Review of effectiveness, environmental impact, humaneness and feasibility of lethal methods for badger control

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1. Executive Summary

i) Defra have identified a potential requirement for the culling of badgers as part of the bovine TB control program. The aim of this desk study has been to collect and collate the relevant information on the methods that might be employed.

ii) The methods considered are fumigation of setts, poisoning, shooting and use of snares or traps followed by shooting. The humaneness, environmental impact, effectiveness and feasibility of each of these methods have been considered.

iii) The use of fumigants cannot be reliably expected to kill all the animals in a complex burrow system due to the difficulty of gases spreading through complex tunnel systems containing blind-ends. No information on the proportion of animals likely to suffer exposure to sub-lethal concentrations is available, but this risk is considered to be significant and needs to be assessed in combination with the consequences of sub-lethal exposure to toxins.

iv) Of the gases considered, it is concluded that carbon monoxide (CO) raises the fewest issues regarding humaneness and feasibility. It is a relatively humane fumigation candidate, but questions remain regarding the manner of its production and use. Very young animals of other species are known to exhibit tolerance to CO, which risks dependent young being left alive if the parents are killed, consequently a close season is recommended. It is concluded that diesel engines are not suitable for the production of CO as insufficient CO is generated to be widely applicable and irritant pollutants are present in the diesel exhaust gases. Models suggest that the use of an idling, badly-tuned petrol engine exhaust without catalytic converter could produce lethal concentrations of CO. Further investigation of how CO is generated by different petrol engines is required.

v) If the use of CO is pursued further, consideration should be given to better assess the risk of sub-lethal exposure arising from the anticipated distribution of CO in setts and whether pollutants in petrol engine exhaust gases have detrimental effects on animals prior to insensibility due to CO toxicosis. Currently CO is not registered as an approved vertebrate control agent in the UK and approval through the usual process would probably require at least one year to obtain and the production of supporting data would cost at least £250,000. An experimental permit, which would constrain the time and area over which a product could be used, could possibly be obtained within 1-6 months with a reduced requirement for supporting data.

vi) There are no currently available poisons that would be effective without causing deaths that would be considered markedly inhumane and/or significant risks to non-target wildlife.

vii) Given that shooting free-running badgers (i.e. those not first restrained) is likely to take only one animal at a time, this approach is more suited for use by those such as farmers or gamekeepers who are regularly patrolling the ground for other purposes. Shooting will be less effective during the winter when the badgers spend more time within their setts.

viii) Badgers are apparently not disturbed when spotlighted and can be shot with a rifle. The maximum effective distance of rifles would be around 150m, with a range of approximately 100m being preferred. Few farmers are likely to own a rifle suitable for killing badgers, this requiring a calibre more normally used by deer stalkers or some gamekeepers. It is unlikely that a shotgun will be effective at killing a badger beyond 30-40 m. Both the effectiveness and potential for wounding will depend on the training and competence of the shooter.

ix) From the limited available information it appears that, when correctly set for badgers by personnel competent to do the work, and frequently checked, both body and padded foot snares cause few injuries. Setting a body snare for a badger is different from the placement of a neck snare for a fox and, therefore, specific competences would be required in order to minimise the risk of compromising the welfare of the trapped badgers or non-target captures. Shooting restrained badgers could be carried out using firearms and ammunition that comply with the Protection of Badgers Act (1992).

x) A variety of potential body snare designs are available for consideration and trials are needed to identify the best designs. No suitable foot snare is currently approved in the UK although assessments are underway. The humaneness of snares used to restrain badgers will need to be assessed as required by the Agreement on International Humane Trapping Standards, as incorporated into the draft EU Humane Trapping Standards Directive (COM (2004) 532).

xi) Cage traps cause few injuries to badgers if frequently checked. Cage traps are more cumbersome to transport and manoeuvre on site than snares. Pre-baiting before setting traps maximises the number of individuals caught on the first trap night but necessarily delays the control operation. The average reported capture rate of snares is twice that of cage traps.

xii) To despatch badgers in cage traps and meet the provisions of the Protection of Badgers Act (1992), a rifle would have to be used and this would pose significant risks to the operator from ricocheting bullets. The use of a shotgun of 20-bore or larger presents similar risks. It thus may not be possible for non-Crown employees to obtain a suitable firearm/ammunition combination to despatch trapped badgers humanely and safely.

xiii) Based on this review it is considered that if a cull of badgers is required, the following methods of killing badgers have potential for further consideration: a) the use of CO to fumigate setts, b) shooting of free-running badgers and c) restraint followed by shooting. However, for each of these approaches there are potential risks and gaps in our existing knowledge relating to their humaneness, effectiveness or impacts on other species.

2. Introduction

Defra have identified a potential requirement for the culling of badgers (*Meles meles L.)* as part of the bovine TB control program. There are several methods that could be employed, however to date no objective assessment has been made of the issues associated with each. The aim of this desk study has been to collect and collate the relevant information currently available that is relevant to this topic.

The methods considered have been:

i) Fumigation of setts,

ii) Poisoning,

iii) Shooting free-running badgers,

iv) Use of snares or traps followed by shooting.

This information has been gathered in order to consider the following aspects of each of these methods:

a) Humaneness,

b) Environmental impact, including non-target species,

c) Effectiveness and feasibility.

In relation to humaneness, there is little information on the reactions of badgers to toxic substances and hence this review depends heavily on data derived from other species. In particular data from the reactions of humans to the compounds have been considered wherever possible in the humaneness assessments. The Littlewood Report (Littlewood, 1965) recommended that procedures (or conditions) which cause pain or distress in humans should be assumed to do so in animals until convincing evidence is available to the contrary; an approach which has received support from a number of sources e.g. Bateson (1992; 1991); Spinelli (1991); Zimmerman(1983). We have also considered the issue of restricting, on welfare grounds, culling during the period when lactating sows might have dependent offspring, and which risks removing the mother while leaving the offspring.

The possible environmental impact of each potential culling method has been reviewed, particularly with respect to non-target wildlife, but no consideration has been given to the ecological consequences of large-scale badger culling.

In relation to efficiency, the review has considered issues reflecting the likely number of animals culled for a given unit of effort while effectiveness has been considered in terms of the proportion of the target population that could reasonably be culled. The feasibility of each approach reflects the practicality of being able to carry out a particular control method, including, for example, the complexity of the equipment, whether good access to sites/setts is necessary, the availability of personnel and resources, legal considerations (although these are dealt in more detail by EWD (2005)), health and safety and training issues. With all methods reviewed it should be borne in mind that repeat applications can potentially increase effectiveness.

General conclusions have been drawn with respect to methods that would not be applicable and recommendations made regarding the key gaps in our knowledge with respect to methods that cannot be ruled out on grounds of humaneness, environmental impact and effectiveness or feasibility.

3. Culling restrictions during the period when sows are lactating

The concept of having a “close” or “closed” season on culling evolved originally for species that are exploited for food (e.g. fisheries) or sport (e.g. game birds with seasons defined by the Game Act (1831)) with the aim of preventing overexploitation that might threaten population viability. The definition of such a season is often, at least partly, aimed at maximising juvenile recruitment and thus loosely linked to the breeding season of the species concerned. However, more recently the concept has been extended to address the welfare issue raised by killing mothers with wholly dependent offspring that will consequently die of starvation or dehydration. This is the principle underlying the cessation of culling between 1st February and 30th April during the current Randomised Badger Culling Trial (RBCT) (Bourne et al., 1998). A more extensive restricted period, from the end of November to the end of June, is employed by English Nature and Defra, when issuing licences under the Protection of Badgers Act (1992) for activities that may damage a sett, disturb the resident badgers or destroy them, in order to protect pregnant sows against potential relocation. The available data on pregnancy, gestation length and weaning for badgers in Britain were reviewed by Woodroffe et al (2005a) and they concluded that all births would occur after mid-January and almost all cubs would be weaned by mid-May. This formed the basis of the no culling period between February and April inclusive during the RBCT. However, the available data suggested that up to 20% of births and 8% of weaning events might occur outside this period. The extent to which this potential is realized has been evaluated by Woodroffe et al (2005a) with a total annual estimate of nine dependent cubs being orphaned by the RBCT culling operations annually between May 1999 and May 2003. This is considerably lower than the prediction made by the National Federation of Badger Groups (NFBG, 1999) that, in the first phase of killing in the proactive areas alone, more than 2,300 cubs would orphaned and starve to death underground. Kirkwood (2000) reports on seeing a lactating sow trapped on 17th May 2000 during his audit of the humaneness of despatch procedures used in the RBCT. He thus recommended that the timing and duration of the period during which culling was suspended should be kept under close review in the light of data on the dates of capture of lactating sows. Based on the currently available data attaining complete prevention of nursing sows being killed would apparently require restriction of culling from mid-January through to mid-May. However, Woodroffe et al (2005a) contend that this would compromise the speedy and effective implementation of badger culling as a candidate TB control policy, with only a modest benefit accruing in terms of the small number of cubs affected.

In previous badger culling strategies involving live-capture the issue was putatively addressed by releasing captured lactating females (Krebs, 1997). However, it is difficult to reliably recognise lactating females in the field (4.3% of females culled in badger control operations in south-west England during 1996 to 1998 were found at *post mortem* to be lactating despite a policy of releasing any captured sows considered to be lactating, Defra unpublished data reported by Woodroffe et al. (2005a)).

If the method of culling offered certainty that any dependent cubs would be killed along with their mother, then there would be no need to cease culling on welfare grounds during the period when sows might be lactating. This could be the case with sett-based control using fumigants. However, given the complex nature of badger setts there is a risk, examined in more detail later in this report, that gassing techniques may not reliably kill all occupants of a sett at a given time. This uncertainty is increased by the possibility that young animals may be less susceptible to such fumigants than adults. For instance, neonatal young are particularly tolerant of high CO_2 levels (Van Zutphen, 1993). Furthermore, neonates have greater tolerance of carbon monoxide exposure than adults (Winston & Roberts, 1978) and fox cubs have been observed to survive CO fumigation although the mother has been killed (Hart et al., 1996a). This high tolerance in juveniles may reflect the same adaptations in respiratory or vascular function that give rise to CO_2 tolerance. Although the duration of this adaptation is unknown, fumigation of fox earths is not recommended in Australia if young less than 4 weeks old might be present. Based on this experience with other species, but in the absence of comparable information on badgers and on the feasibility of obtaining high gas concentrations throughout setts, a close season is therefore recommended on welfare grounds .

4. Fumigation of setts

4.1 Generic issues

When fumigating burrows, gases are either pumped through the tunnel system, or they are produced from tablets, pellets, powders or cartridges and then diffuse through the tunnels. Soil moisture is necessary to generate some gases. Diffusion fumigation, which requires less equipment than power gassing, is most effective in non-porous soils, such as those that are compacted or wet, rather than those of dry sand or cracked clay. However, diffusion is unlikely to result in gas being evenly distributed throughout a burrow system, particularly if the gas is heavier than air. All fumigation chemicals by their very nature are potentially lethal to personnel carrying out the procedure. COSHH assessment and operating procedures to ensure safety of personnel would need to be written and adhered to. As a minimum there should be at least two trained people present during each fumigation attempt and Personal Protective Equipment (PPE) would need to be provided as appropriate. Training courses, such as those designed for pest controllers, are available from many companies and once detailed Standard Operating Procedures (SOPs) have been devised, these courses could be adapted to cover the fumigation of badger setts.

