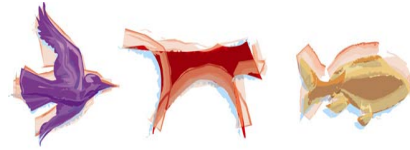




Australian Government

Department of the Environment, Water, Heritage and the Arts



Invasive Animals CRC

**ASSESSING THE HUMANENESS AND EFFICACY OF A NEW FERAL PIG
BAIT IN DOMESTIC PIGS**

**REPORT FOR THE AUSTRALIAN GOVERNMENT
DEPARTMENT OF THE ENVIRONMENT, WATER, HERITAGE AND THE ARTS**

March 2010

Study: PC0409

**Veterinary Services Division,
Institute of Medical and Veterinary Science**

Study Commencement Date: 19 March, 2008

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Signature Page Responsible Personnel

We the undersigned, hereby declare that this study was undertaken as described below, and that the report accurately reflects the results obtained.

Study Position	Name	Signature	Date
Study Director	Ms Susan Porter	_____	24 June 2009
Test Facility Management	Dr Tim Kuchel		24 June 2009

Abbreviations used in this Study Plan.

APVMA	Australian Pesticides and Veterinary Medicines Authority
CRC	Co-operative Research Centre
GPARC	Gilles Plains Animal Resource Centre
HR	heart rate
IA	intra-arterial
IM	intramuscular
IMVS	Institute of Medical and Veterinary Science
metHb	methaemoglobin
mL	millilitre
SOP	standard operating procedure(s)
VSD	Veterinary Services Division

1. Executive Summary

Ten pigs previously trained to eat non-toxic bait were fed either toxic bait (n=5) or non-toxic bait (n=5). On the previous day the animals had a catheter inserted into the carotid artery to facilitate arterial blood sampling for methaemoglobin and clinical pathology analyses.

The amount of toxic bait consumed ranged from 179 to 822 mg/Kg of body weight. All animals that consumed the toxic bait died within two hours of consumption with the exception of the pig with the lowest consumption (179 mg/kg), which died three hours post-consumption.

All animals had methaemoglobin levels greater than 10% within 20 minutes of consuming the toxic bait. At the time of death, the methaemoglobin levels ranged from 77.8-86.5%. The nitrite containing toxic baits fed to the pigs in this study were efficacious. In the opinion of the authors, the symptoms leading to death and duration of display of these symptoms would suggest that sodium nitrite satisfies a general understanding of what a humane poison would be.

2. Sponsor Representatives and Contacts

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3. Animal Welfare and Institutional Animal Care and Use Committee Review

The study was reviewed and approved by the Animal Ethics Committee of the IMVS. The Ethics Committee number for this study was 123/08. This study followed the requirements of the Animal Ethics Committee of the IMVS, including the Australian Code of Practice for the Care and Use of Animals for Scientific Purposes, 7th edition, 2004.¹

4. Introduction

Feral pigs cause a large and diverse range of impacts where they occur. They cause agricultural losses to stock and crops conservatively estimated at \$106 million annually. They cause numerous environmental impacts, including wildlife predation and habitat destruction, and have been listed as a threatening process in NSW and nationally. They spread endemic disease and will potentially spread exotic disease should one enter Australia.

Poisoned baiting of feral pigs is the most efficacious and cost-effective control technique. Currently three toxins are used for feral pig control- sodium fluoroacetate (1080), warfarin and yellow phosphorus. Each of these toxins has potential problems. 1080 is required in doses that potentially compromises the safety of non-target species. 1080 can also lead to profuse vomiting in feral pigs which may cause non-target risks and potential welfare compromises. 1080 in pen trials has never killed all poisoned feral pigs, even at high doses, and the toxin has no antidote in the event of non-

target poisoning. Phosphorus causes severe clinical disease over several days and almost certainly causes welfare compromise in poisoned feral pigs. In addition, its use pattern exposes non-target scavengers to potential poisoning. Phosphorus use is not supported in NSW and Queensland by land management agencies. NSW Primary Industries has assessed phosphorus as inhumane and unacceptable for use. Warfarin is an efficacious feral pig toxin. However, it is not registered for use by the APVMA. Agencies such as the NSW National Parks and Wildlife Service and the ACT Parks and Conservation Service which have used the toxin under experimental permits are phasing out its use due to animal welfare concerns. NSW Primary Industries has reviewed the toxin as inhumane and unacceptable for use in controlling feral pigs.

The PIGOUT[®] project commenced in January 2004 with the aim of developing and registering a manufactured 1080 feral pig bait that would improve the welfare and target-specificity properties of feral pig baiting. To be successful, the bait had to be highly attractive to pigs, cheap, target-specific and easy to use. The 1080 also had to be presented in a way that would minimise non-target exposure and vomiting. This was achieved through the use of an internal core. Field trials conducted in Queensland, New South Wales and South Australia have achieved $78 \pm 4\%$ (S.E.; n=9 sites) population or activity reduction using ground baiting, with little to no non-target take. Based on these results an Australian Pesticide and Veterinary Medicine Authority (APVMA) registration for PIGOUT[®] was granted in December, 2007.

