, there is high uncertainty in this assessment, due to the lack of published scientific information about the range of effects on rabbits of this control method.

Recommendations

- A formal survey should be carried out to determine the extent of use of chloropicrin and the 'Rodenator' for in-burrow rabbit control in New Zealand. This would assist in estimating the number of rabbits potentially affected by these in-burrow control methods.

- MAFBNZ should commission a study of the welfare impacts of the 'Rodenator' (or similar systems) on rabbits to increase the certainty of the welfare impact score assigned.

- MAFBNZ should note the development and pending registration of a carbon monoxide pressure fumigator system in Australia (Invasive Animals CRC) as a potentially useful future addition to rabbit control tools used in New Zealand. Literature indicates that the use of carbon monoxide as a rabbit burrow fumigant is relatively more humane than either chloropicrin or phosphine-generating fumigant formulations.

Summary

- Current best practice for in-burrow rabbit control in New Zealand appears to be the use of magnesium phosphide as a fumigant. This method, of the three registered for this purpose in New Zealand, is indicated by the model as having the relative least negative welfare impact.

2.2. INTRODUCTION

The welfare of wild animals subject to human management as pests is receiving increasing attention in New Zealand and overseas. The growing body of literature on the welfare impacts of tools used to control mammal pests in New Zealand provides a gradually improving basis for gauging the acceptability of different methods on the basis of animal welfare. However, gaps remain in the available information regarding the welfare impacts of vertebrate toxic agents (VTAs) and in particular for the subset of toxic agents that are used as gases for fumigation of rabbit warrens (rather than toxic agents delivered in bait). This lack of information means that it is currently difficult to determine policy directions for the use of VTAs, or for a pest control operator to choose an optimal tool on the basis of overall animal welfare impact. To date, there has been no cohesive evaluation of the animal welfare impacts associated with rabbit control tools used in New Zealand. This is partly explained by the absence of a suitable framework for this type of evaluation. Recently such a framework has been developed in Australia (Sharp & Saunders 2008), and can be used to produce relative impact scores, and therefore rankings, for various lethal and non-lethal pest animal control methods.

This report specifically covers toxic agents used for 'in-burrow' rabbit control methods (e.g. fumigation). The general approach used in an earlier evaluation of the relative welfare impacts of bait-delivered VTAs used in New Zealand (Beausoleil et al. 2010) has been applied here. This included collation of relevant data from scientific literature and pest control guidelines to provide reference material for assigning rankings of welfare impact, using the framework developed by Sharp and Saunders (2008). However, rather than the panel assessment undertaken for the bait-delivered VTAs for a range of target and non-target mammal species, rankings for the in-burrow control methods for rabbits only (as the target pest species) were derived using the expert opinion of the authors and reviewers (Bruce Warburton and Phil Cowan, Landcare Research).

2.3. BACKGROUND

Rabbits (*Oryctolagus cuniculus*) can pose a significant, localised threat to New Zealand production and conservation values, e.g. through competition with grazing livestock, as a prey base supporting feral cat and ferret populations, and modifying vegetation cover and composition and causing significant soil damage and soil erosion (Lough 2009). New Zealand has a long history of rabbit management, particularly using lethal control tools such as poison baiting, shooting, hunting with dogs or ferrets, and warren ripping or fumigation. Removal of rabbit control subsidies in the 1980s placed particular economic pressure on farmers of rabbit-prone land to meet the ongoing costs of rabbit control (e.g. Parkes et al. 2002). The illegal introduction of rabbit haemorrhagic disease (RHD) virus to New Zealand in 1997 provided relief in dramatically reducing rabbit populations, such that conventional rabbit control came to an end on many properties (Lough 2009). The waning efficacy of RHD has prompted a need for increased reliance on rabbit control methods such as baiting with sodium fluoroacetate (1080) or pindone, shooting, and burrow fumigation.

Rabbit burrow fumigation is considered labour-intensive but has an advantage over toxic baiting because it can be used in the breeding season when young, because of their small range, may not encounter or feed on baits (NPCA 2008). Fumigation is suitable for areas where ground-laid poison may not be appropriate for rabbit control, such as close to dwellings or in stocked pasture. Fumigation is not suitable when burrows cannot be sealed or in hilly terrain where sufficient gas diffusion cannot be achieved. While burrow fumigation is

considered a secondary rabbit control measure (Lough 2009), the registration of fumigant formulations and the extent of their operational use across New Zealand appear not to have been summarised recently. A brief review was undertaken to describe the products available and most frequently used in operational rabbit control.

2.4. OBJECTIVES

- Review literature and current information about New Zealand use of 'in burrow' control methods for rabbits, including chloropicrin, magnesium phosphide, cyanide and methods based on flammable gas mixtures.

- Using information from the literature review regarding mode of action and effects on rabbits, apply a framework for assessment of humaneness to evaluate the relative animal welfare impacts of in-burrow rabbit control methods used in New Zealand.

2.5. METHODS

The Register of Agricultural Compounds (ACVM 2010) was consulted to identify fumigant formulations registered in New Zealand and their active agents. In some cases, the registration information indicated a fumigant product for application to soil, but not whether rabbits were listed as a target pest species on the label (see Table 2.1). To confirm which registered fumigant formulations were used for rabbit control, and to investigate the extent of their use in New Zealand, a telephone survey of product registrants and/or agents was made. Internet searches were also used to identify other in-burrow control methods and seek further information such as label instructions and best practice summaries of the nature and extent of fumigant use for rabbit control.

A review of scientific literature was undertaken, with a focus on each of the fumigants used for rabbit control in New Zealand. In particular, specific information was sought about:

- Welfare impacts or humaneness either by review or research on captive animals

- The mode of toxic action or general toxic effects on mammals in general, including information on behavioural, physiological, and pathological responses

- Description of effects, time to death, pathology or other information relevant to evaluating welfare effects

- The toxic effects of a fumigant on humans

Suitable articles were used as reference material to undertake the scoring process and assess relative animal welfare impacts of each in-burrow rabbit control method. This followed the Sharp & Saunders (2008) model, as applied to lethal control methods in an assessment of the killing technique. In applying the Australian model, scores were based on the assumption of 'best practice' in operational use of the control tools; i.e. it was assumed that every rabbit would be killed as the result of application. Consequently sublethal effects were not considered in allocating scores. Welfare impacts in terms of functional impairment/pathology were scored up to the point at which rabbits lost consciousness (where such information was available).

As described by Beausoleil et al. (2010), current methods for assessing the impacts of events, situations or procedures on animal welfare are based on the 'Five Domains of Potential Welfare Compromise' model developed by Mellor and Reid (1994). This model provides a means of clearly separating physical or functional impacts of the animal from the emotional or affective experiences, mental states or feelings that ultimately determine its welfare or well-being. In applying 'Part A' of the model, functional impairment/injury/pathology was scored for domain 3, as separate from the associated affective experience/feeling (e.g. breathlessness, nausea, lethargy/weakness or dizziness) that was scored for domain 5. Reports of affective experiences of human poisoning victims were also considered in extrapolation for scoring the subjective mental states for domain 5.

Both Parts 'A' and 'B' of the model were applied to each rabbit control tool, with corresponding numerical scores for overall welfare impact (Part A) and letter scores reflecting the degree of negative welfare impact of the killing method (Part B).

2.6. RESULTS

2.6.1 Registered rabbit burrow fumigants and other in-burrow control tools in New Zealand

Internationally, fumigation is used to control a range of pest animal species that inhabit burrows, using formulations and devices that generate and disseminate toxic gases, including carbon monoxide, phosphine, hydrogen cyanide, carbon disulfide and methyl bromide (Marks 2009). In New Zealand, rabbit burrow fumigation is carried out only with formulations that generate either phosphine or chloropicrin. A controlled substances licence is required for the use of magnesium phosphide and chloropicrin formulations registered as rabbit fumigants (Table 2.1).

Fumigation of rabbit burrows was carried out in the past with formulations that emit hydrogen cyanide, specifically a product known as 'Cyanogas'. While this product is no longer commercially available in New Zealand, some people may still have supplies although its use is likely to be limited (Anon. 2003). There is currently only one product (Cyanosil) listed on the ACVM register utilising hydrogen cyanide for fumigation purposes for ships holds, containers, warehouses and food factories (ACVM 2010). Instructions for use show no reference to burrow fumigation and it is assumed that it is not labelled for that purpose.

Other in-burrow rabbit control methods use ignition/combustion of flammable gas dispersed within the burrow to physically destroy the burrow structure and/or kill rabbits within. Such methods do not have an active 'toxic agent' and so do not require ACVM registration. Such devices are marketed at least in the United States and Australia. One such product is available in New Zealand, the 'Rodenator R3', distributed by Pestgard, Motueka.

Table 2.1 Burrow fumigants used for rabbit control in New Zealand (from ACVM 2010)

Registration #	Trade name	Registration date	Registrant
P005321	Pic-Fume Chloropicrin	20 Aug. 1998	Leicester's New Zealand Ltd
P001035	Soil Fume CP	10 Aug. 1965	Agricultural Fumigations Ltd
P003380	Magtoxin	25 Feb. 1987	Pharmochem Company

Carbon monoxide (CO) has been identified as a potentially more humane rabbit burrow fumigant than phosphine or chloropicrin (Gigliotti et al. 2009). There are no currently registered CO-generating systems or formulations for in-burrow rabbit control in New Zealand. However, such formulations are registered elsewhere for in-burrow vertebrate pest control, e.g. 'Den-Co-Fume' cartridges for control of red foxes in natal dens in Australia (Animal Control Technologies, Victoria). A pressure fumigator device for delivering CO to rabbit burrows is currently undergoing development and testing towards registration for field use in Australia (http://www.invasiveanimals.com/research/goals/goal-7/10u14c).

2.6.2 Chloropicrin

The ACVM list of registered products (ACVM 2010) currently includes seven formulations containing chloropicrin. Of these, 'Pic-Fume Chloropicrin' and 'Soil Fume CP' are listed for use as a vertebrate toxic agent in addition to use as a fungicide, nematicide, or herbicide. When used for the purposes of rabbit fumigation, chloropicrin is injected into a rabbit burrow and the hole sealed (label instructions for use recommend a 5-mL 'shot' from the windward side). This method relies on natural diffusion of the gas throughout the burrow and appears to have been the only application method used in New Zealand. Current best practice guidelines for rabbit control in New Zealand (NPCA 2008) do not mention the use of chloropicrin as a fumigant.

Diffusion fumigation was commonly practised in Australia before studies recommended application on absorbent materials to decrease wastage and increase gas concentrations (Oliver & Blackshaw 1979, cited in Marks 2009). Chloropicrin may also be introduced into the burrow by power fumigation, forcing the gas in and more efficiently attaining lethal concentrations (Marks 2009). This 'forced diffusion' technique appears not to have been used in New Zealand.