The success of fumigation depends critically on the ability to achieve a lethal concentration of the chosen fumigant throughout the badger sett. This in turn will vary with factors such as the diffusion characteristics of the gas, the rate of input of the gas into the sett, the topography and volume of the sett, and the nature of the substrate (the soil surrounding a sett has a major impact on the spread of some gases within the sett system, (Fuhr et al., 1948)). In addition, badger behaviour itself could influence their exposure to the gas. If gas is detectable by the badgers and found to be aversive, this could cause the animals to move deeper into the sett where the gas is less likely to penetrate in high concentrations, which could increase the chance of sub-lethal exposure. On the other hand, movement of badgers could aid dispersal of the gas to areas otherwise unaffected, although no data are currently available to quantify this.

Badger setts can be large and complicated with many entrances and interconnected tunnels, as well as a number of blind ending tunnels and nesting chambers. In addition, some setts initially identified as large may in fact be composed of several separate setts with no interconnecting tunnels. An added complexity in estimating the size of a badger sett is that they are often found in woods or dense undergrowth, which makes it difficult to detect all entrances.

There is little information on the detailed topography of setts. Table 1 summarises data from five setts where this information has been obtained by excavation. The volumes of these setts range from 0.7 to 25.2 m^3. However, one of these setts was only partially excavated and the estimated total volume of this sett was 38.7m^3 (Roper et al., 1991). Although no firm conclusions can be reached on the basis of this small sample, it is interesting to note that the greatest median distance from an entrance, i.e. where a fumigant would enter the tunnel system, to a blind end in a tunnel, i.e. where an animal could take refuge from the incoming gas, is 6.25m (range 0.75m to 12.75m). This distance is important when calculating the gas concentrations to which animals in a sett are likely to be exposed. However, in another limited description of a small badger sett, the ends of the only two blind tunnels were on average 13m from the entrance (Cowlin, 1967). The proportion of blind tunnels differs per sett, but especially in the larger setts can be around 20%. In the largest sett investigated here 24% of the total tunnel length were blind tunnels (Leeson & Mills, 1977a; Leeson & Mills, 1977b), and up to 37% of these were over 2 meters long. It could be expected that badgers may retreat to chamber areas during fumigation. Of all the chambers found to contain bedding material, up to 28% were situated in blind tunnels. Sloped tunnels could either enhance or worsen gas dispersal, but Roper et al (1991) reported that their excavated setts went mainly horizontally into a hillside. Tunnels are generally around 30 cm diameter (Roper et al., 1991; Leeson & Mills, 1977b; Leeson & Mills, 1977a).

Table 1. The topographical characteristics of five excavated badger setts.

		Sett 1[1]	Sett 2[1]	Sett 3[1]	Sett 4[2]	Sett 5[3]
Distance from blind end to entrance, m	Median	3.25	3.25	2.00	3.63	6.25
	Range	3.25	0.75-5.50	0.75-6.50	0.75-6.50	0.75-12.75
Total No. of blind ends		1	13	39	44	37
Total No. of entrances		5	26	80	38	15
No. Closed entrances		2	10	30	22	3
No. Open entrances		3	16	50	16	12
Ratio of blind ends to entrances		0.2	0.5	0.5	1.2	2.5
Total length of tunnels, m		16	140	354	360	310
Proportion of length of blind to open tunnels		0.05	0.15	0.20	0.19	0.24
Proportion of blind tunnels over 2 m		0	0.15	0.28	0.34	0.37
Total volume of sett m^3		0.7	6.0	14.7*	25.2	15.5
Total no. of chambers with bedding		1	8	12	17	22
No. of chambers with bedding in a blind tunnel		0	2	3	2	8

*Volume of partial excavation of large sett, estimated total volume of this sett was 38.7m^3
[1]Roper et al (1991)
[2]Leeson & Mills (1977) Unpublished report on a survey of a badger sett at Manor Farm, Alverston, Avon
[3]Leeson & Mills (1977) Unpublished report on a survey of a badger sett at Mumbleys, Thornbury, Avon

The humaneness of gassing is dependent on three factors: i) the effects of exposure to a lethal concentration of the gas, ii) the risk of animals only being exposed to sub-lethal concentrations of a gas, and iii) the consequences of such sub-lethal exposure. Due to the complexity of badger setts it is unlikely that a lethal concentration of any of the agents discussed below would always occur throughout the whole of a sett, which is also suggested from theoretical models (below) and experimental data on burrow fumigation on other animals (Ross et al., 1998; Hart et al., 1996b).

To gain more insight into the dispersal of gasses throughout setts, Defra commissioned a modelling study of the dispersal of carbon monoxide using computational fluid dynamics (CFD) in a series of simplified 2D and 3D models (Defra, 2005). CFD is a sophisticated modelling tool capable of modelling detailed aspects of gas and fluid flows, and allows investigation of a variety of effects under controlled conditions not easily obtainable in the field. Modelling CO dispersal, using four different types of generators, in simplified tunnel structures using a number of assumptions, confirmed that it was very difficult to obtain a lethal concentration of 1% for a sustained length of time in blind tunnels. Obtaining this concentration in open tunnels was achievable for two of the methods. It also showed that keeping entrances open (other than the entrance the gas is introduced into) is beneficial for gas dispersal throughout the sett. Dispersal of CO in a model of a more complex sett based on Roper et al (1991) will investigate these findings further and a report on these results will be available at a later date. The currently available results from the simplified models will be described in more detail in the carbon monoxide section. Because of these factors influencing the ability to achieve lethal concentrations throughout the set, it is necessary to consider the effects of non-lethal concentrations on the animals, both in terms of short-term, acute distress and long-term welfare consequences for the survivors. Laboratory experiments assessing the welfare implications of toxic gases have typically involved immersing animals into a known concentration of the compound (e.g. Hansen et al., 1991). For some gases, like carbon monoxide, adverse effects are found when animals are suddenly immersed in a high concentration of the gas, but not found if the gas concentration is slowly increased to the maximum value (Lambooy & Spanjaard, 1980).

Control is likely to be most cost-effective if several animals or an entire social group can be killed with a single application, but this may be difficult to achieve consistently if some setts are exceptionally large, located in inaccessible areas, or sett entrances are difficult to find. It may also be impracticable or unsafe to fumigate during wet or windy weather and some individuals normally resident in the sett may be absent at the time operations commence. Experience from control operations against other burrow-living species shows that the efficacy of fumigation is often unpredictable and, on average, unlikely to achieve more than a 80% reduction of the resident population (e.g. Ross et al 1998). Fumigation at more than one entrance could improve the dispersal of gas through open tunnels, but is unlikely to disperse the gas significantly further into the blind tunnels. Repeat application can improve ultimate fumigation success, although this approach will not necessarily lead to 100% control.

During previous control operations of badgers using cyanide gas repeated fumigation was often considered necessary by personnel carrying out the work.

Minimal environmental impacts are likely to result from the fumigation of setts using the agents discussed below. The gases disperse over time and do not persist in the bodies of killed animals and thus pose no secondary poisoning hazard to predators eating animals killed by this method. Furthermore, as control would be restricted to the fumigation of active badger setts there is a relatively low risk that non-target species would also be present, although there is the possibility that setts may sometimes contain other species. Species that have been reported to share badger setts include woodmouse (*Apodemus sylvaticus*), bank vole (*Clethryonomys glareolus*), Norway rat (*Rattus norvegicus*), rabbit (*Oryctolagus cuniculus*), red fox (*Vulpes vulpes*), pine marten (*Martes martes*), polecat (*Mustela putorius*), weasel (*Mustela nivalis*), feral cat (*Felis cattus*) and wild cat (*Felis sylvestris*) (Neal & Cheeseman 1996). This list is likely to be incomplete. The two species most commonly associated with setts are rabbits and foxes. Rabbits will often be found associated with large setts, usually occupying side burrows of smaller diameter around the perimeter. Foxes are also known to be regular permanent residents of badger setts, living alongside badgers but perhaps occupying a different part of a given underground system. The main species of conservation concern that might be found inside setts would be other members of the mustelid family such as polecats and otters. These two species have both re-colonised parts of their original range in recent years and have sometimes been found in badger setts. Birks and Kichener (1999) and McDonald and Harris (2000) have pointed out the threat to those polecats using rabbit burrows from fumigation targeted at resident rabbits. The extent to which fumigation of badgers setts would pose a similar risk to this species is unknown but would be expected to be less than fumigation of rabbit burrows.

4.2 Phosphine

4.2.1 Humaneness

Phosphine gas is a potent inhibitor of cytochrome oxidase in the respiratory metabolism pathway, which means that those organs with high oxygen requirements are particularly sensitive to damage. Humans inhaling phosphine show symptoms including coughing, choking, breathlessness, nausea, vomiting, severe lung and abdominal pain, severe headache, intense thirst, ataxia, intention tremors, and convulsions before coma (Meehan, 1984; PSD, 1997). Phosphine poisoned animals show similar signs of respiratory irritation, ataxia, convulsions, abdominal pain and other discomfort (CSL, 1991; Meehan, 1984). Animals should be assumed to be sensible during convulsions if they are sensible both immediately before and after the convulsion (CSL, 1993a). Animal studies of phosphine poisoning, involving cats, guinea-pigs, rodents, moles and rabbits (e.g. Klimmer, 1969), have found that animals do not appear to show symptoms until 30 minutes after exposure and, depending on the species, die within 2 to 10 hours (PSD, 1997); phosphine poisoning can therefore cause severe suffering. The higher the concentration of phosphine the shorter is the time to death, but the signs are progressive and cannot easily be equated with distinct concentrations. Concentrations of phosphine above 5 ppm produce accumulative toxicity whilst at concentrations of below 2.5 ppm there is no evidence of accumulation in most species (the mole is the exception being considerably more sensitive to phosphine than other species (PSD, 1997)). The toxicity of phosphine to badgers is unknown. The ability to metabolise phosphine means that animals which receive a sub-lethal dose and move into a phosphine-free environment should recover and show no signs of lasting harm (PSD, 1997). However, discontinuous exposure to phosphine can result in very large increases in times to death and in duration of symptoms (CSL, 1993c). Symptoms are likely to be similar to those of zinc phosphide poisoning, and therefore death can be considered as inhumane. In comparison with other methods, phosphine fumigation is considered to be less humane for rabbit control than hydrogen cyanide fumigation but more humane for rodent control than anticoagulant poisons (PSD, 1997).

4.2.2 Environmental impact

Minimal environmental impacts result from fumigation with phosphine. The gas disperses over time and poses no secondary poisoning hazard to predators eating animals killed by this method. As control would be restricted to the fumigation of active badger setts there is a low risk to non-target species. The Health and Safety Executive (HSE) short-term exposure limit (stel) for PH_3 is only 0.3 ppm; thus fumigation with this compound is more hazardous to operators than fumigation with CO or HCN. Similar precautions to those that are already in place for use of phosphine to gas rabbits would be required. These are wearing protective clothing and gloves at all times, and using suitable protective respiratory equipment when opening the container and handling the tablets. In addition fumigation should not be carried out in wet weather (including heavy mists) or strong winds.

4.2.3 Effectiveness & feasibility

In 1979 clearance was given for the use of products containing aluminium phosphide, which generate phosphine on contact with moisture, for the control of rabbits, rats and moles. Two formulations are currently approved; Phostoxin (30 x 3g) tablets and Talunex (160 x 0.6g) pellets. Each tablet or pellet releases about one third of its weight as phosphine gas. Both are subject to the Poisons Rules and to the Carriage of Dangerous Goods Regulations. With regard to the latter, suppliers may now employ specialist carriers to deliver products at additional cost. Efficacy is likely to vary in relation to factors that can be controlled, such as the placement of tablets or pellets only in active burrows, and those that cannot, such as soil porosity, moisture content and temperature. On average 62% of resident moles are killed by this method (range 43 to 100) according to MAFF (1981) although Lund (1974) reports only 48% (range 0-100). Experience from fumigating the burrows of animals other than badgers, such as moles and rabbits, shows that prior planning and preparation invariably enhances efficacy. In particular, visits to determine the frequency with which burrows are used and surveying the area to find all possible tunnel entrances can prevent much waste of time and materials.