The search for improved feral pig toxins has been ongoing for many decades. A recent 'Achilles Heel' review searched for physiological and biochemical weaknesses in feral pigs. One such weakness identified was the low levels of methaemoglobin reductase in pigs, an enzyme required to convert methaemoglobin back to its oxygen-carrying haemoglobin form. As such, pigs are particularly sensitive to methaemoglobin forming compounds, of which sodium nitrite is perhaps the most commonly available. Ironically, the best known use of sodium nitrite is to preserve pig meats, such as ham, bacon and smallgoods.

During 2006 the Invasive Animals CRC undertook pen trials to assess the lethality of sodium nitrite to feral pigs. Gavage and bait-delivered nitrite trials were both highly successful, with a rapid and humane death occurring in feral pigs in approximately two hours (compared to 6-8 hours with 1080) and with generally unremarkable symptoms. Symptomatology included lethargy, un-coordination, dyspnoea (laboured breathing), loss of consciousness and death. Half of the feral pigs tested vomited one to three times prior to unconsciousness. These results were recently presented to the APVMA,

who were encouraged by the findings and supported the development of sodium nitrite as an additional feral pig toxin.

The development of sodium nitrite as an additional feral pig active will increase the efficiency of feral pig control, as it is more lethal to feral pigs than 1080 and will also be available more readily due to its lower scheduling as a toxin; it will improve the humaneness of control, as time to death, the duration of symptoms and the symptoms themselves are greatly reduced; and will also reduce the non-target impact of control, as most other species are less sensitive to sodium nitrite poisoning, and it has an effective antidote in methylene blue. As such, the principal benefit of this project will be a greater ability of land managers to control feral pigs and in turn to reduced the impact of feral pigs on agriculture and the environment.

5. Objective

The scientific aim of this project was to assess the humaneness of sodium nitrite toxicosis for the lethal control of feral pigs. Pigs are particularly sensitive to methaemoglobin-forming compounds, of which sodium nitrite is perhaps the most commonly available. Therefore, baits containing sodium nitrite were fed to domesticated pigs, and the humaneness, progress of symptoms and physiology of death were assessed in a controlled environment.

6. Key Study Dates

Commencement of Experimental Phase of Study: 12th March 2009

Completion of Experimental Phase of Study: 24th March 2009

Proposed Completion of Final Report: 24th April 2009

7. Study Design

Adult pigs 30-50 Kg in mass were used, which represents the average weight of a feral pig population. Animals were fed nitrite-free baits for two to three days prior to the start of the study. All animals were anaesthetised and an arterial catheter was inserted for blood collection. The next day when the animals had recovered from surgery, baseline blood samples were taken (co-oximetry, clinical pathology). One or two HOG-GONE[®] baits with 20 g of micro-encapsulated sodium nitrite spread throughout the matrix were placed in the pens of the treatment animals. The controls received

the same amount of bait without the sodium nitrite. Animals were provided with water *ad libitum*. All animals were watched until they had eaten the bait.

The animals were closely monitored for clinical signs of distress, and physiological changes (respiration, haematology, biochemistry, cortisol and lactate and methaemoglobin levels) until death. Cortisol and lactate levels were assessed to monitor the internal stress response of the animals. Any animal still alive four hours post-consumption of bait would be humanely killed.

7.1 Clinical Assessment

The animals fed the non-toxic bait remained normal throughout with the exception of Pig #2, who was agitated, particularly when her neck was touched. It was thought that the suture attaching the catheter-holding bag to her neck may have been too tight.

All animals fed the toxic bait died. Four of the five died within 64 ± 13 min (SEM) of bait consumption, the remaining pig (pig number 8 which consumed the lowest dose) died within three hours of bait consumption.

8. Conclusion

The nitrite containing toxic baits fed to the pigs in this study were efficacious and resulted in an apparently humane death. Biochemical changes other than the rise in lactate and cortisol, were not different between test and control animals. The rise in blood lactate concentration was consistent with the level of methaemoglobinaemia. Skeletal muscle metabolism becomes anaerobic as oxygen debt develops because of the reduced oxygen carrying capacity of red blood cells containing methaemoglobin. Further studies would be required to determine which organs contribute most to the lactate load. Lactate at 25 mmol/L is not in itself associated with pain or discomfort, however. The rise in serum cortisol was significant compared to baseline values in the test animals, giving an 8-10-fold increase after consumption of bait. It is of interest that one of the controls had significantly elevated cortisol levels, and this was attributed to stress associated with soreness of the neck, exacerbated by blood collection.

It is the opinion of the authors that the development of methaemoglobinaemia as a result of sodium nitrite ingestion leads to a state of unconsciousness without a prolonged preliminary excitatory state. The behavioural evidence suggests that death from nitrite intoxication is an acceptable method of humane killing for large scale feral animal culling.

9. Acknowledgements

This study was funded by a grant from the Australian Department of the Environment, Water, Heritage and the Arts. Day 2 of the trial was observed by Dr Bidda Jones, Chief Scientist, Royal Society for the Prevention of Cruelty to Animals, Australia, who also had input into the trial design. Day 6 of the trial was observed by Mr Alan Learmonth from the IMVS Animal Ethics Committee. We thank Frank Gigliotti (General Dogs Body P/L) for taking the methaemoglobin measurements, and Dr Simon Humphrys (IACRC) for assisting in the Day 6 trials.

10. References

¹Australian Code of Practice for the Care and Use of Animals for Scientific Purposes, 7th edition, National Health and Medical Research Council, Australian Government, 2004