Worldwide, chloropicrin is almost exclusively used to treat soil, for example to control soil borne fungi, diseases and nematodes through fumigation before planting (Raman 2005). This also appears to be the case in New Zealand, where the use of chloropicrin formulations for the labelled purpose of rabbit control has declined and have not been in wide use for a number of years (Appendix 2.1). The main reasons for declining use for rabbit control appears to be the hazard posed to operators by liquid chloropicrin formulations in comparison to the more easily used tablet formulation of phosphine-generating alternatives.
Estimates of the quantities of chloropicrin sold and used in New Zealand could not be obtained through a telephone survey (Appendix 2.1). However, condition 53 pursuant to the ACVM Act 1997 requires that a register of sales be kept for a minimum of three years, detailing both the amount and to whom the product was sold; however, this information was not available to the authors. Some past users of chloropicrin may retain unused 'stockpiles' of the substance and continue to use it for rabbit control.

Description and mode of toxic action

Chloropicrin is a clear, colourless, non-flammable oily liquid that vaporises slowly at room temperature. It is a potent sensory irritant, causing tear production(lachrymation) and intense irritation of the respiratory tract. It was widely used in World War I as an agent of chemical warfare (Williams et al. 1995). Chloropicrin is highly toxic by ingestion or by direct contact with skin or eyes, but inhalation is the most significant exposure pathway. Animal studies have established a 4-h inhalation LC50 value in rats of 11.9 ppm (Extoxnet 1999).

Metabolism of chloropicrin in vivo is thought to generate metabolites that react with cellular substrates. This occurs via conversion of chloropicrin to thiophosgene, characterised as the cyclic cysteine adduct. This is then thought to react with glutathione and other thiol proteins. Oxidation of protein thiols by chloropicrin itself suggests possible inhibition of enzymes containing a critical thiol component, with this being involved in acute mammalian toxicity. The interference of chloropicrin with oxygen transport is also related to its reaction with protein thiol groups, such as haemoglobin (Raman 2005).

Target organs for chloropicrin toxicity include eyes, skin, respiratory tract and tissues associated with the likely routes of exposure. Toxicity is primarily due to effects on the small and medium bronchi, with death resulting from pulmonary oedema, bronchopneumonia and bronciolitis obliterans (Clayton & Clayton 1981, cited in Gigliotti et al. 2009).

Toxic effects in rabbits as a fumigant

Sensory irritation occurs chiefly by stimulation of the terminal nerves in the nasal mucosa. Animals that are obligate nose breathers (which include rabbits) are likely to be more prone to severe sensory irritation given their inability to divert air through the mouth (Buckley et al. 1984, cited in Williams et al. 1995).

In rabbits exposed to inhaled chloropicrin concentrations of 14–75 ppm, the time to death was estimated at 15–135 min for 50 percent of the population. For rabbits exposed to higher concentrations of 144 ppm, 50 percent of the population was expected to perish within 5 min (Gleeson & Maguire 1957, cited in Marks 2009). Fumigation trials in burrows where 5 mL chloropicrin was applied at the burrow entrance showed that variable concentrations occurred throughout the burrow. Higher concentrations were present at the point of introduction and at low points in the burrow (chloropicrin is denser than air). The rate of diffusion was slower than that of phosphine and incomplete mixing or diffusion resulted in low or undetectable chloropicrin concentrations at the terminal ends of a burrow for up to 4 hours (Oliver & Blackshaw 1979, cited in Marks 2009).

In a trial investigating the relative efficacy of rabbit fumigants (Gigliotti et al. 2009), chloropicrin was introduced into a natural warren by power fumigation (concentration range 11.7–99.7 ppm). A chamber was integrated into the warren, and a rabbit confined in the chamber was remotely observed by video camera. Progression of the signs of poisoning were:

- Immediate irritation upon exposure to chloropicrin characterised by rapid blinking and nose twitching
- Periodic bursts of distress vocalisation (squealing), which became less frequent as breathing became laboured
- Profuse lachrymation and nasal discharge
- Laboured breathing associated with ‘gurgling’ until collapse/prostration
- Uncoordinated paddling of limbs following collapse, and death after 70–95 min

Overall, rabbits exposed to chloropicrin displayed signs of intense irritation and extreme distress during a lethal toxicosis lasting a mean of 82.5 min (Gigliotti et al. 2009). Gross pathology following chloropicrin toxicity is characterised by pulmonary oedema, bronchopneumonia and bronciolitis obliterans (Clayton & Clayton 1981, cited in Gigliotti et al. 2009). Rabbits that escape a warren following sublethal chloropicrin exposure may die several weeks later on account of the debilitating effects of the exposure to lung and bronchial tissue (Gigliotti et al. 2009).

Toxic effects in humans

Studies of chronic human exposures have demonstrated that concentrations of less than 1 ppm chloropicrin can elicit severe irritation and destructive changes to lung tissue (TeSlaa et al. 1986, cited in Williams et al. 1995). Decreased respiration rate in mice has been used as an indicator of the extent of respiratory irritation caused by chloropicrin, with the airborne concentration that produces a 50 percent decrease in respiratory rate (RD50) being identified (Kane et al. 1981, cited in Williams et al. 1995). This was used as a benchmark for concentrations representing 'intolerable irritation' arising from repeated or chronic exposures in humans. The RD50 in mice exposed to chloropicrin was found to be 7.98 ppm, with exposure at this level for 6 h a day over 5 days resulting in ulceration and necrosis in the lungs and olfactory epithelium (Buckley et al. 1984, cited in Williams et al. 1999).

Inhalation exposure of humans to relatively low concentrations (0.3–0.37 ppm) for 3–30 s results in eye irritation, with lachrymation occurring in some individuals below this threshold (0.15–0.3 ppm). Exposure to moderate concentrations (4 ppm) for a few seconds may cause some degree of incapacitation, while exposure to higher concentrations can result in respiratory damage. Beyond 8 ppm, exposures are reported to be painful and incapacitating in humans (Marks 2009). Inhalation of concentrations exceeding 15 ppm can induce severe vomiting, and if exposure is sufficient may result in pulmonary oedema and death (Extoxnet 1999). In the USA, 165 people living in the vicinity of a farm were exposed to drifting chloropicrin vapour (O'Mallery et al. 2004, cited in Marks 2009). The 1-h average concentration in that case was estimated at 0.2 ppm with peak concentrations exceeding 1 ppm.

The following signs and symptoms were recorded:

- Eye and upper respiratory tract symptoms recorded in nearly all (99 percent) victims with these persisting for up to 11 days in seven people
- Lachrymation (82 percent)
- Gastrointestinal upset (47 percent)
- Vomiting (22 percent of victims)
- Nausea, cough and headache

Welfare impact score of chloropicrin for rabbits

Recent Australian research (Gigliotti et al. 2009) and a review (Marks 2009) suggested that chloropicrin is not considered a humane control tool for rabbits. The overall score we assigned using the model was 4F (Table 2.2), based on a combination of moderate to severe domain impacts and extreme negative welfare impacts over a duration of minutes.

Table 2.2: Categories and overall scores assigned for chloropicrin fumigation for rabbit control, using the model developed by Sharp and Saunders (2008)

Impact categories from model	Category assigned	Basis
Part A Domain 1. Water deprivation, food deprivation, malnutrition	No impact	Duration of adverse effects and times to death (up to 135 min) not sufficient to result in water or food deprivation
Part A Domain 2. Environmental challenge (e.g. heat/cold)	Mild impact	Potential for slight temperature increase in warren due to presence of chloropicrin vapour, and/or slightly decreased ability of rabbits to thermoregulate through effects of chloropicrin on respiratory rate
Part A Domain 3. Injury, disease, functional impairment	Severe impact	Gross pathology pulmonary oedema, bronchopneumonia and bronchiolitis obliterans Assumed that these would compromise survival in sublethally exposed rabbits.
Part A Domain 4. Behavioural, interactive restriction	Mild impact	Attempted flight on exposure to chloropicrin, warren entrances blocked in best practice applications. Effects on respiration likely to result in short-term reductions in normal movement or interactions
Part A Domain 5. Anxiety, pain, fear, distress	Severe impact	Distress vocalisation (squealing) reported in rabbits. Human reports of distress and pain associated with altered respiratory function and mucosal irritation
Overall impact Part A	Severe	Numerical score Part A: 5
Duration	Minutes	
Part B mode of death	Extreme suffering	Inhaled vapour causing extreme irritation and respiratory distress
Duration	Minutes	
		Letter score Part B: F
Overall score Parts A&B	5F	

2.6.3 Magnesium phosphide

The ACVM list of registered products (ACVM 2010) includes one formulation containing magnesium phosphide ('Magtoxin'). In use according to label instructions, the formulation produces phosphine gas through a reaction of water with magnesium phosphide. Magtoxin is supplied as solid tablets/pellets each weighing 0.6 g and composed of 66 percent magnesium phosphide in addition to ammonium carbamate and inert ingredients. This dry formulation counteracts the risk of phosphine gas being generated before application and potentially igniting, with the ammonium carbamate reacting to release ammonia and carbon dioxide upon phosphine generation. An application rate of 2 g/m3 (10 pellets/m3) is recommended for rabbit warrens, liberating 330 mg/g phosphine (Magtoxin instruction sheet).

Magtoxin works as a rabbit warren fumigant through passive diffusion of phosphine from wetted tablets. After administration of phosphine-generating tablets it may take many hours to achieve maximal (toxic) concentrations within rabbit warrens. The time taken is variable and governed to a large extent by moisture availability (Oliver & Blackshaw 1979, cited in Williams et al. 1995). Best practice guidelines for rabbit control in New Zealand (NPCA 2008) describe the use of Magtoxin: 'hydrogen phosphide is heavier than air and has a distinctive warning odour, pungent and unpleasant even at low concentrations. Rabbits that are exposed to the gas and escape may die up to a week later. It works well in soil temperatures above 15ºC, with over 10 percent soil moisture, the more moisture the faster the gas liberation'. Magtoxin appears to be the most-used rabbit fumigant in New Zealand, with an estimated minimum of 300 kg sold in 2009–10 (see Appendix 2.1).

Phosphine (hydrogen phosphide) is used internationally as both a vertebrate toxic agent and insecticide (Villalobos 2005). It can also be generated from aluminium phosphide, and much of the available toxicity data are based on aluminium phosphide formulations (Extoxnet 1996a). Magnesium phosphide can be considered to be synonymous with aluminium phosphide as they both produce phosphine gas, and both are regulated as such by the United States Environmental Protection Agency (US EPA 1998).

Description and mode of toxic action

Phosphine is a colourless gas with an odour of garlic or decaying fish. It is extremely flammable and may ignite spontaneously upon contact with air. Phosphine is an in vivo inhibitor of oxidative phosphorylation, via inhibition of cytochrome oxidase producing chemical asphyxiation of cells in a similar manner to cyanide, depressing central nervous system and respiratory function.

Exposure to phosphine gas produces little effect on the skin or eyes. It is, however, acutely toxic via inhalation exposure. The rodent 4-h inhalation LC50 for phosphine is 10.7 ppm (Extoxnet 1996). Inhalation experiments have been conducted examining phosphine concentration versus time relationship for mortality in a number of species including cats, guinea pigs and rabbits (Klimmer 1969, cited in US EPA 2003). Inhalation exposure of rats to concentrations of 25 ppm for a period of 8 h resulted in death, as did exposures to 406 ppm for 0.6 h. The phosphine concentration versus time relationship was shown to closely predict mortality in a range of mammal species.