4.3 Hydrogen cyanide

4.3.1 Humaneness

Cyanides are rapidly absorbed and are among the most rapidly acting of mammalian poisons (Egekeze & Oehme, 1980) and can be administered via fumigation or in poisoned bait. Cyanide is a centrally acting chemical that inhibits cytochrome oxidase, an essential link in the chain of mitochondrial respiration, thereby preventing the cellular use of oxygen and causing cytotoxic anoxia. Gross symptoms include suppression of central nervous system (CNS) activity, rapidly leading to respiratory suppression, cardiac arrest, coma and death (Gregory et al., 1998). Low doses of cyanide have caused a number of symptoms in humans including convulsions and anxiety but never pain (Meehan, 1984; PSD, 1997). Higher doses will kill within minutes with symptoms of respiratory and cardiac stimulation before unconsciousness, convulsions and death (PSD, 1997). The convulsions that may be seen during cyanide poisoning are not thought to be distressing, because they occur after the start of progressive loss of reactivity to external stimuli and because they are of relatively short duration (Gregory et al., 1998). Time to death from HCN poisoning is a function of dosage. At doses above 400 ppm rabbits die within 30s after the first abnormal symptoms are observed (PSD, 1997). Sub-lethal doses of HCN may cause long-term consequences. In particular damage to the central dopaminergic systems manifests itself as Parkinsonism (Schmidt et al., 1978). Animals lethally or sub-lethally poisoned by cyanides have exhibited optic nerve and retinal damage. Repeated administration of cyanide caused central nervous system lesions involving degeneration of the myelin sheath in rats and monkeys (Grant, 1986). However, damage is not inevitable and animals and humans have recovered completely from sub-lethal cyanide exposure (e.g. Gregory et al., 1998). “On the basis of rapid death (seconds to minutes) after onset of symptoms, and possible onset of insensibility before death, efficacious concentrations of hydrogen cyanide are considered to be relatively humane”(PSD, 1997).

Between 1975 and 1982 badger setts were fumigated with HCN as part of the bovine tuberculosis campaign. In 1980 Lord Zuckerman’s review of the role of badgers in the spread of bovine tuberculosis recommended that gassing techniques should be investigated which resulted in the humaneness of this approach being questioned. It was because of these concerns regarding humaneness that gassing with HCN was replaced by cage trapping in 1982. As part of this investigation a small-scale toxicity trial involving four badgers was conducted and this experiment provides the only information of the effects of HCN on badgers. One animal was exposed for 30 minutes to a gas concentration of 75 ppm. It began to show signs of gasping, vomiting and intoxication after 6 minutes and collapsed unconscious after 23 minutes. Upon removal from the gas it recovered consciousness after three hours and made a full recovery. Another animal exposed for 30 minutes to a concentration of 165 ppm showed no abnormal signs until 27 minutes after the beginning of the exposure when vomiting, gasping and staggering began. It was semi-conscious when removed from the gas and also made a full recovery. A third badger exposed to a concentration of

277 ppm showed no symptoms until it died after 21 minutes. The final animal was exposed to a concentration of 297 ppm. It showed no symptoms until it collapsed and stopped breathing after 12 minutes. The flow of gas was stopped at this time and the animal spontaneously started breathing again 2 minutes later. This animal also recovered. There were, therefore, no signs of convulsions and the three animals that did not die, subsequently recovered.

4.3.2 Environmental impact

Hydrogen cyanide is quickly absorbed by moist soil. In an experiment examining this property of poisonous gases, only 14% of the introduced hydrogen cyanide remained in a test chamber containing soil after 1 hour (Fuhr et al., 1948). In soil with pH <9.2 hydrogen cyanide is expected to be highly mobile, and in cases where cyanide levels are toxic to micro-organisms (i.e., landfills, spills), it may leach into groundwater. In sub-surface soil, cyanide present at low concentrations would probably biodegrade (Hall & Rumuck, 1986). The HSE stel for hydrogen cyanide is 4.7 ppm. COSHH assessments for HCN use during rabbit fumigation recommended use of protective clothing, gloves and respirator. The respiratory mask should be worn when loading or removing containers from the pump, but then removed to ensure communication between personnel. Aquatic species are especially vulnerable to HCN poisoning and therefore use in warrens constructed in limestone or sandstone areas was advised against. Gassing should not be carried out in rain or swirling blustery wind conditions. In addition only 10% of people can smell cyanide gas and it is recommended that if operators cannot smell the gas they should not be involved in the fumigation operation.

4.3.3 Effectiveness & feasibility

The most common use of HCN, produced from the rapid hydrolysis of sodium cyanide, was the gassing of rabbits in burrows. On average 78% (range 43 to 100) of resident rabbits were killed by this method (Ross et al., 1998). Sodium cyanide was commercially sold for use against rabbits in a powdered form that was either spooned into burrow entrances or forced through via a mechanical powder pump. Limited trials were carried out by MAFF in 1975 to assess the distribution of hydrogen cyanide gas in badger setts following applications of powder with a pump. The results indicated a variable concentration of gas reflecting the uneven distribution of powder, suggesting that cyanide did not readily disperse through the tunnels. Sodium cyanide is no longer approved under Control of Pesticides Regulations (1986; COPR). The company marketing the product could not source the chemical and therefore did not support it through Stage 4 of the EU review of chemical products. Sodium cyanide is still manufactured in India (e.g. Spectrum Chemicals, Mumbai) where it is currently used as a fumigation insecticide on cotton prior to export.

4.4 Carbon dioxide, with and without argon

A medical definition of some terms used in the literature relating to carbon dioxide and oxygen levels in the blood is provided below for clarity (Stedman, 1995).
Asphyxia is the impairment of ventilatory exchange of oxygen and carbon dioxide, i.e. combined hypercapnia and anoxia.
Anoxia is the absence, or almost complete absence, of oxygen from arterial blood and tissues.
Hypercapnia is an abnormally increased arterial carbon dioxide concentration.

4.4.1 Humaneness

Carbon dioxide is an asphyxiant and the most powerful cerebral vasodilator known. Carbon dioxide initially stimulates respiration and then causes respiratory depression resulting in death. Concentrations over 10% are known to cause suffocation and death in humans (Anonymous, 2004). Although high concentrations of the gas do result in oxygen-deficient environments, the hypercapnia effects are thought to cause death before oxygen-deficiency is a factor. At concentrations above 60%, CO_2 can be used as an anaesthetic agent that causes rapid loss of consciousness (Green 1987). Moreover CO_2 is the most commonly used method for euthanasia of laboratory animals (Conlee et al., 2005) at concentrations greater than 40%, although concerns over its humaneness have recently been acknowledged (e.g. HSUS, 2004). Studies have now shown that rats find exposure to CO_2 very aversive (Leach et al., 2001) even at concentrations as low as 25%. These results support the findings in other animals that exhibit excitement, gasping and escape responses during exposure to lethal concentrations of CO_2 (e.g. pigs and cats: Raj & Gregory, 1995; 1996; pigs: Lambooy, 1990). Lower concentrations of CO_2 take longer to kill animals, rats in a pest control situation can take up to 24 hours to die (Meehan, 1984). For humans, inhalation of air containing CO_2 concentrations over 50% are described as unpleasant whereas concentrations over 80% are more likely to be described as painful (Danneman et al., 1997). Behavioural tests on mink (a mustelid as are badgers) showed they too find CO_2 highly aversive (Cooper et al., 1998). When mink were trained to enter a chamber to investigate a novel object, they would do so rapidly when the chamber contained air, but would not do so at all when the chamber contained 80% CO_2. They also rapidly recoiled, and coughed and sneezed, upon inhaling the gas. Furthermore, a report of an EC Working Party on laboratory animals does not recommend its use for any adult carnivore because of the behavioural distress it causes (Close et al., 1996).

Anoxia followed by death occurs when inert gases displace oxygen in the air to a level that can no longer support life. The typical oxygen content of air is 20.2%. In humans, air containing less than 20% can lead to physiological effects as detailed in Table 2. In badger setts it is not unusual to find oxygen levels as low as 19.5% in occupied chambers (Roper & Kemenes, 1997), therefore suggesting that badgers are relatively tolerant of low levels of oxygen.

Table 2. Symptoms of exposure to low levels of O_2 in humans

Oxygen Content (vol. %)	**Inert gas content (vol. %)**	**Effects and symptoms (at atmospheric pressure)**
20 – 14	0 - 30	Diminution of physical and intellectual performance without person's knowledge.
14 - 10	30 - 50	Judgement becomes faulty. Severe injuries may cause no pain. Ill temper easily aroused. Rapid fatigue on exertion. 11% oxygen, risk of death.
10 – 6	50 – 70	Nausea and vomiting may appear. Loss of ability to move vigorously or at all. Inability to walk, stand or crawl is often first warning and it comes too late. Person may realise they are dying but does not care. Resuscitation possible if carried out immediately.
0 – 6	70 - 100	Fainting almost immediate, painless death ensues, brain damage even if rescued.

Source: Compressed Gas Association (2001)

Raj and Mason (1999) have shown that mink can detect anoxia induced experimentally by means of argon, and that they find it aversive. The mink's responses to anoxia induced by argon differed from those to CO_2. They would enter an anoxic chamber containing a novel object (i.e. the gas appeared to be initially undetectable, or at least not inherently aversive) but then leave the chamber, panting, after a very short time. This did, however, not deter them from repeatedly returning to the enclosure containing argon, even though they would always leave it again after a few seconds. Thus mink could detect argon-induced hypoxia, and would act to rectify it given the opportunity to leave the chamber. Rats show signs of panic and distress before unconsciousness when in an atmosphere containing 39% nitrogen or argon (Andrews et al., 1993). Mink and rats thus differ from pigs, poultry and humans, who do not find anoxia detectable or aversive(Raj, 1999; Raj & Gregory, 1995).

Argon/CO_2 60/30% mix is suggested as the most appropriate gas for stunning pigs in the EU (Scientific Panel on Animal Health and Welfare, 2004). However, even at this concentration, exposure for 7 minutes is required to ensure all pigs are dead (Raj, 1999), the duration required for lower concentrations to achieve death are unknown. The responses of badgers to CO_2, argon, or argon/CO_2 mixes are also unknown.

Fumigation of setts is likely to expose animals to slowly rising levels of the fumigant rather than abrupt change in concentrations as investigated in all the above studies. Some studies have looked at gradual induction using CO_2. Britt (1987) specifically compared pre-filling and gradual induction methods of euthanasia using rats and mice. He noted that even though time to collapse was shorter with rapid induction, there were more signs of distress with that method. The abnormal behaviors included: shaking (frequent), moving in reverse, tail thrashing (uncommon) and increase in frequency of urination and defecation. Behavioral responses varied between species and individuals.