Toxic effects on rabbits as a fumigant

In a trial investigating the relative efficacy of rabbit fumigants (Gigliotti et al. 2009), phosphine was introduced into a natural warren by use of an aluminium phosphide tablet formulation registered in Australia (assumed to have a similar action to the New Zealand Magtoxin formulation, in that phosphine gas is released following exposure of the tablet to water). An observation chamber was integrated into the warren, to enable rabbit behaviour to be observed via a video camera. There were no initial signs of distress seen in rabbits exposed to phosphine as gas concentrations built to peak levels. The first symptom of toxicosis was increased activity, e.g. sudden and agitated movements, seen at a mean of 235.3 min (SD = 106.8 min) after first exposure to phosphine. The mean time between first symptoms and death was 29.4 min (SD = 55.5 min) and there was no apparent laboured breathing, lachrymation, or distress vocalisation. Collapse was sudden and associated with convulsions, paddling and agonal gasping, although it was not possible to confirm whether the rabbit was conscious at this time and before death (Gigliotti et al. 2009). Phosphine gas killed 10 of 12 rabbits, in a mean of 225.3 min. The <100 percent mortality in rabbits was attributed to the low rate of gas production and passive diffusion through the warren, reflected in highly variable warren concentrations (Gigliotti et al. 2009).

Phosphine concentrations of 400 ppm were observed to be lethal in rabbits within 30 min, with test animals often remaining immobile during lethal exposures, suggesting exposure to phosphine during burrow fumigation may not produce severe sensory irritation (Oliver & Blackshaw 1979, cited in Williams et al. 1995). Villalobos (2005) describes inhalation exposure of rabbits to high (assumed lethal) phosphine concentrations as characterised by dyspnoea, paralysis, convulsions, hepatotoxicity and renal toxicity. However, chronic

exposures to low levels of phosphine did not result in subacute or chronic poisoning (Klimmer 1969, cited in Williams et al. 1995).

Toxic effects on humans

Human perception of phosphine ranges from a carbide or garlic-like odour, to an absence of perceived odour without sensory irritation (Sexton 1983, cited in Williams et al. 1995). Phosphine is a respiratory irritant, however, and acute short-term inhalation exposures of humans have been followed by headaches, dizziness, fatigue/drowsiness, burning substernal pain, nausea, vomiting, coughing/laboured breathing, chest tightness, pulmonary irritation/oedema and tremors. Data from a cohort of occupationally exposed Indian agricultural fumigation workers undergoing single exposures of approximately 0.71–2.22 ppm phosphine revealed reversible (within 2 weeks) symptoms of mild acute exposure (Extoxnet 1996). The following observations were reported in an examination of 22 workers subsequent to fumigation with aluminium phosphide (mean concentration 0.78–0.98 ppm) (US EPA 2003):

- Respiratory symptoms including tightness, suffocation and breathing difficulty lasting from 15 min to 3 h
- Neurological and gastrointestinal effects
- Numbness and paraesthesia (pins and needles) in the fingers of those who touched the fumigation tablets

Severe cases of phosphine poisoning can progress to convulsion, cyanosis and coma (Villalobos 2005). Inhalation exposure to concentrations exceeding 2000 ppm is lethal to humans, with death occurring in less than a minute (Sexton 1983, cited in Williams et al. 1995).

Welfare impact score of phosphine for rabbits

The overall score we assigned using the model was 3D (Table 2.3), based on a combination of mild domain impacts and moderate negative welfare impacts over a duration of minutes.

Table 2.3: Categories and overall scores assigned for phosphine fumigation for rabbit control, using the model developed by Sharp and Saunders (2008)

Impact categories from model	Category assigned	Basis
Part A Domain 1. Water deprivation, food deprivation, malnutrition	No impact	Duration of adverse effects and times to death not sufficiently long to cause significant water or food deprivation
Part A Domain 2. Environmental challenge (e.g. heat/cold)	Mild impact	Potential for slight temperature increase in warren due to presence of phosphine vapour, confinement of rabbits in warrens with sealed entrances and/or slightly decreased ability of rabbits to thermoregulate through effects of phosphine on respiratory rate
Part A Domain 3. Injury, disease, functional impairment	Moderate impact	Defined functional respiratory impairment for mammals but assumed moderate from reference to apparent full recovery from sublethal exposure in humans and rabbits
Part A Domain 4. Behavioural, interactive restriction	Mild impact	Warren entrances blocked in best-practice applications so restrict movements. Effects on respiration likely to result in short-term reductions in normal movement or interactions
Part A Domain 5. Anxiety, pain, fear, distress	Mild impact	No initial signs of distress seen in rabbits, first behavioural change during toxicosis was increased activity seen at a mean of 235.3 min (SD = 106.8 min) after first exposure
Overall impact Part A	Mild	Numerical score Part A: 3
Duration	Minutes	
Part B mode of death	Moderate	Mean time to death in rabbits 225 min (i.e. > 2 h) assumed to be conscious until soon before death; however, the mean time between first symptoms and death was 29.4 min (SD = 55.5 min). Moderate respiratory irritation considered likely based on other mammalian studies
Duration	Minutes	
		Letter score Part B: D
Overall score Parts A&B	3D	

2.6.4 The ‘Rodenator’

The Rodenator (Fig. 2.1) is a mechanical device for in-burrow rabbit control. The device is used to inject a calibrated mixture of liquefied propane gas (LPG) and compressed oxygen (O2) into burrows. The proportion of gases is approximately 2 percent LPG and 98 percent compressed O2, with injection of the gases being monitored over time to ensure the amount is within a specified protocol (Anon. 2009). Injection times can range from 2 s to 3 min depending on the type and size of the burrow.

Figure 2.1 Components of the ‘Rodenator R3’ (Rodenator 2010).

Once this flammable/explosive gaseous mixture is adequately distributed through the burrow, an electronic ignition module is used to initiate a high voltage spark, detonating the mixture (Anon. 2009). This is claimed to create a concussive force that travels rapidly through the burrow system (Fig. 2.2). Combustion of LPG and O2 produces carbon dioxide and water as by-products, so there are no resulting toxic residues.

The Rodenator is manufactured by Meyer Industries (Emmett, Idaho, USA) and distributed in New Zealand by Pestgard, Motueka. The variant marketed in New Zealand is the R3, which differs from earlier versions in that it allows detonation at a distance of up to 25 feet (c. 7 m), with a claimed 20 percent increase in concussive power (Rodenator R3 press release n.d.).

Figure 2.2 Operational use of the Rodenator (Rodenator 2010).

In the United Kingdom, Rodenator-type devices must not be used for killing wild animals (Department for the Environment, Food and Rural Affairs statement, May 2007) as it is illegal under the Wildlife and Countryside Act 1981 Section 11(1) to use any explosive, other than ammunition for a firearm, for the killing or taking of wild animals. Legally, the Rodenator can be used in the United Kingdom to collapse burrows and tunnels if there are no animals present, and used in this fashion they do not require a licence. Best practice in this instance includes the operator of such a device being satisfied that the burrows or tunnels have been cleared of all animals and that they have not been reoccupied before using the Rodenator to collapse these structures (http://www.basc.org.uk/en/departments/game-and-gamekeeping/gamekeeping/the-rodenator-and-other-similar-devices.cfm).

Operational use of the Rodenator in New Zealand for rabbit control appears limited because it is a relatively new product (Appendix 1). Best practice in New Zealand is consequently not defined but would presumably include use on burrows occupied by rabbits, leaving burrow entrances open to facilitate complete ignition of the gas, and ensuring sufficient penetration of gas into the burrows to achieve an adequate 'concussive blast' on ignition.

Welfare impact score of the Rodenator for rabbits

The manufacturer states that rabbits are killed instantly by the concussive blast generated by the Rodenator, which also causes the burrow structure to collapse (Anon. 2009). The only relevant published information found describes post-mortem observations of two rabbits recovered from a warren following the use of a device similar (if not the same) as the Rodenator in the UK (Bidewell et al. 2008). Lesions observed included burnt fur, partial alopecia and associated excoriation and extensive pulmonary haemorrhage, considered consistent with the effects of an explosion. However, there is no further information regarding the specific causes and times to unconsciousness and death in rabbits. This creates high uncertainty in assigning scores using the model, particularly for Domain 5 in Part A and overall for Part B, because it is not clear if the concussion of the explosion kills all rabbits 'instantly' (as claimed), or in some cases rabbits die as the result of severe external and internal burns, some are left (with or without injury from the explosion) within a collapsed burrow to die of suffocation or crushing, or some injured rabbits escape a treated burrow to die later as a result of their injuries.

The overall score assigned using the model was 5E (Table 2.4), based on a combination of domain impacts ranging from none to extreme and extreme negative welfare impacts over a duration of immediate to seconds.

Table 2.4: Categories and overall scores assigned for use of 'The Rodenator' for rabbit control, using the model developed by Sharp and Saunders (2008)

Impact categories from model	Category assigned	Basis
Part A Domain 1. Water deprivation, food deprivation, malnutrition	No impact	Duration of adverse effects not sufficiently long to cause significant water or food deprivation
Part A Domain 2. Environmental challenge (e.g. heat/cold)	Severe impact	Exposure to extremes of heat from a gas explosion
Part A Domain 3. Injury, disease, functional impairment	Extreme impact	Consistent with the effects of concussive injury through proximity to a gas explosion
Part A Domain 4. Behavioural, interactive restriction	Mild impact	Presence of human activity moves and keeps rabbits in burrow during application. Burrows not sealed during operational application so rabbits could bolt
Part A Domain 5. Anxiety, pain, fear, distress	No impact	Presence of gas before ignition presumably not at concentrations that induce escape behaviour through exiting burrow.
Overall impact Part A	Mild–extreme	Numerical score Part A: 5
Duration	Immediate to seconds	Assuming that death is practically simultaneous with complete ignition of gas filling the warren but low certainty of whether this very short duration always occurs
Part B mode of death	Extreme	Letter score Part B: E
Duration	Immediate to seconds	
Overall score Parts A&B	5E	

An assessment of the Rodenator, also applying the model described by Sharp and Saunders (2008) was recently completed by an expert panel in Australia (Sharp & Saunders 2010). Their overall rating of the Rodenator was 3B–3C, which contrasts with the 5E rating allocated in this assessment. This is most likely due to differences in the way Part A was applied to lethal control methods; the Australian assessment (Sharp & Saunders 2010) applied Part A to the processes before the killing method is applied – in the case of the Rodenator this was presumably the moving of rabbits into burrows before the gas mixture was applied to the burrow. However, in the assessment reported here, Part A was applied overall to a lethal control tool, as described by Beausoleil et al. (2010), so that Part A category ratings were inclusive of the domain impacts of the killing method.

The Australian panel applied Part B only to an assessment of the mode of death (Sharp & Saunders 2010) and determined that the level of suffering was 'mild', but could be none to moderate depending on the effectiveness of the application and characteristics of the rabbit burrow system. The degree of negative impact in the Part B assessment reported here was, in contrast, considered 'extreme' (Table 2.4), being through violent concussive injury following exposure to a gas ignition explosion. The Australian panel noted that duration could range from immediate to seconds to minutes, noting 'when the method is done well [the time to insensibility] will be immediate, but there could be animals that remain conscious for hours. There is no data on how often this happens…' (Sharp & Saunders 2010). These comments reflect the uncertainty noted above in assigning a duration for the Part B assessment, and further highlight a requirement for field-based data on the welfare impacts of the Rodenator, or similar systems, on rabbits.

2.7. CONCLUSIONS

- The in-burrow rabbit control methods currently registered and available for use in New Zealand are chloropicrin and magnesium phosphide (fumigants) and 'The Rodenator', a device designed to create an explosion in rabbit burrows through the ignition of flammable gas. Of these, magnesium phosphide appears to be recommended as 'best practice' and most commonly used. The extent of chloropicrin and Rodenator use for rabbit control in New Zealand was difficult to determine.

- The overall score assigned to chloropicrin using the model was 5F, based on a combination of moderate to severe negative welfare impacts (inhalation of toxic vapour causing extreme irritation and respiratory distress in rabbits) over a duration of minutes. The availability of recently published, formal evaluations of the humaneness of chloropicrin relative to other rabbit fumigants gave a high certainty to this assessment.

- The overall score assigned to phosphine (the gas generated by magnesium phosphide) using the model was 3D, based on a combination of mild to moderate negative welfare impacts (moderate respiratory irritation in rabbits) over a duration of minutes. The availability of recently published, formal evaluations of the humaneness of phosphine relative to other rabbit fumigants gave a high certainty to this assessment.

- The overall score assigned to the Rodenator using the model was 5E, based on a combination of welfare impacts ranging from none to extreme, with potentially extreme negative welfare impacts over a duration of immediate to seconds (consistent with close proximity of rabbits to a large explosion and very high temperatures). However, there is high uncertainty in this assessment, due to the lack of published scientific information about the range of effects on rabbits of this control method.

2.8. RECOMMENDATIONS

- A formal survey should be carried out to determine the extent of use of chloropicrin and the Rodenator for in-burrow rabbit control in New Zealand. This would assist in estimating the number of rabbits potentially affected by these in-burrow control methods.

- MAFBNZ should commission a study of the welfare impacts of the Rodenator (or similar systems) on rabbits to increase the certainty of the welfare impact score assigned.

- MAFBNZ should note the development and pending registration of a carbon monoxide pressure fumigator system in Australia (Invasive Animals CRC) as a potentially useful future addition to rabbit control tools used in New Zealand. Literature indicates that the use of carbon monoxide as a rabbit burrow fumigant is relatively more humane than either chloropicrin or phosphine-generating fumigant formulations.

2.9. ACKNOWLEDGEMENTS

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2.11. APPENDIX - AGENCY LIST

List of agencies contacted regarding availability and use of rabbit fumigant products and other 'in burrow' control tools.

Product	Agency /Contact	Nature/date of contact (2010)	Summary
Pic-Fume (chloropicrin)	*Leicesters Soil Fumigation* *6 Waitane Place* *Onekawa, Napier City,* *Hawke's Bay* *Phone: 0800 658 158* (Brian)	Phone conversation (14:00pm 27 July)	Product could be used for the purposes of rabbit control this practice had fallen out of favour, and it was almost exclusively used for the control of soil-borne pests. Was unable to give a figure regarding quantities sold/used.
Soil Fume CP (chloropicrin)	*Agricultural Fumigations Ltd* *143 Marua Rd* *Ellerslie, Auckland* *Phone: (09) 274 0484* (Anonymous)	Phone conversation (11:00am 28 July)	The product label shows that it may be used for rabbit control. Contact declined to comment on use.
Magtoxin	*Excel Biosecurity* *North Canterbury* *Phone: (03) 313 5461* (Paul Clifford)	Phone conversation (14:00pm 29 July)	With respect to fumigants for rabbit control they use Magtoxin exclusively – c. 50–60 kg of Magtoxin used at various locations around Canterbury in the last year. Chloropicrin not widely used for rabbit fumigation anymore on account of difficulties associated with using it. Likely that unused 'stockpiles' of the substance exist
Magtoxin	*Pharmochem Company* *6 Cebel Place* *Albany, Auckland* *Phone: (09) 915 3330* (Uwe Balzak)	Email (21 July)	Approximately 250 kg of Magtoxin was sold in the preceding 12 months to customers likely to be involved in rabbit control.
The Rodenator	*Pestgard* *409 High St* *Motueka 7143* *Phone: 0800 737 838* www.pestgard.co.nz (Russell Smith)	Phone conversation (13:00pm 29 July)	No figure as to how many R3 units have been sold in New Zealand but its a new product and not widely used at this stage. Had recently been demonstrated in Otago for rabbit control.

3. Leg hold traps, rotenone, alphachloralose and DRC-1339

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Report LC76

October 2010

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3.1. ABSTRACT

Project Code

- BNZ 11326: Original RFP title: How humane are our pest control tools?

- Part 1 report prepared by Beausoleil et al. (2010): Assessment of animal welfare impacts of vertebrate toxic agents and kill traps used for controlling mammalian pests in New Zealand.

- Part 2 report prepared by Fisher & Campion (2010): How humane are our pest control tools? Part 2: In-burrow rabbit control methods. Landcare Research Contract Report LC27, 26 p.

- This report comprises Part 3 of the project and covers live-capture (restraining) devices, the piscicide rotenone and the avicides alphachloralose and DRC-1339.

Business/Institution: Landcare Research, Pest Control Technologies team, Lincoln

Programme Leader: Penny Fisher

Goal

- Review literature and current information about New Zealand use of leg-hold traps for pest mammals, use of rotenone for the control of pest fish and the use of alphachloralose and DRC-1339 for the control of pest birds.

- Using information from the literature review regarding mode of action and effects of each control tool on nominated target pest (vertebrate) species, apply a framework for assessment of humaneness to evaluate the relative animal welfare impacts of each control tool.

- Identify gaps in the available information about the effects of each control tool that could be addressed in future to improve the certainty of assessments of welfare impacts.

Context of the project

- Increasingly, information on the animal welfare impacts of pest control methods is required to inform the decisions of pest management operators and policymakers. To date, there has been no comprehensive evaluation of welfare impacts associated with pest control methods used in New Zealand. The purpose of this project was to apply a recently developed Australian process to produce a ranking of the relative welfare impacts of live-capture devices, rotenone, and two avicides as used in New Zealand.

Approach

- Registration in New Zealand of the VTA products used for pest fish or pest bird control was reviewed, and the availability and operational use of these methods assessed. Literature for each of the active ingredients in the VTA formulations was reviewed to summarise mode of toxic action and reported effects in the target vertebrate pest species, mammals and humans. Information forming the basis of the welfare assessment was

primarily sourced from articles and reports published in the scientific literature, supplemented with some unpublished reports and communication with pest control experts.

- This information was used by the authors to assess the welfare impacts using a welfare assessment framework developed in Australia (Sharp & Saunders 2008) to produce a ranking of the relative welfare impacts of control tools on targeted vertebrate pest species. Each control method was assessed using both Parts A and B of the Australian framework (Sharp & Saunders 2008), with some modifications as described by Beausoleil et al. (2010). Assessment assumed 'best practice' application of each control tool in killing pests (where this was defined), so welfare impacts associated with sublethal effects were not assessed.

Outcomes

- Based on the impacts on possums from Victor No. 1 padded and unpadded leg-hold traps and the duration of exposure to these impacts, the overall welfare score assigned was 5E based on a combination of moderate domain impacts over a duration of hours (Part A) and extreme negative welfare impacts over a duration of seconds (Part B).

- For the use of alphachloralose to control black-backed gulls, sparrows, pigeons, mynahs, Euopean thrushes, starlings, blackbirds or magpies, the overall welfare score assigned using the model was 5C, based on a combination of moderate domain impacts and negative welfare impacts over a duration of hours.

- For the use of DRC-1339 to control rooks or starlings, the overall welfare score assigned using the model was 7G, based on a combination of severe negative welfare impacts over a duration of hours. A high level of uncertainty in this score was noted, due to information gaps about some of the effects of DRC-1339 in birds.

- The overall welfare score assigned to the use of rotenone for pest fish (*Gambusia*, rudd, tench, goldfish/koi/carp) control using the model was 5E, based on a combination of moderate negative welfare impacts over a duration of hours. The assumption was made that fish experience pain and distress in a similar way to mammals.

- Variable amounts of information were available in relation to the effects on mammals, birds or fish of the respective lethal control tools assessed, which affected the certainty with which scores could be allocated to domain impacts. A number of particular information gaps were identified, addressing which would improve the robustness of relative assessments of humaneness.

Recommendations

- Information gaps to be addressed for leg-hold traps include assessments of welfare impacts of various leg-hold trap types, e.g. smaller double-coil No. 1 sized traps such as the Duke, Bushmaster, BMI, Sleepycreek, and the longspring trap Bushmans Best. The apparent lack of any information relating to injury or stress incurred by feral cats or ferrets for any leg-hold trap type meant that assessments using the framework could not be made for these species.

- The degree to which alphachloralose affects consciousness in birds and whether it has anaesthetic effects in birds would be useful information, as this may indicate that welfare impacts of this control tool are reduced in terms of perception of pain.

- There was little specific information about the nature, progression and duration of the signs of DRC-1339 poisoning in birds, especially with regard to when birds lost consciousness ('became comatose') relative to the time to death. Addressing this information gap would improve the certainty of the overall scoring of the welfare impacts of this control tool.

3.2. INTRODUCTION

The welfare of wild animals subject to human management as pests is receiving increasing attention in New Zealand and overseas. A growing body of literature relevant to the welfare impacts of tools used to control vertebrate pests in New Zealand provides some basis for gauging the acceptability of different methods on the basis of animal welfare. In conjunction with a process recently developed in Australia (Sharp & Saunders 2008), such information can be used to produce relative welfare impact scores, and therefore rankings, for various lethal and non-lethal pest animal control methods.

This general approach was used by Beausoleil et al. (2010) to evaluate the relative welfare impacts of bait-delivered VTAs used to target pest mammal species in New Zealand (1080, cyanide, anticoagulants, cholecalciferol, phosphorus) or under development for registration in New Zealand (zinc phosphide, sodium nitrite and p-aminopropriophenone). For that assessment, a review panel of people with expertise in animal welfare research, pest animal management and research, and veterinary toxicology used collated reference material to apply the Australian welfare-assessment framework and to identify gaps in existing knowledge. A similar, further assessment of the relative welfare impacts of 'in-burrow' rabbit control methods (fumigants and methods based on flammable gas mixtures) was also undertaken (Fisher & Campion 2010) using the Australian process, but through expert opinion rather than panel review.