Rabbits, similar to badgers in being a fossorial (tunnel dwelling) mammal, appear to be particularly resistant to anoxia. When made to breathe 9 % oxygen in nitrogen (i.e. 55% inert gas) no significant effects were observed on respiratory or heart rates, and the rabbits were alert and apparently unstressed after 3 h of exposure (Hayward & Lisson, 1978). Similar adaptation to CO_2 exposure has been found in several fossorial mammals (e.g. kangaroo rats: Soholt et al., 1973; pocket gophers: Darden, 1972). It is thought that these mammals possess an increased buffering capacity against respiratory acidosis (Chapman & Bennett, 1975). Carbon dioxide levels higher than ambient have also been recorded in badger setts (Roper & Kemenes, 1997), suggesting that badgers may also possess this adaptation to burrow living. Sub-lethal treatments were seen in a burrow fumigation trial where insufficient amounts of dry ice failed to reach the maximum concentration of 45-50% CO_2 for at least one hour (Hayward & Lisson, 1978). Evaluation of survival time in a range of carbon dioxide concentrations from 30 to 60% has not been carried out for any other species apart from rabbits. However, the survival time for 50% of mice at a concentration of 42.35% CO_2 was 3h 50min (Stupfel et al., 1971). It is unknown whether badgers are more or less tolerant of CO_2 than these two species. Neonates have also been found to be particularly tolerant of CO_2 (Van Zutphen, 1993) depending on maturity at birth (those that are born more mature are less tolerant of CO_2).

The long-term effects of sub-lethal concentrations of CO_2 or inert gases are dependent on the degree of anoxia. At low to moderate levels full recovery is normal, however if severe anoxia is experienced permanent brain damage could occur (Compressed Gas

Association, 2001). Klemm (1964) examined whether 15 minutes of sustained CO_2 anesthesia can be used in cats without causing brain damage, no brain damage occurred as a result of frequent exposure to CO_2.

4.4.2 Environmental impact

Minimal environmental impacts would result from fumigation with CO_2, argon, or a CO_2/argon mixture. The gases disperse over time and pose no secondary poisoning hazard to predators eating animals killed by this method. The liquid or solid form of CO_2 is not hazardous to personnel and as fumigation will take place outside there is no risk of inhaling a lethal concentration of the gas.

4.4.3 Effectiveness & feasibility

CO_2 without argon is currently approved (under COPR) as a pesticide product in the form of a lethal trap for the control of mice. The gas is released from a small cartridge when a mouse presses on a treadle to close gas-tight doors. CO_2 is also approved (until 31 December 2008) as a ‘commodity’ substance (i.e. a non-formulated technical substance) for use as a pesticide in the following circumstances: 1) as an insecticide, acaricide and rodenticide in food storage practice, 2) as a rodenticide to kill trapped rodents, and 3) to despatch birds covered by general licences issued by the Agriculture and Environment Departments (under Section 16(1) of the Wildlife and Countryside Act (1981)) where the birds have been either trapped or stupefied with alphachloralose/seconal baits.

The estimates of the volumes of badger setts vary from 0.7 to 40 m^3. (Table 1). For a 30%/60% mix of CO_2/argon, up to 12 m^3 of CO_2 and 24 m^3 of argon will be required, assuming that no gas escapes the sett. The largest readily available pressurised CO_2 cylinder weighs 40 kg and produces 18 m^3 of gas at atmospheric pressure, and the largest argon cylinder weighs approximately 80 kg and produces 5.3 m^3 of gas. Therefore it would require one CO_2 carbon dioxide cylinder and five argon cylinders, with a combined weight of 440 kg, to treat a large sett. This is a minimum as it is likely that large volumes of gas will be lost due to dispersal in the soil or will not penetrate evenly throughout the sett (see Defra, 2005). It will also not be possible to predict the volume of gas required for a particular sett as there is no method to predict burrow volume from external characteristics. As many badger setts are relatively inaccessible, being found in woodland or on hillsides, the transport and use of an uncertain number of large and heavy gas cylinders is not a practical proposition for many circumstances. CO_2 being heavier than air, will pool in the lowest parts of burrow systems, thus leading to an inadequate distribution of the gas along the tunnels (Hayward & Lisson, 1978). The combination of inadequate distribution of the gas and adaptation to increased CO_2 for rabbits led Hayward & Lisson (1978) to conclude that CO_2 was likely to be ineffective as a fumigation agent in Australia.

4.5 Carbon Monoxide

4.5.1 Humaneness

The toxic action of carbon monoxide (CO) is due to competition with oxygen for binding sites on haemoglobin leading to tissue hypoxia (Ginsberg, 1985). CO has a 250 times greater affinity than oxygen to the binding sites and forms carboxyhaemoglobin (COHb) (Roughton & Darling, 1944). COHb is fully dissociable with active respiration: in air the half-life of COHb is 320 minutes (Klaasen, 1985).

Stewart (Stewart, 1976) gives details of the equilibrium levels of COHb at concentrations of CO from 1–90000 ppm and the effects in man associated with increasing % of COHb. From these data it can be deduced that exposure to 0.1% (i.e. 1000 ppm) can, after as little as 40 minutes, produce severe headache and nausea, and after 300 minutes result in coma followed by convulsions. Exposure to concentrations of 0.3% or more will result in death but there is no indication of the time required. However Tietz (1976) reports that CO concentrations of 0.4% or above are fatal in less than 1 hour. Stewart (1976) notes that exposure to CO concentrations greater than 1% can result in loss of consciousness without the symptoms of headache, nausea and vomiting; i.e. at higher concentrations loss of consciousness can occur before the onset of unpleasant effects.

CO has been used for animal, in particular dog, euthanasia for many years. In a review of animal euthanasia Green (Green, 1987) reported that CO concentrations between 0.5 and 14% have been used and that the animals are unconscious before showing signs of stress. Dogs exposed to 0.25% collapse within 10 to 15 minutes with no signs of distress (Burrell et al., 1914) although higher concentrations (i.e. 2 to 8%)

are generally used for the euthanasia of dogs. After exposure to concentrations above 2% dogs show sudden prostration followed by myoclonic contractions with vomiting, urination and defaecation (De Vries et al., 1977). However, the EEG from these animals indicates that they were in a comatose state from the first behavioural signs of intoxication, i.e. from the initial falling over, and the dogs showed no signs of suffering before they became unconscious. Similar observations were made by Moreland (1974) on dogs exposed to 1.9-6.3% CO, as pure CO or as exhaust gas. Mink placed into 1% CO generated by an engine displayed excitement and myoclonic convulsions for 10 seconds before falling into a coma 29 seconds after gas introduction, whereas when placed into 3.4% CO generated from a compressed gas cylinder the convulsions occurred after the animals were in a comatose state, on average at 27 seconds (Lambooy et al., 1985). The results from this study led to the EU restriction on using any exhaust fumes for euthanasia of mink (Scientific Committee on Animal Health and Welfare, 2001). However Hansen et al (1991) concluded that, as CO affects the cerebral cortex before any other part of the nervous system, the consciousness of the animals must be already reduced to some degree before muscle inco-ordination occurs, which was also acknowledged by Lambooy et al (1985).

After reviewing the literature CSL (CSL, 1993b) concluded that any proposed use of CO for the fumigation of mammals should seek to ensure exposure to concentrations greater than 1% and to gradually increase concentrations to prevent the onset of convulsions before insensibility. When pigs were exposed to gradual rises of CO, i.e. 0.5% over 30 min, convulsions were not observed (Lambooy & Spanjaard, 1980). Convulsions had been observed when CO concentrations reached at least 1% within 1 min. The Defra (2005) report has shown that where lethal concentrations are achieved this occurs relatively quickly, i.e. within a few minutes. Although this is of concern all the studies looking at CO poisoning acknowledge that brain activity is diminished during any convulsions observed (e.g. Lambooy & Spanjaard, 1980).

The adaptation to burrow living that results in greater tolerance of carbon dioxide may also be important in metabolism of COHb, leading to increased resistance to CO poisoning. For example, rabbits exposed to sub-lethal doses of CO over an extended period were able to eliminate COHb much more quickly than guinea-pigs or dogs

(Semerak & Bacon, 1930). One rabbit exposed to 0.25% CO for over 3 hours showed no signs of distress, whereas at the same concentration a dog collapsed in 10-15 minutes (Burrell et al., 1914). However, at higher concentrations the differences are not so clear-cut (Oliver & Blackshaw, 1979). Neonates have a greater capacity to tolerate hypoxia after carbon monoxide exposure than adults (Winston & Roberts, 1978). Specifically, when two-day old mice were exposed to 2% CO they were more resistant than young adult mice to the lethal effects of CO. Old mice (150 days) were also more resistant than young adults. A similar effect was found by Hart et al (Hart et al., 1996b) during fumigation of fox dens with CO, where two of the dens contained two-day-old cubs. In one of the dens all five cubs survived, whereas in the second den, two out of six were still alive. This was despite the vixens having been killed. Consequently, fumigation of fox earths with CO in Australia is not recommended if young less than 4 weeks old are suspected to be present. In the absence of data to the contrary it seems reasonable to assume that neonate badgers would also be more resistant to the effects of CO.

The long-term effects of sub-lethal concentrations of CO are dependent on the degree of anoxia. At low to moderate levels full recovery is normal, however if severe anoxia is experienced permanent brain damage can occur. For example, Van Oettingen (1941) reported that the initial symptoms of CO poisoning in humans are headache and sometimes nausea, followed by deep unconsciousness. During the latter stage muscular convulsions and spasms may occur due to the stimulation by CO of the motor centre of the brain. Local bleeding in the motor centre may result in paralysis. Ginsberg & Myers (1974) exposed monkeys to up to 0.3% CO for up to 325 min. Four out of 19 survived and three of these suffered neurological deficits, including limb paralysis, alterations of muscle tone, blindness and deafness. In another study, Schwerma et al (1948) exposed dogs to a longer duration of CO at 0.3%, which increased the severity of the neurological secondary effects. If the use of CO is pursued further it is recommended that further consideration is given to the likelihood of sub-lethal effects arising from the fact that lethal concentrations might not be obtained everywhere in the sett, as suggested by the computational models (Defra, 2005).

In some of the experiments on CO toxicity, CO was produced from filtered vehicle exhaust gas rather than pure CO and this raises the question as to whether the humaneness of CO is altered by the means of its production. Because cars exhaust systems are continuously evolving to emit lower levels of CO, only idling, badly tuned petrol engines (by restricting the air intake to the engine) without a catalytic converter would be suitable for producing CO, Further investigation of how CO is generated by different petrol engines is required. Diesel engines are not suitable for reasons outlined below.

In a study that directly compared CO produced from petrol engine exhaust and pure CO, Lambooy et al. (1985) found slight differences in behaviour between the two sources of CO. However, these differences could have been due solely to the significantly different concentrations of CO in the chamber, in combination with the animals being placed directly into these relatively high concentrations. The only published study that compared filtered petrol exhaust gases with a similar final concentration and flow rate of pure CO found no differences in behaviour or signs of distress (Moreland, 1974). In addition, they speculated that the nitric oxide (up to 80 ppm) and unburnt hydrocarbons (up to 245 ppm) in the exhaust gas were unlikely to cause any irritation in the short time (3 minutes) before loss of consciousness. The concentrations of these gases were similar in filtered petrol exhaust gas to unfiltered petrol exhaust. Although several reports state that the high temperature and other pollutants in exhaust fumes have a detrimental effect on animals (Scientific Committee on Animal Health and Welfare, 2001; Scientific Panel on Animal Health and Welfare, 2004; Close et al., 1996), an extensive literature search has found no published information on this. If badgers were able to detect the fumes and find them aversive, they might move further into the sett where the fumes do not penetrate at a high concentration, therefore increasing the risk of sub-lethal exposure to CO and possibly pollutants. However, in the one paper that describes the responses of rabbits to fumigation with petrol engine exhaust fumes no signs of aversion or distress were observed before collapse (Oliver and Blackshaw, 1979). Nevertheless, if the approach of CO generated by petrol engines is pursued further then more information on the potential detrimental effects of exhaust pollutants on animals prior to insensibility would be beneficial.