However, no assessment has been made of welfare impacts of VTAs used to control pest fish (rotenone) and pest birds (alphachloralose), or for the use of leg-hold traps for pest mammal control in New Zealand. In the latter case, this lack of information means that it is currently difficult for a pest control operator to choose an optimal trap for a targeted pest species on the basis of overall animal welfare impact. Accordingly, after collation of relevant data from scientific literature and pest control guidelines to provide reference material for assigning rankings of welfare impact, an assessment of these control tools was made using the framework developed by Sharp and Saunders (2008). As with Fisher & Campion (2010) expert opinion was used rather than a panel assessment.

3.3. BACKGROUND

3.3.1 Leg-hold traps

Restraining devices for live capture include leg-hold (foothold) traps, cage and box traps. Live-capture snares and net traps are also restraining devices, but are hardly used in New Zealand. Because leg-hold traps are the most commonly used restraining devices, they are the focus of the welfare assessment reported here. Although not rigorously quantified, the number of possums captured in leg-hold traps exceeds 100 000 per year (B. Warburton, pers. comm.). Leg-hold traps are used to a lesser extent for capturing feral cats and ferrets, although no

estimates on numbers caught annually were found. Some restrictions pertain to the sale and use of leg-hold traps in New Zealand, with recent regulatory changes prohibiting the use of certain-sized leg-hold trap types (MAF 1999).

As discussed in Chapter 1, in New Zealand the welfare impacts of traps (both kill and restraining) are evaluated using a National Animal Welfare Advisory Committee (NAWAC) guideline (http://www.biosecurity.govt.nz/animal-welfare/nawac/policies/guideline09.htm). The guideline sets out how to test leg-hold trap systems by measuring relevant welfare impacts (i.e. physical traumas), and assessing these against the pass/fail requirements specified. The guideline allows for two classes of traps (Class A and B). For Class A traps the guideline requires less frequent injuries and injuries of lower severity. Section 3.6.1 (below) has further details on the leg-hold trap types currently used in New Zealand, and those that have been assessed against the NAWAC guideline.

There is a legal requirement to inspect each leg-hold trap within 12 h after sunrise on each day the trap remains set (NPCA 2009) in order to release or euthanase trapped animals within a practicable minimum time. The requirement for acceptable euthanasia is to render the animal irreversibly unconscious as quickly and painlessly as possible, but no single euthanasing method is suitable for all mammal species. The options for euthanasia of trapped animals are (i) a blow to the head, (ii) captive bolt and (iii) firearm. Specifications for the optimal implementation of these options are given in best practice recommendations for the use of leg-hold traps for capture of possums, ferrets and feral cats (NPCA 2009). These recommendations also include other considerations of animal welfare, such as ensuring that traps are checked for captures as early as possible, placing traps in the shade during warm weather, and minimising stress to trapped animals from the approach of humans or dogs.

3.3.2 Avicides

While birds do not have as high a pest profile as mammals in New Zealand, a range of bird species including rooks, black-backed gull, blackbird, feral pigeon, myna, sparrow, starling and European thrush are regional pests, and are subject to lethal control methods. These include shooting, trapping and the use of the avicides alphachloralose (1,2-O-(2,2,2-trichloroethylidene-α-D-glucofuranose)) and DRC-1339 (3-chloro-p-toluidine hydrochloride). Alphachloralose is a narcotic that acts on the central nervous system, depressing brain activity and slowing heart and respiration rates. It was first used to control birds in the 1940s and has been used in New Zealand for bird control since the 1950s. DRC-1339 was developed by the USDA.APHIS Wildlife Research Centre in the United States, to meet the need for an effective, safe, slow-acting toxicant to allow control of bird pests including starlings and blackbirds. It was identified following screening of over 400 compounds against criteria of high toxicity and palatability to bird species, low mammalian toxicity, and a delayed onset of toxic effects that would reduce induction of bait shyness in birds (ACVM 2002b).

3.3.3 Rotenone

Rotenone has been used for fisheries management and research in many countries, and is the only VTA registered for use as a piscicide in New Zealand. Its application in aquatic environments as a pesticide is largely due to its acute toxicity to fish at concentrations that are non-toxic to other wildlife and humans. Fish are generally more sensitive than invertebrates to rotenone. Rotenone has been used effectively to control a wide range of invasive freshwater fish overseas, but is comparatively new to New Zealand. It has been used in localised control operations since 2000–2001 in the South Island when the first *Gambusia* and carp populations

were discovered there. In most instances these sites have involved highly modified or man-made ponds that contain relatively little indigenous biodiversity.

3.4. OBJECTIVES

- Review literature and current information about New Zealand use of leg-hold traps for possums, feral cats and ferrets, the use of rotenone for the control of pest fish and the use of alphachloralose and DRC-1339 for the control of pest birds.

- Using information from the literature review regarding mode of action and effects of each control tool on nominated target pest (vertebrate) species, apply a framework for assessment of humaneness to evaluate the relative animal welfare impacts of each control tool.

- Identify gaps in the available information about the effects of each control tool that could be addressed in future to improve the certainty of assessments of welfare impacts.

3.5. METHODS

To assess the welfare impacts of leg-hold traps, published and unpublished literature related to trap welfare performance testing was reviewed. Because the injuries resulting from capture are very species specific, the assessment of traps could only be based on reports that had tested the traps on the species targeted in New Zealand. To date testing has been limited to possums because of the large numbers that are trapped relative to the other two target species (feral cats and ferrets).

For rotenone, alphachloralose and DRC-1339, the register of Agricultural Compounds (ACVM 2010) was consulted to identify relevant formulations registered in New Zealand. A review of scientific literature was undertaken for each of the VTAs. In particular, specific information was sought about:

- Welfare impacts or humaneness either by review or research on captive mammals, birds or fish (for rotenone)

- The mode of toxic action or general toxic effects on fish, birds and mammals in general, including information on behavioural, physiological, and pathological responses

- Description of effects, time to death, pathology or other information relevant to evaluating welfare effects

- The toxic effects of the VTA on humans

Suitable articles were used as reference material to guide the scoring process and assess relative animal welfare impacts of each control method. This followed a modified version of the Sharp & Saunders (2008) framework, as applied to lethal control methods in an assessment of the killing technique. In applying the Australian framework, scores were based on the assumption that every targeted bird or fish would be killed following operational 'best practice' for each technique. Consequently sublethal effects were not considered in allocating scores. Welfare impacts in terms of functional impairment/pathology were scored up to the point at which animals lost consciousness (where such information was available), after which additional consequences would not be sensed by the animal.

As described in Chapter 1, current methods for assessing the impacts of events, situations or procedures on animal welfare are based on the 'Five Domains of Potential Welfare Compromise' model developed by Mellor and Reid (1994). This model provides a means of clearly separating physical or functional impacts of the animal from the emotional or affective experiences, mental states or feelings that ultimately determine its welfare or well-being. In applying 'Part A' of the framework, functional impairment/injury/pathology (domain 3) was scored as separate from the associated affective experience/feeling, e.g. breathlessness, nausea, lethargy/weakness or dizziness (domain 5). Reports of affective experiences of human poisoning victims were also considered in extrapolation for scoring the subjective mental states for domain 5.

Both Parts 'A' and 'B' of the framework were applied to each control tool, with corresponding numerical scores for overall welfare impact (Part A) and letter scores reflecting the degree of negative welfare impact of the killing method (Part B).

3.6. RESULTS

3.6.1 Leg-hold traps

The leg-hold traps most commonly used now are the smaller double-coil No. 1 sized traps such as the Victor, Duke, Bushmaster, BMI and Sleepycreek. An equivalent sized longspring trap (Bushmans Best) has recently been developed and marketed in New Zealand. Of these, the only trap types that have been assessed for their welfare impacts are the padded and unpadded Victor No. 1 and 1½ double-coil spring traps. The only target animal that has been assessed is the brushtail possum, identifying an immediate information gap for evaluation of the welfare impacts of leg-hold traps on feral cats and ferrets.

The first assessment of injuries caused by leg-hold traps to brushtail possums was reported by Warburton (1982). Injuries were then simply classified as either skin injuries (lacerations) or bone fractures (Table 3.1), noting that the smaller No. 1 sized traps were not commercially available in New Zealand at that time.

Table 3.1: Percentage of possums captured in leg-hold traps with either skin lacerations or bone fractures

	Skin lacerations	Bone fractures
Montgomery 1½*	54.5	16.0
Victor 1½	49.1	25.4

A more extensive categorisation of potential injuries was developed in the USA for assessing restraining traps (Olsen et al. 1988), which assigned scores to each injury category – higher numbers indicated injuries of increasing severity. Using this scoring system, further trials compared the injuries to possums caused by No. 1 and 1½ sized Victor double-coil traps as well as the padded version of both these traps (Warburton 1992). These trials showed that padded traps, especially the No. 1 padded trap, caused significantly less injuries than the other traps (Table 3.2). The injury scoring system developed by Olsen et al. (1988) was further modified and finally adopted as part of the ISO standard for leg-hold traps. The modified list from the ISO standard was eventually included into the NAWAC trap testing guideline (NAWAC 2005).

Table 3.2: Percentage of cumulative injury scores to possums from leg-hold trap types. A score of 0–5 is assigned to limbs that are normal or that have slight oedematous swelling. A score of 10–35 represents cutaneous, tendon or ligament lacerations. A compound fracture is assigned 200 points. Multiple injuries are additive

Trap type	*n*	0–5	10–35	40–55	60–95	100–200	205–300
No. 1½ soft catch	82	77	16	2	0	5	0
No. 1½ unpadded	74	23	58	1	1	8	8
No. 1 soft catch	63	89	10	2	0	0	0
No. 1 unpadded	72	36	51	4	7	1	0

In a more recent trial of the value of adding chain springs to reduce capture injuries in the larger traps that are either currently or about to be prohibited, Victor No. 1 unpadded traps were used as the experimental control (Warburton & Poutu 2008). The injuries reported from this trial (Table 3.3) were based on the NAWAC guideline, which categorised the injuries as mild, moderate, moderate/severe, and severe. The Victor No. 1 unpadded trap in this trial did not meet the requirements of the NAWAC guidelines for either a class A or B trap. It caused moderately and moderately severe trauma to 13 and 7 animals respectively, and these numbers exceeded the allowable number in the guidelines.

Table 3.3: Frequency of injuries in four injury categories caused to possums captured in Victor No. 1 unpadded traps

Trap type	*n*	Mild	Moderate	Moderate/Severe	Severe
Victor No. 1 unpadded	30	10	13	7	0

Apart from using the nature and extent of trap-induced injury to assess welfare impacts, there have been two trials that examined the level of stress caused by leg-hold and cage traps. One trial (Buddle et al. 1992) compared haematological values and lymphocyte responsiveness in possums that were caught for the first time in cage traps with those that had been caught on a frequent basis. The single-capture possums had haematological values and lymphocyte responses to T-cell mitogens, consistent with animals in a stressed state. A second trial (Warburton et al. 1999) assessed the concentrations of serum enzymes, electrolytes, hormones, and muscle pH in trapped possums and compared these with levels in untrapped possums (these were shot at a bait station without being aware of human presence). The traps tested were the unpadded Lane Ace gin trap, No. 1 Soft Catch trap, and wire cage trap. Cortisol levels in possums captured and held for 8 h in cage traps (37.7 nmol/L) were not significantly higher than in control animals (10.9 nmol/L), but possums captured in the Lanes-Ace and Soft Catch leg-hold traps had significantly higher levels (112.2 nmol/L and 175.9 nmol/L respectively) and serum enzymes associated with muscle damage and/or exercise showed significant increases at 8 h post-capture.

Welfare impact score of leg-hold traps for possums

The modified Sharp & Saunders (2008) framework was used to assess the use of Victor No. 1 padded and unpadded traps for possum capture (Table 3.4), using the research-based data outlined above. Similar research-based assessments of stress and injury caused to possums caught in other types of leg-hold traps currently used in New Zealand are not available, and no comparable published research information was found for stress and injury caused to feral cats or ferrets caught in any type of leg-hold trap. However, on the basis of a recent Australian assessment where the Sharp & Saunders (2008) framework was applied by an expert panel, the overall score for leg-hold trapping (trap types not specified) of feral cats was 5B (moderate impacts over a duration of hours) (Sharp & Saunders 2010).
Based on the impact caused to possums from leg-hold traps and the duration of exposure to these impacts, the overall score assigned using the framework was 5E (Table 3.4), based on a combination of moderate domain impacts over a duration of hours (Part A) and extreme negative welfare impacts over a duration of seconds (Part B). The data available for this assessment enable the scores to be assigned with certainty.

Table 3.4: Categories and overall scores assigned for Victor No. 1 padded and unpadded traps for possum control, using the framework developed by Sharp and Saunders (2008)

Impact categories from framework	Category assigned	Basis
Part A Domain 1. Water deprivation, food deprivation, malnutrition	Mild impact	Duration of adverse effects (up to 18 h) could result in food deprivation. The degree of water deprivation would depend on ambient temperatures.
Part A Domain 2. Environmental challenge (e.g. heat/cold)	Mild impact	Potential for temperature increase or decrease depending on ambient temperatures, and trap location.
Part A Domain 3. Injury, disease, functional impairment	Moderate impact	Pathology shows some lacerations and occasional fractures. Elevated serum enzymes associated with muscle damage and/or exercise.
Part A Domain 4. Behavioural, interactive restriction	Mild impact	Animal constrained by capture.
Part A Domain 5. Anxiety, pain, fear, distress	Moderate impact	Cortisol levels elevated to levels significantly higher than in control animals.
Overall impact Part A Duration	Moderate Hours	Numerical score Part A: 5
Part B mode of death Duration	Extreme suffering Seconds	Animal killed by a blow to the head but this does not always cause instantaneous insensibilty. Letter score Part B: E
Overall score Parts A&B	5E	

3.6.2 Alphachloralose

Animal Control Products is the only New Zealand registrant of alphachloralose, which is available in variety of formulations depending on the intended use and target bird species (Table 3.5). A controlled substances license is not required to possess or use alphachoralose, and it can be used for the control of black-backed gulls, sparrows, pigeons, mynahs, European thrushes, starlings, blackbirds and magpies (Table 3. 5). Alphachloralose is delivered to birds in bait types including maize, peas or wheat for pigeons and wheat, bread or oatmeal for house sparrows.

Table 3.5: Details of alphachloralose formulations registered for use against targeted pest bird species in New Zealand (AVCM 2010). Annual sales estimates for 2009/10 were provided by Animal Control Products

Registration number	Trade name (est. annual NZ sales 2009/10)	Formulation details and active concentration	Target species
V009536	Alphachloralose powder (44 kg)	97% w/w powder for the preparation of 'bread baits' with a concentration of 8–10% w/w. Achieved by mixing 500 g of margarine with 280g alphachloralose powder. This is then spread on slices of bread and cut into 25 mm x 25 mm squares	Black-backed gulls (*Larus dominicanus*)
V002611	PESTOFF TREATED WHEAT (5600 kg)	Wheat treated with alphachloralose at a concentration of 22 g/kg (2.2% w/w) for control of birds in wheat crops	Sparrows (*Passer domesticus*)
V003282	PESTOFF TREATED PEAS (500 kg)	Peas treated with alphachloralose at a concentration of 25 g/kg (2.5% w/w) for control of birds in pea crops	Pigeons (*Colombia livia*)
V004001	PESTOFF BIRD CONTROL PASTE (370 kg)	Paste containing 25 g/kg (2.5%w/w) alphachloralose for the control of nuisance birds in horticulture and agriculture. The paste is spread on slices of bread and cut into squares (25 mm x 25 mm)	Mynahs (*Acridotheres tristis*) Blackbirds (*Turdus merulus*) European thrushes (*Turdus philomenos*) Magpies (*Gymnorhina tibicens*) Starlings (*Sturnus vulgaris*) Sparrows
V003622	PESTOFF TREATED BARLEY (400 kg)	Barley treated with 25 g/kg (2.5% w/w) alphachloralose for the control of birds in barley crops	Sparrows

In New Zealand, alphachloralose has been used to control house sparrows in piggeries and poultry runs in Hawke's Bay (ACVM 2010). Aerial application of bread treated with alphachloralose gave an 85 percent reduction in the number of black-backed gulls at Napier Airport (Caithness 1968). Although alphachloralose is effective against pigeons and sparrows in rural areas, it is not recommended for use in agricultural districts due to the risks to non-target species (ACVM 2010).

Description and mode of toxic action

Alphachloralose is a narcotic agent that induces disassociation and somnolence, with essential bodily functions and processes remaining unaffected. It has been in use for many years as an immobilising agent in birds, as a rodenticide, and as a general anaesthetic in laboratory animals. In human medicine it has been used as a soporific and anaesthetic (Krieger 2001). It is a condensation product of glucose and chloral hydrate, existing as both the alpha and beta isomers, with only the alpha isomer possessing narcotic properties. It has selective oral toxicity to birds, with mammals apparently less susceptible (Table 3.6).

Alphachloralose possesses both stimulant and depressant properties, with its mechanism of action upon the central nervous system (CNS) not clearly understood. Following ingestion alphachloralose is hydrolysed to chloral, which is then reduced to trichloroethanol, itself a CNS depressant. This then undergoes additional metabolism; however, its overall role in alphachloralose intoxication is unclear (Segec et al. 2006). At low exposures excitation of the CNS occurs through suppression of descending inhibitory mechanisms, while at higher exposures neuronal suppression in the ascending reticular activating system results in CNS depression (Balis & Munroe 1964).

Table 3.6: Comparative LD50 values of alphachloralose in a range of species (Krieger 2001)

Species	Oral LD50 (mg/kg)
Rat	300–400
Cat	100
Dog	600–1000
Starling	75
Redwing blackbird	32
Crow	42
Pigeon	178
House sparrow	42
Mallard	42

Toxic effects on birds

Typically, active concentrations of 2–2.5 percent alphachloralose in bait will stupefy birds that ingest it, allowing them to be collected and either recovered and released, or killed humanely. Best practice use of alphachloralose is as a stupefying/immobolising agent rather than a lethal poison, as this allows collection and rehabilitation of non-target bird species for release. Lethal concentrations in bait are greater than 6 percent, with lethal exposures augmented by bait application in cool weather (Nelson 1994), presumably due to the effects of alphachloralose on thermoregulation. Symptoms of poisoning/narcosis in birds proceed through the following stages (Agricultural Chemical Board 1977, cited in Nelson 1994):

- Cessation of activity and inability to perch properly. Affected birds will stagger 'drunkenly' when disturbed. Eyes remain open and affected birds cannot readily be captured.

- Affected birds stand in a hunched position with eyes closed or flickering. They will not move if approached quietly but may elude capture if disturbed. Can be caught if some degree of care is exercised.

- Affected birds become recumbent with head drooping and eyes closed. They remain still, apart from occasional wing and tail flapping, but will move when touched or handled. Birds can be captured with ease.

- Affected birds remain motionless even when touched and may die if left undisturbed.

Studies of the effects of alphachloralose on birds report 'alphachloralose-induced sedation did not appear to cause stress...untreated birds showed no negative response to birds in the same cage undergoing alphachloralose-induced sedation' (Woronecki et al. 1992). Affected birds in this study displayed torticollis (lateral flexion contracture of the cervical spine musculature so that the head is tilted to one side), fluid in the oral cavity, respiratory irregularities and shivering. Tonic convulsions such as those induced by strychnine poisoning were not observed, but convulsion-like was behaviour observed when birds in mid- to deep sedation were disturbed or startled by other affected birds staggering in near proximity (Woronecki et al. 1992). The latter observation is suggestive of hypersensitivity, and was also reported with alphachloralose use on gulls (Woronecki et al. 1990).

Woronecki et al. (1990) report that affected waterfowl including Canada geese (*Branta canadensis*) and mallard ducks (*Anas platyryhnchos*) did not appear 'harassed', there were no painful or stressful symptoms, the birds appeared to be in a deep sleep when they were removed from the [baited] area and unaffected birds did not react to affected birds. A reported advantage of using alphachloralose for bird control was that lethally exposed (immobolised) birds could be collected for euthanasia before death occurred (Woronecki et al. 1990).

Summary information (ACVM 2002a) noted that pigeons in the early stages of narcosis seem to fly quite normally, even though they can neither stand upright nor judge distances for landing or taking off. The average period between first feeding and first symptoms of narcosis was 28 min (range 10–120 min), with the period of narcosis usually lasting some 10–20 h (range 2–72 h). The effect of chloralose on corvids is to disturb their equilibrium after 10–20 min, and then to produce complete torpor, causing perched birds to fall to the ground, with this narcosis lasting 5–12 h.

Toxic effects in humans

Tempe and Kurtz (1972, cited in Krieger 2001) examined 22 cases of acute chloralose poisoning in humans (none fatal), with most of these arising from attempted suicides. The characteristic symptom of chloralose poisoning in humans was the induction of a comatose state, which may be preceded by vomiting, trembling, vertigo and feelings of inebriation. In sufficiently large doses coma may be induced within minutes, although typically it occurs 1–2 h following ingestion. Awakening from the comatose state may take upwards of several hours in cases of mild poisoning and as much as 96 h in cases of severe poisoning. It is usually accompanied by headache, stiffness and weakness; however, there are generally no longer term effects (Tempe and Kurtz 1972, cited in Krieger 2001).

Welfare impact score of alphachloralose for pest bird control

Best practice recommendations maximise the chances of capturing targeted birds during alphachloralose baiting programmes, and minimise interference with bait by non-target species (ACVM 2002a). Bait is laid about half an hour before dawn, at a density suggested by takes during prebaiting (slightly more than the average daily take during prebaiting). For maximum efficacy, more than one day's baiting is usually necessary allowing at least 2 days between successive applications. Operators watch the baited area from a concealed position in order to observe in which direction affected birds fly off. The search for affected (narcotised) birds should start within half to three-quarters of an hour after the last bird has fed, or after the first bird has shown signs of being affected, whichever is earlier. The whole baited area should then be searched, especially around the roosting points. A final search should then be undertaken three-quarters of an hour after uneaten bait has been picked up. Search effort should take account of observed dispersal of affected birds from the baited area; for example, house sparrows appear to disperse more rapidly after feeding and may be harder to find than larger birds. Birds should not be picked up before they are immobile and all affected birds should be collected. Any bird not positively identified as belonging to the pest species must be placed in appropriate confinement until it recovers and can be released. Affected target birds must all be killed quickly and humanely – this could be through cervical dislocation, although gassing with carbon tetrachloride is also recommended.