In addition to CO, petrol engine exhaust gases typically contain 6% CO_2. This could increase susceptibility to CO poisoning. In rats exposed to 0.25% CO in the presence of 5.25% CO_2 more animals died during and post exposure, and the rate of formation of COHb concentrations was 1.5 times faster than with CO alone (Levin et al, 1987). Acidosis was more pronounced and prolonged, and recovery periods were considerably longer (onset at 60 minutes as opposed to 5 minutes after exposure). This effect was most pronounced at 0.25% CO and 5% CO_2. Exposure to increased concentrations of CO_2 causes an increase in respiratory rate, and the synergy effect of these two gases was attributed to a combination of respiratory and metabolic acidosis. Oliver and Blackshaw (1979) also mention the putative increased toxicity of exhaust fumes, but in a direct comparison Moreland (1974) did not observe a shorter time to death when comparing time to death from two sources of CO.

Diesel engines create significantly more irritant NOx compounds than petrol engines, ranging from 40 to 1500 ppm (Lindgren & Hansson, 2004) i.e. concentrations that are lethal. In one experiment where mice, guinea pigs and rabbits were exposed to diesel fumes, no behavioural effects were observed apart from lethargy in mice (Pattle et al., 1957). At post mortem pulmonary congestion, oedema, consolidation and emphysema were found in all animals, including those that survived the exposure. Death in rabbits only occurred after at least 7 hours exposure and was attributed to NOx as well as CO toxicity. In a final test the air intake was obstructed to simulate a badly tuned diesel engine. The resulting exhaust fumes were very dense and white, and also caused intense pain to the eye of a human observer within a few seconds. No other behavioural effects were observed though death occurred between 3 hours 20 minutes and 4 hours 35 minutes after initial exposure.

The temperature of vehicle exhaust fumes varies between 120 and 150^{o}C and this could have welfare implications. Although there are no data on the dissipation of the heat from exhaust gases in burrows there are data on the dissipation of the heat from the ignition of CO cartridges in the entrances to a rabbit warren (Ross et al., 1998). Much higher temperatures (800^{o}C) were measured near to the burning CO cartridges, but this heat was confined to the treated entrances and lasted for less than one minute. Most of the rabbits in this trial were found dead near the warren entrances. One rabbit had a small burn (1 cm) on its chin and several had singed fur. However as there was no evidence of smoke inhalation, it was thought that the rabbits became unconscious and fell onto hot material from the cartridge and died without regaining consciousness. Given the larger size of badger tunnels and the animals' ability to move away from the hot source, this is not thought to be a major concern.

4.5.2 Environmental impact

Minimal environmental impacts result from fumigation with CO. The gas disperses over time and poses no secondary poisoning hazard to predators eating animals killed by this method. The HSE short-term exposure limit (stel) for CO is 300 ppm; thus fumigation with CO is less hazardous to operators than fumigation with HCN or PH_3. In addition as the operation is undertaken outdoors and the generated gas is released within the sett no exposure to the operator should occur, provided the entrance around the CO generator is blocked. No PPE would be required.

4.5.3 Effectiveness & feasibility

CO is not absorbed by soil and has been used successfully for vertebrate pest control in the United States and Australia (Oliver & Blackshaw, 1979; Savarie et al., 1983; Deng & Chang, 1986; Pelz & Gemmeke, 1988) against a range of species including burrowing rodents, rabbits and large mammals like coyotes and foxes. CO for pest control has been produced in a variety of ways including charcoal burning in a stove mounted on a vehicle, from engine exhaust, and from combustion of cartridges containing a source of carbon (usually charcoal) and a source of oxygen (potassium nitrate or sodium nitrate). All methods are virtually unaffected by wet weather, but can be affected by windy conditions (CSL, 2001; Ross et al., 1998). On average 79% (range 50 to 100) of resident rabbits were killed by a prototype cartridge that has been developed and tested by CSL (Ross et al, 1998). Currently CO is not registered as an approved vertebrate control agent in the UK and approval through the usual process would probably require at least one year to obtain and the production of supporting data would cost at least £250,000. An experimental permit, which would constrain the time and area over which a product could be used, could possibly be obtained within 1-6 months with a reduced requirement for supporting data.

Vehicle petrol engines can produce a maximum concentration of 2% CO, if they do not have a catalytic converter fitted and are running fuel rich, i.e. at idling

(Harikrishna & Arun, 2003). In order to increase CO production Gigliotti et al. (Gigliotti et al., 2001) in Australia have developed an engine for fumigation that runs on methanol and produces substantially more CO, 6%. They argue that the exhaust fumes contain a lower concentration of hydrocarbons, sulphur dioxide and nitrous oxides (NOx) than petrol engine exhaust. However, the International Program on Chemical Safety report that methanol fuelled engines emit approximately 20 times more formaldehyde than petrol engines; formaldehyde being one of the most irritating chemicals in exhaust fumes. This engine is currently being evaluated in Australia for use with foxes and rabbits but is as yet unavailable commercially. Unlike methanol, using ethanol as fuel decreases the CO content of the exhaust fumes by up to 50%, and therefore it is not suitable for this application (Magnusson et al., 2002) .

Diesel engines are far less efficient than petrol engines at producing CO. The greatest concentration of CO that has been measured in diesel exhaust is 0.2% CO (Lindgren & Hansson,2004). In one experiment where mice, guinea pigs and rabbits were exposed to diesel engine exhaust gases, rabbits did not die after five hours exposure when CO levels were below 0.06 % and NOx levels were between 46 and 209 ppm (Pattle et al., 1957). When the exposure time was extended to 14 hours (0.05% CO and 46 ppm NOx) all rabbits and guinea pigs died but 10% of mice survived. Because of the low concentration of CO and the high concentration of irritants, diesel engine exhaust should not be used for fumigation purposes.

A lethal concentration of CO has been achieved in rabbit warrens (Oliver and Blackshaw 1979), and fox earths (CSL, 2001) using both vehicle exhaust fumes (rabbit warrens, Oliver and Blackshaw 1979) and cartridges (rabbit warrens, Ross et al., 1998; fox earths CSL, 2001) A CO generating engine (Deckson) failed to achieve 1% CO in one study of rabbit warren fumigation (Oliver and Blackshaw 1979), but exceeded this concentration in a second study (Thompson, 1969). These differences can be explained by differences in soil composition and demonstrates the importance of this factor. The soil in the former study was 96% sand, through which the gas can dissipate quickly. CO produced from a cartridge (DEN-CO-FUME) has also been used to kill adult foxes, but not all neonates, in natal earths (Levin et al, 1996). The foxes were found at the blind ends of tunnels between 5 and 10 m from the fumigation point. Although one of the foxes was still alive but unconscious when taken out, it would have almost certainly died when left in the fumigated warren. The volumes of only two dens were estimated, which were 0.35 and 1.6 m^3. The data presented above are from smaller and less complex tunnel systems than those that are typical of badgers. Ross et al (1998) found that 79% of resident rabbits were killed when fumigated with CO generated from cartridges.

To investigate the dispersal of CO further, particularly with respect to its distribution into blind tunnels, Defra (2005) evaluated four different methods of introducing CO gas into computer models of badger setts under a number of different conditions. The release methods were i) a CO fumigator, which is currently in development in Australia, hence referred to as the ‘Australian generator’ (Gigliotti et al., 2001); ii) exhaust fumes from an idling, badly tuned petrol engine without catalytic converter; iii) exhaust fumes from an idling diesel engine; and iv) CO cartridges as described by Ross et al. (1998) . The concentrations and flow rates of CO generated by each method are specified in Table 3. For all methods the gas dispersal in open and blind tunnels was investigated in a simplified 2D model, as well as the effects of soil porosity and wind effects (petrol engine and cartridge release only). The idling, de-tuned petrol engine without catalytic converter was selected as the most likely candidate for CO fumigation in the field and an additional set of models were run with this method, investigating the dispersal in a 3D model of a branched tunnel. In this model, tunnel ends and gas inlet entrance were blocked systematically.

It is very important to emphasise that every reference to petrol engine exhaust fumes refer to fumes from an idling, badly tuned engine without catalytic converter. With a catalytic converter the CO content in the exhaust fumes is significantly lower (approximately 0.2%), and resembles CO concentrations found in diesel engine exhaust fumes. CO concentration is highest when an engine is idling and de-tuning the engine by partially blocking the air inlet causes the combustion process to be incomplete, which also leads to higher concentrations of CO.

Table 3. Specifications, flow rates and concentration generated by different CO release methods

Release type	**Specification**	**Flow rate (lpm)**	**Concentration (%)**
Australian generator		800	6
Idling petrol engine (badly tuned and without catalytic converter)	3 litre engine idling at 600 rpm	900	2
Diesel engine	4.4 litre engine idling at 900 rpm	1980	0.2
Cartridges	Flow rate based on ignition of six cartridges	29.4 litres at 0.08m/s	100

The main conclusion of the report was that only the Australian generator and the idling, badly tuned petrol engine without catalytic converter were able to reach lethal concentrations (1% at 1 hour) in part of the sett, provided they ran continuously throughout this period. Generally, penetration of the gas in lethal concentrations into blind tunnels was difficult beyond very short distances of 2-3 meters at medium soil porosity after approximately 20 minutes. The diesel engine never reached the lethal concentration at any point and is therefore unsuitable for fumigation. The petrol engine and the Australian fumigator performed very similarly, although the Australian generator reached higher levels of CO (6% versus 2%). However, even the Australian generator did not generate lethal concentrations throughout the sett, and this suggests that the CO fumigation technique will not always be 100% effective, and that there is the potential for sub-lethal exposure to badgers. Higher concentrations were reached locally by blocking the entrance around a CO generator. Gas dispersed slightly better in a longer than a shorter blind tunnel.

Generation of CO by cartridge release would only be suitable for fumigation of small setts. With this method the lethal concentration of CO was only obtained in the first few meters of a blind tunnel, and made very little progress over time. Ignition of six cartridges into an open tunnel saw the lethal concentration of CO travel slightly further, but again could not be sustained. In a different, more complex, model that included wind effects and a downward slope, the lethal CO concentration had again only moved a few meters into a blind tunnel after five minutes. After this period the concentration reduced rapidly. Cartridges have the advantage of rapidly achieving high concentrations, therefore rendering the animals unconscious quickly and preventing escape, however the rapid increase in concentration may not be as humane as other methods. Fumigation success with CO may be improved if cartridges are used in conjunction with other generation methods.

The target lethal concentration of 1% for 1 hour could clearly only be sustained in parts of the sett if continuous generator methods were running throughout this length of time. When compared against experimental studies of CO dispersal, the models are conservative, probably as a result of only two criteria being modelled at any one time, and the various assumptions that were made. The modelled sett is also very small and simple compared to the large and complex setts that many badgers occupy. However, the modelling results confirm the difficulties of dispersing gas into a blind tunnel, the important effects of soil porosity, and also confirm that diesel engines are unsuitable for the generation of lethal CO concentrations.