The scores shown in Table 3.7, in evaluating the welfare impacts of alphachloralose for pest bird control, are on the assumption that best practice as described above is followed and that narcotised birds are euthanased, and non-target birds allowed to recover before release. The overall score assigned using the framework was 5C (Table 3.7), based on a combination of moderate domain impacts and negative welfare impacts over a duration of hours. There was a high degree of certainty in assigning the scores because of the extent of available information about the effects on bird species, particularly given that alphachloralose is recognised as a narcotic with potentially anaesthetic properties in birds.

Table 3.7: Categories and overall scores assigned for alphachloralose for pest bird control, using the framework developed by Sharp and Saunders (2008)

Impact categories from framework	Category assigned	Basis
Part A Domain 1. Water deprivation, food deprivation, malnutrition	No impact	Narcotised birds are collected within hours
Part A Domain 2. Environmental challenge (e.g. heat/cold)	No impact	Ability of birds to thermoregulate may be affected; however, this is not an environmental challenge but a functional impairment (domain 3)
Part A Domain 3. Injury, disease, functional impairment	Moderate impact	Incoordination, loss of muscle function, followed by immobility. Thermoregulation may be reduced in very cold or hot environmental conditions, but assumed birds are collected before this impinges on survival. Domain 3 impacts may occur over a number of hours until birds are collected after searching
Part A Domain 4. Behavioural, interactive restriction	No impact	No restraint of birds in unaffected state
Part A Domain 5. Anxiety, pain, fear, distress	Moderate impact	Human handling of birds that are conscious but narcotised to the point of immobilisation may occur. This may induce moderate anxiety or distress until euthanasia is undertaken
Overall impact Part A Duration	Moderate Hours	Numerical score Part A: 5
Part B mode of death Duration	Moderate Seconds	Either through cervical dislocation or inhalation of carbon tetrachloride Letter score Part B: C
Overall score Parts A&B	5C	

3.6.3 DRC-1339

Animal Control Products is the only New Zealand registrant for DRC-1339, which is available as a powder for incorporation into preparations for rook and starling control (Table 3.8). A controlled substances licence is required for its possession and use. Poisoning with DRC-1339 in bread and fat dripping bait is the recommended method for controlling larger populations of rooks (NPCA 2006); alphachloralose has been used in the past for rooks, but is not considered as effective as DRC-1339.

Table 3.8: Details of DRC-1339 formulations registered for use against targeted pest bird species in New Zealand (AVCM 2010). Annual sales estimates for 2009/10 were provided by Animal Control Products

Registration number	Trade name (est. annual NZ sales 2009/10)	Formulation details and active concentration	Target species
V002624	DRC-1339 (10 kg)	97% w/w powder for the preparation of 'bread baits'. 2.5g DRC-1339 is mixed with 1 L melted dripping into which 20 mm x 20 mm squares of bread are then dipped. The baits are rolled in flour, dried and then frozen for later use	Rooks (*Corvus frugilegus*) Starlings

Description and mode of toxic action

Technical-grade DRC-1339 is a pale yellow solid that is highly soluble in water and other polar solvents. The commercially available product is a fine white powder (ACVM 2002b).

Following absorption into the bloodstream DRC-1339 impacts upon liver and kidney function, with death most likely resulting from uraemic poisoning. Damage to the kidneys prevents the excretion of waste metabolites/products causing these to accumulate to lethal levels (ACVM 2002b). Toxic doses of DRC-1339 are highly variable across species, with the compound being highly toxic to some families of birds and to cats, yet only slightly to moderately toxic for other avian families and most mammals (Dawes 2006). The mode of action of DRC-1339 appears to differ depending on the susceptibility of the species. While not definitively understood, it is thought that the presence of renal deacetylase is the main determinant of toxicity. Highly susceptible bird species possess renal deacetlyase, whereas less sensitive mammals and bird species do not (Eisemann et al. 2003). In sensitive species death is caused by liver failure and the build-up of uric acid deposits in the cardiovascular system. This leads to circulatory impairment, nephrotoxicity, uraemic poisoning and ultimately death (Nikodemusz & Imre 1982, cited in Dawes 2006). In less sensitive species death appears to be a caused by an alternative mechanism acting to depress the central nervous system, resulting in cardiac or respiratory arrest (Eisemann et al. 2003).

Toxic effects on birds

The symptoms of DRC-1339 poisoning in birds are characterised by the following (ACVM 2002b):

- An initial increase in water consumption, followed by a sharp drop in water intake.
- Approximately 4 h prior to death affected birds cease to eat and drink, becoming listless and inactive. Birds appear to doze and perch with feathers ruffled.
- Breathing rate increases and becomes more laboured prior to affected birds lapsing into a comatose state. Death follows shortly after.

Ingestion of a lethal dose is not accompanied by signs such as as flapping, convulsing or vocalisation. In birds, time to death through DRC-1339 toxicity is dose-dependent, i.e. one particle of the treated bait is sufficient to kill a feeding starling, but the more baits eaten, the sooner death occurs (ACVM 2002b). Thus, the time of death and the consequent dispersal of

dying birds can be controlled to some extent by varying the ratio of treated to untreated bait. A high concentration of treated bait, and the increased possibility of one bird eating three, four, or five poisoned baits, reduces the dispersal area of the poisoned birds (DeCino et al. 1966). Depending on the number of baits consumed, death of starlings has been observed 3–50 h after ingestion, but occasionally up to 80 h (Dawes 2006).

Most rooks die within 1 to 3 days after consuming treated bait (ACVM 2002b), and most die at their roosts and along flight lines to the feeding areas, or else under trees or beside water between the bait line and the rookery or roost. In midsummer, however, dead rooks have been found up to 20 km from the bait line. Nikodemusz and Imre (1982, cited in Dawes 2006) have shown that there are no changes to gross and microscopic pathology in birds (rooks and pheasants) following ingestion of a sublethal dose.

Given the apparent differences in the effects of DRC-1339 on birds and mammals, information regarding effects on humans and mammal species (see below) was not considered directly in undertaking the welfare impact assessment for bird species.

Toxic effects on humans and other mammals

No specific reports of toxic effects in humans were found in the literature. Using data obtained from animal studies, the United States Environmental Protection Agency has placed DRC-1339 in Toxicity Category I for exposure via inhalation, and for its effects upon the skin and eyes (US EPA 1995). This suggests that DRC-1339 has, at least, strong irritant and/or corrosive properties to mucous membranes. Various MSDS information for the product 'Starlicide' describe signs/symptoms of exposure in humans as depression, haematuria, diuresis, and burning of skin and eyes.

Felsenstein et al. (1974) found that DRC-1339 induced methaemoglobinemia when given in lethal doses to mice and rats, but not in chickens. Death in rodents was preceded by CNS depression, muscular weakness and cyanosis occurring at toxic doses. In laboratory studies where rats and cats were administered DRC-1339 intravenously or interperitoneally, Borison et al. (1975) concluded that the compound appears to be directly cytotoxic, producing superficial necrosis on serosal surfaces after 3–8 h of contact.

Welfare impact score of DRC-1339 for pest bird control

Best practice recommendations for the use of DRC-1339 (ACVM 2002a) are that the bait should be applied before the birds arrive for their first morning meal, and that one bait application should last up to 3 days. Other recommendations for best practice use in rook control mostly deal with maximising bait uptake by targeted populations (NPCA 2006).

The overall score assigned to the use of DRC-1339 for pest bird control using the framework was 7G (Table 3.9), based on a combination of severe negative welfare impacts over some hours. The allocation of a duration in the order of 'hours' was conservative as there was little information about when the onset of signs of DRC-1339 poisoning occurs in birds, and also when birds lost consciousness ('became comatose') relative to the time to death. Thus there was uncertainty as to the likely duration of potential pain or distress due to toxic effects, before death.

Table 3.9: Categories and overall scores assigned for DRC-1339 for pest bird control, using the framework developed by Sharp and Saunders (2008)

Impact categories from framework	Category assigned	Basis
Part A Domain 1. Water deprivation, food deprivation, malnutrition	Mild	Drop in water intake reported as an eventual effect of DRC-1339 toxicosis
Part A Domain 2. Environmental challenge (e.g. heat/cold)	No impact	
Part A Domain 3. Injury, disease, functional impairment	Severe impact	Uraemic poisoning and damage to kidneys prevents the excretion of waste metabolites/products causing these to accumulate to lethal levels
Part A Domain 4. Behavioural, interactive restriction	No impact	
Part A Domain 5. Anxiety, pain, fear, distress	Moderate	Distress through laboured breathing, assumed pain from kidney damage and moderate thirst
Overall impact Part A Duration	Severe Days	Numerical score Part A: 7
Part B mode of death Duration	Severe Days	Loss of consciousness is not immediate - uraemic poisoning is assumed to cause pain, if not outward signs of distress, before death Numerical score Part B: G
Overall score Parts A&B	7G	

3.6.4 Rotenone

Rotenone is a naturally occurring compound derived from the roots of certain tropical plant species, and is acutely toxic to fish and invertebrates. It has been used for centuries by the indigenous people of areas where the plants grow (Southeast Asia and South America) to harvest fish for food, as a commercial insecticide for at least 150 years, and for fisheries management purposes since 1930.

Current New Zealand registrations of formulations containing rotenone include Derris dust (insecticidal garden product) and Pestene insect powder (ecoparasiticide), which are available to the public. Rotenone is the only active ingredient registered for use as a piscicide in New Zealand, in the form of cube root powder and slurry. It is not registered as a piscicide by ACVM but was approved for such use by the Environmental Risk Management Authority (ERMA) in 2003, at a maximum concentration of 200 µg/L or 0.2 ppm (after mixing).

The Department of Conservation applies rotenone to selected water bodies to eradicate introduced pest fish. The majority of fish control operations in New Zealand have been undertaken since 2001, with approximately 470 kg of cube root powder used over the following two summers to treat 20 water bodies (from 16 to 69 000 m3). Before 2001, rotenone liquid and pellet formulations were applied in New Zealand to remove grass carp from two lakes in 1981 and 1999, respectively (Dean-Spiers 2009).

Description and mode of toxic action

Cube root powder (6–9 percent rotenone) is derived from the ground roots of rotenone-bearing plants. Rotenone formulations are available as crystalline preparations, emulsified solutions, and dusts/powders.