Petrol engine exhaust has the advantage of continuous, prolonged introduction of CO into a tunnel, and the computational modelling results show that it can fairly easily reach lethal concentrations in open tunnels, although penetration into blind tunnels was still problematic (Defra, 2005). Oliver and Blackshaw (1979) used a small artificial warren to investigate the effects of fumigation of rabbits with petrol exhaust fumes. All animals died within 16 minutes and were exposed to high levels of CO ranging from 2.3 to 6%. However, the simple artificial structure contained three tunnels and three entrances, which were blocked when fumes were seen coming out. This effectively mimics open tunnels throughout the sett, which the computational models show aids gas dispersal considerably (Defra, 2005). In a larger sett with heavy soil (28% clay content) using various introduction points, a concentration of approximately 0.7% was achieved in a 3.5m long blind tunnel. After four hours this had decreased to only 0.5%, despite the engine being switched off within 8 minutes of introduction of the gas; however, 1% was achieved and sustained in a 2.5m blind tunnel.

Ross et al. (1998) measured gas concentrations at various points in an artificial rabbit warren. Depending on wind speed and direction, in some blind ends no CO was measured at all, whereas in others concentrations of CO were over 1% and sustained for 1 hour (in one example at approximately 4 meters from the introduction point). Ross et al (1998) used two cartridges in all entrance holes, together capable of producing a maximum of 20% CO throughout the warren. This fumigation scenario differs from the scenario modelled in Defra (2005) in two important ways: i) the model of the cartridge only produced a theoretical maximum of 2% CO in the tunnel, whereas Ross et al's (1998) approach could have generated far higher concentrations; and ii) Ross et al. (1998) placed cartridges in all entrances and sealed these immediately after introduction, whereas the Defra (2005) models only replicate one point introduction. In a larger warren fumigated with CO, described in the same paper, 21% of the animals survived, indicating that lethal concentrations had not penetrated far throughout the warren.

Trials carried out in an empty artificial fox earth (CSL, 2001) also showed that 1% CO generated by cartridges penetrated into blind tunnels (up to 6 meters) and could be sustained in calm conditions. These results were generated in sandy soil with high expected losses through diffusion into the soil, but as the earth was constructed above ground it may have benefited from improved air mixing within the tunnels, which is supported by the higher losses during windy weather. Also, the CO concentrations generated by the cartridges in this study were again higher than the projected maximum in the Defra (2005) CFD model. Levin et al (1996) killed most, but not all foxes with a single CO generating cartridge (capable of generating 3% CO), where most foxes were found dead in blind tunnels on average 7.1 m from the introduction point. Also, in a real life situation, movement of the animals within the set might help disperse gas to areas otherwise unaffected.

The assumptions in the Defra (2005) CFD models have in general led to conservative estimates of gas concentrations, but highlight the observed difficulties of dispersing gas into blind tunnels. The extent that this could potentially lead to sub-lethal exposure with consequent negative welfare consequences is unknown, especially as exposure to sub-lethal concentrations may become lethal after prolonged periods of time.

In summary, the results presented by the CFD models are likely to provide an important, but conservative approach to understanding CO dispersal in underground systems. If introduced gradually in high enough concentrations of 1% sustained for one hour, carbon monoxide can lead to a humane death. However, there remains the possibility that some animals could be exposed to sub-lethal levels of CO, potentially leading to neurological damage that could compromise welfare in survivors.

4.6 Summary & Conclusions

1) The relative humaneness, environmental impact and cost effectiveness are reviewed for gases that could potentially be used to fumigate badger setts. These gases are a) phosphine, b) hydrogen cyanide, c) carbon dioxide with and without argon, and d) carbon monoxide. However, as there are no relevant data on the reactions of badgers to potential fumigants, apart from hydrogen cyanide, it is necessary to try to extrapolate from results obtained with other species, including humans.

2) The use of fumigants could be suitable for smaller burrow systems, but cannot be reliably expected to kill all the animals in a complex system. The use of fumigants against other fossorial species (e.g. rabbits and moles) suggests that typically 20% of resident animals survive fumigation of their burrows.

3) The likelihood that significant numbers of non-target wildlife would be present in active badger setts is considered to be low and no gas poses a secondary poisoning hazard to predators eating animals killed by this method.

4) Phosphine is considered to be inhumane as well as a dangerous poison. However it is currently registered for use against moles and rabbits in the UK.

5) Both carbon monoxide alone and a carbon dioxide/argon mixture are considered to be humane provided sufficient concentrations of these gases can be achieved within badger setts. Carbon dioxide and hydrogen cyanide are considered to be moderately humane. Carbon dioxide is the only one of these gases that is registered in the UK.

6) Using the carbon dioxide with argon mixture will be very difficult to carry out, due to having to deliver a large number of heavy cylinders to a sett in order to create a sufficient volume of gas.

7) It is concluded that carbon monoxide is a relatively humane fumigation candidate, but questions remain regarding the manner of its production and use, and the occurrence of sub-lethal effects. Computer modelling of gas movement and diffusion through tunnels suggests that, with continuous CO generation methods at least, it is possible to achieve lethal concentrations of CO in open ended tunnels, but that gas movement into blind-ended tunnels is much more restricted and problematic. Excavated badger setts suggest that approximately 20% of total tunnel length comprises blind-ended tunnels. There is a risk with all methods that animals in blind-ended tunnels may not be exposed to lethal concentrations of gas. This risk is increased if the gas has an aversive effect on the animals as they might retreat into deeper areas of the sett. Dependent young may also be less susceptible to many of the gases, also increasing the risk of animals receiving sub-lethal exposure. In addition other features of the sett, such as slope and soil type, might influence CO dispersal.

8) It is concluded that diesel engines are not suitable for the production of CO as insufficient CO is generated to be widely applicable and irritant pollutants are present in the exhaust gases.

9) Models suggest that the fumigation with exhaust gases of an idling, badly tuned petrol engine without catalytic converter could produce lethal concentrations of CO, although this effect is limited by sett structure, in particular blind tunnels. If this method is to be used, the potential detrimental effects of pollutants on animals prior to insensibility need to be considered.

10) CO cartridges are unlikely to be effective for use in large setts.

11) The most effective method identified for generating high concentrations of CO was a methanol engine, although there are concerns about the potential severe irritancy associated with the likely presence of formaldehyde in such exhaust. A

prototype methanol engine is being evaluated in Australia but is as yet unavailable commercially.

12) If the use of CO is pursued further it is recommended that studies are undertaken to determine a) the likelihood of sub-lethal effects arising from the anticipated distribution of CO in setts and b) if the pollutants in petrol engine exhaust gases have detrimental effects on animals prior to insensibility due to CO toxicosis.

13) Currently CO is not registered as an approved vertebrate control agent in the UK. Registration would take a minimum of 1 year and carries considerable associated costs of at least £250,000.

5. Poisons

5.1 Generic issues

Poisons are usually added to foods that are normally eaten or preferred by the target species. Sufficient poison must be added to minimise the likelihood of sub-lethal poisoning and to ensure that an animal dies as quickly as the mode of action allows. In general, poisons that do not produce symptoms quickly are preferred. Animals can be very cautious of new foods and will often stop further feeding if they do not ingest a lethal dose before the first symptoms of poisoning occur. The vulnerability of badgers to various poisons is recorded in the Wildlife Incident Investigation Scheme (WIIS) reports (available on the Defra website), from cases where the cause of death has been confirmed as pesticide poisoning. Reports for the years 1998-2003 reveal that badgers were killed with the anticoagulant rodenticides warfarin, bromadiolone and difenacoum. Other pesticides implicated in badger deaths included metaldehyde and methiocarb (molluscicides), mevinphos (insecticide, approval withdrawn 1993), and aldicarb and endosulfan (insecticides). Given that badgers are clearly susceptible to some pesticides, there would appear to be some scope for the development of a poison formulation for use against them, although their susceptibility to the active ingredients listed later in this section is not known in sufficient detail to predict what bait formulations would be fully effective.

For some species, notably rodents, poisoning is regarded as an effective control method. However, conservation and welfare issues associated with the widespread use of broad-spectrum poisons against rodents have led to significant concerns regarding the use of this approach, and similar issues are likely to apply to any poison use against badgers. Despite recent initiatives designed to enhance the safety of rodenticide treatments, such as the use of tamper-proof bait boxes and the introduction of bait formulations that are less palatable to non-target species, accidental poisoning of domestic animals and livestock still occurs, and rodenticide residues are frequently found in wildlife. In the US, exposure of non-target wildlife to acute and chronic rodenticides is well-documented (Stone, 1999; Colvin, 1988), while in the UK, recent WIIS reports suggest that as well as badgers, a range of non-target species are exposed to anticoagulant residues during the course of rodent control operations, including red kites (*Milvus milvus*), buzzards (*Buteo buteo*), and foxes (Barnett et al., 2002b; Barnett et al., 2002a). Anticoagulant rodenticide residues have also been detected in British barn owl (*Tyto alba*) and polecat (*Mustella putorious*) carcasses (Shore et al., 1999; Newton et al., 1990). In most of these cases it is likely that the route of exposure was by secondary poisoning (consumption of poisoned rodents) rather than by primary poisoning (direct consumption of the bait) as most rodenticides are applied in cereal-based formulations that would not be attractive to carnivores or scavengers. Red kites readily take rat carcasses during rodenticide treatments (Ntampakis & Carter, 2005) and are therefore at a high risk from secondary poisoning, while other predators may be at risk from eating small mammals that have consumed rodenticides (Cox & Smith, 1990).

It is possible that bait formulations and delivery systems could be developed to maximise bait uptake by badgers, and minimise risks to non-target species. For instance, baits might be placed inside setts and the entrances blocked. However, it is not certain that badgers would necessarily eat baits underground (e.g. rabbits are reluctant to consume baits placed down their burrows, (Cowan et al., 1984)). Furthermore, it is likely that badgers could dig their way through most feasible blocking attempts and thus potentially eject uneaten baits from setts (e.g. badgers regularly eject bedding material etc. from their setts, (Neal & Cheeseman, 1996.)). In addition, a variety of wildlife may be permanently or temporarily resident in badger setts (section 4.1) and it is unlikely that risks to such non-target animals could be entirely eliminated. In particular, Birks and Kichener (1999) and McDonald and Harris (Mcdonald & Harris, 2000) have pointed out the threat to those polecats using rabbit burrows from fumigation targeted at resident rabbits. The extent to which sett based control of badgers using poisons might pose a similar risk to this and other species such as otters is unknown.

The poisons considered in detail in the following sections were selected when they met one or more of the following criteria: i) they include active substances that currently either have at least one approved use in a wildlife management context in the UK; or ii) they are used extensively outside the UK in a wildlife management context; or iii) they are the subject of research in the UK or elsewhere for lethal control of wildlife populations. Any poisoning approach would need to undertaken by personnel specifically trained in such use. While there are training courses regarding rodenticide use that would cover many generic issues associated with poison use there would be additional badger specific issues that would need to be covered by such training.

5.2 Alphachloralose

5.2.1 Humaneness

Alphachloralose is an anaesthetic and soporific, which is metabolised to chloral and was used as an anaesthetic in human and animal medicine. The therapeutic application of alphachloralose for humans was abandoned due to toxic side-effects. The oral LD_{50} ranges between 100 – 1000 mg/kg body weight depending on species. Alphachloralose depresses brain activity, slows metabolism and results in a lowering of body temperature with consequent death from hypothermia when used as a mammalian pesticide (Cornwell, 1969). Although alphachloralose is generally regarded as a humane pesticide, convulsive effects prior to insensibility have been recognised since 1893 (Hanriot & Gautier, 1897). Under anaesthetic or near-anaesthetic conditions humans can enter a state of “active coma” during which myoclonia (twitching muscular spasm) is seen, principally confined to limbs receiving a stimulus. However, this state is not reported as painful (Shita et al., 1981): the sense of pain is lost but there is increased reactivity to touch and sound. In humans, hyperactivity or hyperexcitability are usually the initial response. Small doses of alphachloralose cause myoclonus in preanaesthetic stages. Light anaesthesia results in generalised myoclonic convulsions in response to peripheral stimuli, and these convulsions cease in deep anaesthesia.

Symptoms reported in animals closely mimic those found in humans. Time to narcosis can be as low as 30 minutes following oral dosing, with death generally occurring within 4 hours. Although there is a report (PSD, 1997) of a conscious rat exhibiting violent tonic (continuous tension) convulsions within 10 minutes of eating the alphachloralose bait, within four minutes these convulsions had subsided into myoclonic twitches. Convulsions are suggestive of extreme distress; however, they occur in relatively few animals and, drawing from the human data, are of a shorter duration and are far less extreme than, for example, convulsions induced by strychnine (PSD, 1997). Animals ingesting non-lethal levels rapidly recover to full normal functioning (Meehan, 1984). “Alphachloralose is considered to be a relatively humane vertebrate control agent” (PSD, 1997).

5.2.2 Environmental impact

Secondary poisoning has killed buzzards and red kites. As the poison works rapidly bodies can be found above ground, however the risks are thought to be low for larger predators (PSD, 1997). No antidote exists for alphachloralose, hence there is a significant risk of accidental poisoning of wildlife from consumption of baits placed in the vicinity of badger setts. Poisoning of domestic animals has been recorded (Smith & Boyd, 1972) although fatalities are rare, as simply keeping the animal warm until recovery is usually a sufficient therapy.

5.2.3 Effectiveness & feasibility

Alphachloralose is currently approved only for use indoors to control mice. A concentrate is available (although subject to COPR) which could be mixed with bait suitable for badgers. However, because alphachloralose is a narcotic that induces death by hypothermia, it is most effective at temperatures below 10^{o}C and against small animals with rapid metabolisms. In practice alphachloralose is unlikely to be effective against badgers, as the temperature within the sett is usually greater than 10^{o}C (Moore & Roper, 2004), while their relatively large body size means that badgers are not particularly vulnerable to heat loss.

5.3 Anticoagulants

5.3.1 Humaneness

Anticoagulant poisons include the so-called 'first-generation' compounds warfarin and chlorophacinone, the 'second-generation' compounds difenacoum and bromadiolone, and the 'single-feed' compounds flocoumafen and brodifacoum. All act by interfering with Vitamin K-l metabolism and hence prothrombin formation and platelet-mediated clotting. Prolonged inhibition causes clotting factor deficiency, eventually leading to haemorrhaging which is fatal. The initial symptoms in rats are mild piloerection (erect hairs), oligaemia (blood volume deficit), slightly bloody eyes and nose, laboured breathing, and loss of body weight. These symptoms become progressively severe and other symptoms, such as subcutaneous haemorrhage, hypothermia, paralysis, blood in urine and faeces, may become apparent. Lethal doses are normally ingested after two or more days of feeding, but brodifacoum can be fatal after one feed. Death occurs within 5-7 days, but can be sooner and occasionally very much later. The animals typically remain conscious until just prior to death (PSD, 1997).

Haemorrhages have been reported in most places within the bodies of humans, rats and dogs poisoned with anticoagulants, and badgers are likely to experience similar symptoms. The anatomical sites include the gastro-intestinal tract, retroperitoneal region, adrenals, thorax, pericardium, brain, spine, joints, skin, liver, kidney, spleen and gonads. Whilst bleeding per se is not regarded as a painful process, the accumulation of blood, thereby causing swelling within confined spaces in tissues generally is (compartment syndrome effect) (PSD, 1997). Human case studies indicate that internal haemorrhage can cause severe pain, and behaviour indicative of severe pain has been observed in poisoned rodents (Mason & Littin, 2003; PSD, 1997). In addition loss of blood pressure may result in coma-like prostration of animals without loss of consciousness or loss of pain perception. Inactivity can indicate severe discomfort because haemorrhage into joints makes movement very painful. Therefore "as severe discomfort, which can last for several days, occurs in a large proportion of all the reported studies anticoagulant rodenticides must be regarded as being markedly inhumane." (PSD, 1997). Continued use of anticoagulants against rodents, despite the markedly inhumane mode of action, reflects the absence of effective alternative approaches that would be more humane.

5.3.2 Environmental impact

Wildlife casualties are common if baits are not adequately protected, indicating that anticoagulants are toxic to most, if not all, mammals and birds in the UK. Second-generation anticoagulant poisons have a relatively long half-life in the body of the poisoned animal thereby posing an additional danger to non-target species. Accumulated anticoagulant poisons have been found in livers of many wild carnivores and can reach levels causing symptoms even if poisoned carcasses are eaten only once every few days (e.g. Cox & Smith, 1990; Carter & Grice, 2000).

5.3.3 Effectiveness & feasibility

Anticoagulants are the most widely used pesticides to control rodent pests in Europe and North America. In relation to rodent control, there is considerable knowledge and experience concerning their effectiveness and the best methods of application. They are approved in the UK for the control of rats and mice and are widely available as ready-to-use (RTU) baits; concentrates are also available, subject to certain conditions relating to need and safe handling procedures. Some compounds, such as brodifacoum and flocoumafen, are restricted to indoor use only to minimise secondary poisoning risks. Most farmers will be familiar with the use of anticoagulant poisons through the control of rodent infestations on their land. However, although WIIS reports indicate that badgers are susceptible to these compounds there are no data on dose-response, which would be needed to develop effective formulations.

5.4 Calciferol

5.4.1 Humaneness

The term calciferol, better known by its common name vitamin D, encompasses a number of chemically similar compounds, all with very similar physiological action. Cholecalciferol is naturally produced in the body by the action of ultraviolet light on the provitamin 7-dehydrocholesterol. Ergocalciferol does not occur naturally but is produced artificially by the action of ultraviolet light on the provitamin ergostel. Both the ergo- and the chole- forms of calciferol have been used to control a variety of vertebrates. A toxic dose of calciferol increases intestinal absorption of calcium, stimulates bone resorption and increases renal tubular reabsorption of calcium. As a result there are abnormally high levels of calcium in the blood and, through a little understood process, this excess calcium is then deposited at normally uncalcified, soft tissue sites around the body (metastatic calcification e.g. Hass, 1956). Acute symptoms are usually observed after 24 hours from ingestion of the poison. Time to death has been reported between one and thirteen days in rats, however four to five days is an approximate average (PSD, 1997). In most cases death results from acute renal failure (Anning, 1948; Lund, 1974), although the anorexia and weight loss during calciferol intoxication can be so severe as to result in death from starvation.

Calcification resulting from calciferol poisoning can occur in many body tissues and cause severe suffering (CSL, 1994). Kidney and bladder calcification results in severe pain, leaves the animal moribund and ends in kidney failure. Both acute and chronic intestinal pancreatitis causes severe abdominal pain. Eye haemorrhage has been found and is known to cause blindness and severe pain in humans. Severe jaw pain has also been recorded. Brain congestion and haemorrhage causes intense headaches and sometimes convulsions. In addition significant suffering can result through severe dyspnoea (laboured breathing) and interstitial pneumonia. This mobilisation of calcium is also associated with a general feeling of malaise in humans. Sub-lethally poisoned animals are ill and anorexic for several days and will probably be left with long-term renal damage (Peterson et al., 1991). Given that calciferol can cause such severe suffering over several days it is not surprising that it “should be considered to be comparable to the anticoagulants as far as humaneness is concerned”, i.e. markedly inhumane (PSD, 1997). Use of calciferol against rodents, despite the markedly inhumane mode of action, is based on the absence of effective alternative approaches, particularly with respect to rodents resistant to anticoagulants.

5.4.2 Environmental impact

In general there is a low secondary poisoning risk as animals tend to cease eating calciferol bait after a lethal dose has been ingested and the compound is quickly metabolised. However, some mammals, including dogs, appear to be particularly susceptible to calciferol (Clarke & Clarke, 1975) and may therefore be at high risk of accidental poisoning through consumption of baits intended for badgers.

5.4.3 Effectiveness & feasibility

Calciferol is currently approved for the control of rats and mice. It is commercially available as Deerat (a concentrate from Rentokil) and Fatal (a ready-to-use bait from Sorex, although a Fatal concentrate has also been approved). Calciferol is also available in combination with difenacoum for mouse control. Sub-lethal doses induce anorexia (stop-feed effect). This can, to some extent, be avoided by pre-baiting to encourage animals to take a lethal dose on their first feed. However, experience shows that this is seldom fully effective and complete eradication is rarely achieved. Repeat treatments may be even less successful, as survivors exposed to sub-lethal doses during previous use often become bait shy.

5.5 Zinc Phosphide

5.5.1 Humaneness

Zinc phosphide releases phosphine in damp conditions, and especially at low pH. The toxicity of zinc phosphide is primarily due to the release of phosphine in the alimentary canal, particularly in the stomach (Casteel & Bailey, 1986). As already discussed in section 4.2, after absorption phosphine is a potent inhibitor of cytochrome oxidase. Organs with higher oxygen requirements are particularly sensitive to damage, with the main symptoms including cardiac congestion, cardiac oedema, cardiac arrhythmia's, pulmonary oedema, pulmonary congestion, respiratory distress, severe abdominal pain associated with gastric ulcers, nausea, vomiting, stupor, coma, intense headache, and liver congestion. Final symptoms include convulsions, paralysis and then coma until death (PSD, 1997). Most studies indicate that lethal intoxication results in death within a few hours (PSD, 1997), although intoxication can occur over several days in those rodents that do not die overnight (Timm, 1994). Data on recovery from phosphine poisoning are limited. However there is some evidence that rodents that receive a sub-lethal dose and manage to survive the illness period, have no long-term illness consequences (PSD, 1997). Use of zinc phosphide against rodents, despite the severe symptoms prior to death, is based on the absence of effective alternative approaches, particularly with respect to rodents resistant to anticoagulants.

5.5.2 Environmental impact

Primary poisoning of wildlife has been recorded, especially in seed-eating birds and waterfowl (Colvin, 1988). Secondary poisoning may occur if predators eat poisoned animals that contain a large dose in their alimentary canal (Guale et al., 1994). However because there is no accumulation in body tissue the risk of secondary poisoning is thought to be low (PSD, 1997).

5.5.3 Effectiveness & feasibility

Approval for advertisement and sale of zinc phosphide has expired, but it remains registered for a short period so that existing stocks can be used to control rats and mice. Against rodent pests, it is seldom more than 70-80% effective. Furthermore, extensive pre-baiting is necessary to minimise sub-lethal poisoning and consequent bait aversion in the survivors.

5.6 Para-aminopropiophenone (PAPP)

5.6.1 Humaneness

Para-aminopropiophenone (PAPP) is a potent methaemoglobin- (MetHb)-forming compound (Marrs & Bright, 1986). As MetHb cannot carry oxygen, sufficient concentrations of this compound cause anoxia and death (Kurata et al., 1993). In trials of the compound on foxes (Marks et al., 2004) no abnormal activity was observed until 10–24 minutes after dosage, where staggering and lethargy preceded either collapse or the fox adopting a prostrate position before rolling onto its side. In four out of five cases the fox later attempted to stand but was unable to do so, as it appeared to become progressively more lethargic. In all foxes, no activity was detected after 30–43 min and death was confirmed by loss of corneal reflex after a mean of 43 minutes. The coyote (*Canis latrans*) and swift fox (*Vulpes velox*) were found to have an oral LD_{50} for PAPP of 5.6 mg/kg body weight and 14 mg/kg body weight respectively. The sensitivity of badgers to PAPP is unknown.