Rotenone acts by disrupting cellular aerobic respiration, which is a series of metabolic processes which living cells use to produce energy through the oxidation of organic substances derived from food. Specifically, rotenone is an inhibitor of the complex I electron transport system in cell mitochondria. Cellular uptake of oxygen is blocked and the production of cellular energy is greatly reduced. Death results from tissue anoxia, generally through cardiac and neurological failure. Signs of exposure include vomiting in mammals, lack of coordination, muscle tremors, clonic convulsions, and respiratory failure. Most mammal species are relatively resistant to oral intake of rotenone (Ling 2003), although the lethal-dose values indicate that the degree of toxicity varies between mammal species. Rotenone is not water-soluble, so that absorption of the compound in the stomach and intestines is relatively slow and inefficient, which may account for its much lower oral toxicity compared with parenteral routes. Rotenone delivered parenterally is relatively fast acting, with death in as little as 2 h (Hayes 1982).

Toxic effects on fish

Rotenone can inhibit cellular respiration in almost every living organism, but fish are highly susceptible because rotenone can efficiently and quickly enter the blood stream through the gills. This is compounded by the fact that the gills have a relatively high lipid content that the rotenone favours, since it is water insoluble. Rotenone usually kills fish within 24–36 h. When poisoned with rotenone, fish swim erratically and move to shallower water or come to the surface, showing gasping / gaping behaviour. After that, they lose equilibrium, ventilation rates decline and they die. Some dead fish sink to the bottom, while other fish remain at the surface near the edges of the treated water.

Common carp dosed with rotenone exhibited symptoms, such as erratic darting around, then hanging vertically near the water surface, within 30 min after gavage. Fish that died slowly sank to the bottom and had slow opercular movement for 20–40 min before dying within 16 h (Fajt 1996).

Reaction times (time to loss of equilibrium) were measured in the laboratory and in the field for a number of freshwater fish species. Times in the laboratory ranged from 7.5 min in the Natal yellowfish (*Barbus natalensis*) to 60 min for the common carp (*Cyprinus carpio*). Field tests showed similar average times, but greater variation between individuals was observed (Rowe-Rowe 1973). Time to loss of equilibrium varied with fish size and water temperature, but in almost all cases reaction time was within 2–3 h (Rowe-Rowe 1973).

Chadderton et al. (2001) report general behavioural observations of fish (*Gambusia*, rudd, tench, shortfin eels, goldfish/koi) at or near the surface following rotenone treatment of ponds in New Zealand. Reaction rates to rotenone varied between species although the order of response was consistent. *Gambusia* and rudd were the first fish observed, usually within 10–25 min of application, whereas eels, tench and goldfish were usually not observed until 40 min after treatment.

The initial reaction for most species was 'frantic' swimming at the water surface. As the length of exposure time increased, activity at the surface lessened. Fish usually remained at the surface, upright and still swimming, although at regular intervals individuals darted across the surface and then resumed swimming upright very slowly. They would then begin to lose equilibrium or orientation and often while still swimming they would roll over onto one side, right themselves and roll over again. Eventually the fish were no longer able to right themselves and hung on their sides or sometimes upside down at the water surface, often twitching. Finally the fish became completely immobile, usually floating on their sides or belly up in a state of torpor or having died.

Gambusia was usually observed coming to the surface within about 10 min, and progressed to slow surface swimming then into the darting behaviour characterised by short bursts. Smaller fish began to lose orientation after as little as 15 min and succumbed within about 20–25 min and typically sank once torpor had set in, whereas large fish often maintained orientation and movement for 20–30 min, and in some instances they were still alive up to 3 h post-poisoning.

Rudd appeared at the pond surface typically within 25–30 min, with a more active reaction to rotenone, including fast active surface swimming that progressed into frantic jumping out of the water and thrashing around at the surface. They rapidly lost buoyancy control and orientation and were unable to maintain the tail horizontally at the surface, but this behaviour was often punctuated by rapid darting and additional jumping behaviour. Total loss of buoyancy control rapidly followed and the fish lay on their side or belly up at the surface as torpor set in. The last rudd were usually dead by approximately 1 h after rotenone application.

Tench, shortfin eels and goldfish were slower to react. The first large eels were usually observed within 30–40 min of application, whereas in the absence of juvenile fish it was often over an hour before the first tench appeared. Mature tench, rudd, goldfish, koi and perch followed similar behaviour patterns to *Gambusia* but were also observed porpoising and lunging through the surface water and sometimes jumping clear of the water. These behaviours were repeated at intervals. As the effects of rotenone progressed, the larger fish slowed down and some began to roll over repeatedly at the water surface as they were swimming. Large adult tench were often the last fish observed doing slow barrel rolls at the surface 2–3 h after poisoning. Eels reacted by swimming across the water surface, and as they became more stressed they would swim frantically toward the edge of the pond and attempt to leave the water. Where successful, they lay motionless on the banks, gulping air. Otherwise they often accumulated at the pond edges, lying on their sides in an apparent state of torpor. However, if disturbed they were capable of swimming away in short rapid bursts that made capture difficult. Torpid eels became evident after about an hour, but active fish continued to appear over the next 2 h and live adults were often found the following morning.

Toxic effects on humans and mammals

Rotenone is moderately toxic to mammals, but there is a wide variation between species, with pigs being especially sensitive. Signs of acute poisoning in mammals include initial respiratory stimulation followed by respiratory depression, incoordination, clonic or tonic convulsions, muscle tremors and death from respiratory failure (Ray 1991).

Human fatalities are rare, perhaps because rotenone formulations sold usually have low concentrations (1–5 percent), and its irritating action causes prompt vomiting. Acute local effects (from inhalation of dust formulations) include conjunctivitis, dermatitis, sore throat,

congestion, and vomiting. Inhalation of high doses can cause increased respiration followed by depression and convulsions (Anon. 2001). Two reports were found of human fatalities from consumption of commercially available rotenone, though ingestion of the roots of plants known to contain rotenone has been reported as a method of suicide in natives of Papua New Guinea (Wood et al. 2005). A 47-year-old woman died after ingestion of approximately 200 mL of 0.8 percent rotenone solution. She presented for medical treatment with a reduced level of consciousness, metabolic acidosis and respiratory compromise but subsequently died (Wood et al. 2005). A 3½-year-old girl died after ingesting 10 mL of Galicide (containing 6 percent rotenone). The child gradually lost consciousness over a 2-h period and died of respiratory arrest 6 h later (De Wilde et al. 1986).

Welfare impact score of rotenone for control of pest fish

The overall score assigned to the use of rotenone for pest fish control using the framework was 5E (Table 3.10), based on a combination of moderate negative welfare impacts over a duration of hours. The assumption was made that fish experience pain and distress in a similar way to mammals, i.e. the toxic effects of rotenone exposure across fish gills being primarily those of cellular oxygen deprivation causing 'breathlessness' and distress, and scores allocated using the behavioural information for fish described above.

Table 3.10: Categories and overall scores assigned for rotenone for pest fish control, using the framework developed by Sharp and Saunders (2008)

Impact categories from framework	Category assigned	Basis	
Part A Domain 1. Water deprivation, food deprivation, malnutrition	No impact	Initial action of rotenone within minutes, death within hours	
Part A Domain 2. Environmental challenge (e.g. heat/cold)	No impact		
Part A Domain 3. Injury, disease, functional impairment	Moderate	Cellular oxygen deprivation that is reversible if exposure to rotenone in water is discontinued, especially in earlier stages of toxicosis before buoyancy is lost.	
Part A Domain 4. Behavioural, interactive restriction	Mild	Forces fish to water surface, presumably in search of oxygenated water as a response to metabolic effects of rotenone	
Part A Domain 5. Anxiety, pain, fear, distress	Moderate	Assume 'darting' and 'skimming' behaviour of fish indicates mild distress, but that fish that have lost buoyancy experience at least moderate distress	
Overall impact Part A	Moderate	Numerical score Part A: 5	Duration: Hours
Part B mode of death	Moderate	Letter score Part B: E	Duration: Hours
Overall score Parts A&B	5E		

3.7. CONCLUSIONS

- Based on the impacts on possums from Victor No. 1 padded and unpadded leg-hold traps and the duration of exposure to these impacts, the overall welfare score assigned was 5E based on a combination of moderate domain impacts over a duration of hours (Part A) and extreme negative welfare impacts over a duration of seconds (Part B).

- For the use of alphachloralose to control black-backed gulls, sparrows, pigeons, mynahs, Euopean thrushes, starlings, blackbirds or magpies, the overall welfare score assigned using the model was 5C, based on a combination of moderate domain impacts and negative welfare impacts over a duration of hours.

- For the use of DRC-1339 to control rooks or starlings, the overall welfare score assigned using the model was 7G, based on a combination of severe negative welfare impacts over a duration of hours. A high level of uncertainty in this score was noted, due to information gaps about some of the effects of DRC-1339 in birds.

- The overall welfare score assigned to the use of rotenone for pest fish (*Gambusia*, rudd, tench, goldfish/koi/carp) control using the model was 5E, based on a combination of moderate negative welfare impacts over a duration of hours. The assumption was made that fish experience pain and distress in a similar way to mammals.

- Variable amounts of information were available in relation to the effects of the mammal, bird and fish of the lethal control tools assessed, which affected the certainty with which scores could be allocated to domain impacts. A number of particular information gaps were identified, addressing which would improve the robustness of relative assessments of humaneness.

3.8. RECOMMENDATIONS

- Information gaps to be addressed for leg-hold traps include assessments of welfare impacts of various leg-hold trap types, e.g. smaller double-coil No. 1 sized traps such as the Duke, Bushmaster, BMI, Sleepycreek, and the longspring trap Bushmans Best. The apparent lack of any information relating to injury or stress incurred by feral cats or ferrets for any leg-hold trap type meant that assessments using the framework could not be made for these species.

- The degree to which alphachloralose affects consciousness in birds, and whether it has anaesthetic effects in birds, would be useful information, as this may indicate that welfare impacts of this control tool are reduced in terms of perception of pain.

- There was little specific information about the nature, progression and duration if the signs of DRC-1339 poisoning in birds, especially with regard to when birds lost consciousness (‘became comatose’) relative to the time to death. Addressing this information gap would improve the certainty of the overall scoring of the welfare impacts of this control tool.

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4. Appendices

Appendix 1

Scoring matrix for Part A: overall welfare impact, described by the Sharp & Saunders (2008) framework (from http://www.daff.gov.au/__data/assets/pdf_file/0007/929905/humaneness-pest-animals-appendix.pdf)

Overall impact on welfare	Duration of impact				
	Immediate to Seconds	Minutes	Hours	Days	Weeks
EXTREME	5	6	7	8	8
SEVERE	4	5	6	7	8
MODERATE	3	4	5	6	7
MILD	2	3	4	5	6
NO IMPACT	1	1	1	1	1

Scoring matrix for Part B: assessment of mode of death, described by the Sharp & Saunders (2008) framework (from http://www.daff.gov.au/__data/assets/pdf_file/0007/929905/humaneness-pest-animals-appendix.pdf)

Level of suffering	Time of insensibility				
	Immediate to Seconds	Minutes	Hours	Days	Weeks
EXTREME	E	F	G	H	H
SEVERE	D	E	F	G	H
MODERATE	C	D	E	F	G
MILD	B	C	D	E	F
NO IMPACT	A	A	A	A	A