5.6.2 Environmental impact

A substantial difference in the relative sensitivity of canids to oral doses of PAPP compared with other species has been found. Other mammal species were estimated to be between 10 and 30 times less sensitive than the swift fox and coyote respectively. There are indications that PAP is probably less toxic to mustelids, in part because vomiting is induced resulting in less poison being absorbed. Birds, such as the golden eagle (*Aquila chrysaetos*), were estimated to have an LD_{50} value approximately 29 and 71 times higher than that of the swift fox and coyote (Savarie et al., 1983)).

5.6.3 Effectiveness & feasibility

A formulation of PAPP, dimethylsulphoxide (DMSO) and condensed milk has been developed in Australia to control foxes. It is not approved for UK use and no products are available. The method of delivery was a spring-loaded ejector, which shot the formulation to the back of the mouth as the fox attempted to bite. This method seemed to be reasonably target-specific in Australia, although it is likely that domestic dogs would be particularly vulnerable in UK contexts.

5.7 Sodium monofluroacetate (1080)

5.7.1 Humaneness

Upon absorption, sodium monofluoroacetate is converted to fluorocitrate within the mitochondria. This inhibits the enzymes aconitase and succinate dehydrogenase resulting in citrate accumulation and interference with energy production and other cellular functions. Death occurs within 24 h from ventricular fibrillation or respiratory failure. Although not tested against European badgers, it appears to be toxic at 1-1.5 mg/kg body weight to American badgers (*Taxidea* sp).

Reports describing human cases of fluoroacetate poisoning identify anxiety, irritability, verbosity, agitation, hyperactivity, rapid heart rate, confusion, epigastric pain, headache, nausea and vomiting, faecal incontinence, respiratory distress, hyperaesthesia, muscular twitches, muscular pain, tetanic spasms, cardiac irregularity, gradual loss of alertness leading to coma, epileptiform convulsions, tonic convulsions, periods of flacidity, periods of lucidity between convulsions, and partial paralysis (Gajdusek & Luther, 1950; Peters, 1952; Brockmann et al., 1955; McTaggar.Dr, 1970; Reigart et al., 1975; Trabes et al., 1983; Chung, 1984; Chi et al., 1999; Chi et al., 1996; Robinson et al., 2002)

In the early stages of poisoning animals are typically reported as displaying a range of signs including; lethargy, retching and vomiting, trembling, faecal and/or urinary incontinence, unusual vocalisations, hyperactivity, excessive salivation, muscular weakness, uncoordination, hypersensitivity to nervous stimuli, and respiratory distress. Local neurological signs including muscular twitches (often affecting the face e.g. nystagmus, blepharospasm, etc.), and titanic spasms of the tail and limbs commonly follow. Neurological involvement may then progress to generalized convulsions, initially of a tetanic (tonic) nature, then of a clonic-tonic form (rapid successive relaxation and contraction of muscles), convulsions usually occurring cyclically(Chenoweth & Stjohn, 1947; Foss, 1948; Gajdusek & Luther, 1950; McIlroy, 1982). The time taken for symptoms to appear is between 30 minutes and 4 hours after ingestion, and death in foxes occurs approximately 90 minutes later (Marks et al., 2000). Animals receiving sub-lethal doses show signs of poisoning, but they metabolise and excrete the by products within 1-4 days and then recover; although partial paralysis (sometimes lasting for prolonged periods) is also common (Sherley, 2004).

5.7.2 Environmental impact

There is no antidote to 1080 and it can pose a significant secondary poisoning risk to a range of mammals. Residues of 1080 from uneaten baits are metabolised by soil micro-organisms (King et al., 1994). Although under favourable conditions (11–20 ^{o}C and 8-15% moisture) 1080 can degrade within 1-2 weeks, under cold and/or dry conditions it may persist in baits or the soil for several months (King et al. 1994). Similarly, although 1080 is degraded by aquatic plants and organisms, it can remain in cold water for 2 weeks (Ogilvie et al., 1996) and be detected after 10 months (Bowman, 1999). Nevertheless in New Zealand there is no evidence of 1080 in tap water delivered by reticulation, and no evidence of significant or prolonged 1080 contamination in surface or ground waters (e.g. Eason, 1997). As 1080 has primarily been used on a large scale in New Zealand where there are no native terrestrial mammals, the risks to non-target species has focussed on invertebrates, birds, domestic animals and humans (Eason et al., 1994). Birds are poisoned mainly by eating baits that have fragmented (Spurr, 2000). The persistence of 1080 in invertebrates is short-lived and hence the risks to insectivores are confined to a short period after the baits have been laid. Dogs are particularly susceptible to 1080 but other mammals such as stoats, weasels and cats are also killed (Murphy et al., 1999). Despite rigorous control of poisoning operations in New Zealand there have been cases where livestock have found and eaten sub-lethal amounts of 1080 baits thereby raising the possibility that contaminated meat and/or milk could be consumed by humans. However, as 1080 is rapidly absorbed and excreted it is thought unlikely to bioaccumulate in the food chain (Eason et al., 1994).

5.7.3 Effectiveness & feasibility

1080 has been used to control introduced mammals in New Zealand, Australia, Israel, Mexico and the USA and thus might be expected to be effective against badgers. It has typically been distributed by dropping baits from aircraft, a method that would not be appropriate for use in the UK. Cereal-pellet or carrot baits (usually containing 15% 1080) are used. 1080 is no longer approved and is not available in the UK.

5.8 T3327

5.8.1 Humaneness

T3327 is a carbamate insecticide and a potent poison. It works through inhibition of acetylcholinesterase (AChE), which influences the neuromuscular junction and the mechanics and control of respiration. Due to its configuration, toxicity is heavily influenced by route of administration as it is poorly absorbed across membranes (i.e. across intact skin). Oral LD_{50} ranges from 0.5-2.0 mg/kg body weight in laboratory animals and about 2.0 mg/kg body weight in foxes, but administered via broken skin or intravenously the toxicity increases 20-250 fold.

Symptoms are characteristic of cholinesterase inhibition, and include salivation, cardiac brachy-asystole, lachrymation, respiratory difficulties, restlessness, fasciculations, muscle weakness and paralysis, prostration and sometimes vomiting (Weinbroum, 2005). Death occurs as a result of respiratory failure, at the oral LD_{50} dose within approximately 47-90 minutes. With higher doses death can occur in foxes within minutes, although it can be as long as up to 1 hour 30 min. Convulsions occurred in two out of three foxes while still conscious before death. Sub-lethal doses of T3327 in rats and rabbits are either asymptotic, or lead to mild signs such as salivation and a depression in activity. Recovery is usually complete within 3 hours, with no lasting pathological effects. In foxes that had taken a high dose every animal that showed symptoms invariably died. They showed rapid, progressive locomotor impairment soon after the initial symptoms occurred, which prevented the animals from dispersing. Vomiting did not affect mortality. There is one report of accidental poisoning in badgers, in which 11 badgers were found dead. None of the carcasses showed any indications of vomiting or other signs of injury.

5.8.2 Environmental impact

The half-life of T3327 is approximately 5 days, with a decline to 20% by 7 days and to 5 % by 14 days (in bait left outside in 10-15 degrees C ambient temperature). Cold conditions increase the half-life. Capsules presented in mechanically recovered meat (MRM) baits are designed so that the T3327 will leach out over time and will bind to the soil, thereby effectively removing it from the environment. The potential for secondary poisoning is unknown, although T3327 is likely to be toxic to all animals that consume the poison. Field trials with foxes did result in accidental poisoning of badgers. Badgers were found dead between 50 and 470 m from bait stations. No other animals were found dead. In these trials the baits were buried approximately 6 inches under a turf of soil or grass. Trials using bait containing a single capsule with a lethal dose found that this increased the risk of T3327 falling out of the bait during ingestion, thereby posing an additional secondary poisoning risk.

Precautions for safe handling follow those for any toxic anti-cholinesterase, having particular regard to the large difference in toxicity between direct routes such as intravenous or subcutaneous and indirect routes such as oral or dermal. With encapsulated T3327 there is low risk to trained operators and encapsulated T3327 is very stable over time. The treatment of anti-cholinesterase poisoning (e.g. in case a capsule ruptures) involves a combined therapy of atropine plus oxime (P2S). Ingesting T3327 orally may outlast the effective period of a single therapeutic injection, and should be supported by booster doses.

5.8.3 Effectiveness & feasibility

The optimal dose of T3327 per capsule would need to be identified for use against badgers, and the appropriate encapsulated bait then produced. The baiting operations using T3327 that would take place in the event of a rabies outbreak are to be undertaken by trained personnel, and presumably the same restrictions would apply if T3327 were to be used against badgers. T3327 is not approved for use as a vertebrate control agent in the UK or anywhere else.

5.9 Summary & conclusions

1) The relative humaneness, environmental impact and cost effectiveness are reviewed for orally delivered poisons that might be used to kill badgers. The potential poisons considered include active substances that currently either have at least one approved use in a wildlife management context in the UK; or are used extensively outside the UK in a wildlife management context; or are the subject of research in the UK or elsewhere for lethal control of wildlife populations. These poisons are a) alphachloralose, b) anticoagulants, c) calciferol, d) zinc phosphide, e) Para-aminopropiophenone and f) Sodium monofluroacetate (1080) and g) T3327. However, as there are little relevant data on the reactions of badgers to these potential poisons it is necessary to try to extrapolate from results obtained with other species.

2) All poisons carry significant risks of non-target poisoning, either through consumption of the baits, or for some compounds through secondary poisoning when carcasses are scavenged. Training of operatives and design of baits to reduce non-target exposure can reduce these risks, but the risks remains significant.

3) Alphachloralose is considered to be a relatively humane vertebrate control agent. However, because it induces death by hypothermia, it is most effective at temperatures below 10^{o}C and against small animals with rapid metabolisms. Hence, alphachloralose is unlikely to be effective against badgers

4) Anticoagulants are regarded as being markedly inhumane. There is a relatively high potential for non-target wildlife casualties, which are common if rodenticide baits containing anticoagulants are not adequately protected (this applies to all poisons). Although wildlife incident reports indicate that badgers are susceptible to these compounds there are no data on dose-response, which would be needed to develop effective formulations.

5) Calciferol can cause severe pain and suffering lasting for a number of days prior to death. It is thus also considered to be markedly inhumane.

6) Zinc phosphide poisoning symptoms indicate substantial suffering although lethal intoxication generally results in death within a few hours. Although secondary poisoning risk to scavengers is likely to be low, non-target species that consume baits would be at risk of poisoning. Effectiveness may be compromised by sub-lethal poisoning leading to subsequent avoidance of baits.

7) Para-aminopropiophenone causes a relatively rapid death with limited negative symptoms with respect to welfare. It shows a degree of specificity for carnivores, although there are some indications that mustelids (e.g. badgers) might be less susceptible than canids (e.g. foxes). There is no formulation currently available in the UK and the compound is not approved for use here.

8) Sodium monofluroacetate (1080) causes death up to 4 hours after onset of toxicosis causing a wide range of symptoms prior to death including convulsions. There is no antidote and there is a significant secondary poisoning risk. The compound is not approved for use in the UK.

9) The carbamate T3327 has the potential to kill badgers. It causes death within a few hours but does cause convulsions prior to death in foxes. It is a non-specific poison so there are likely to be risks to non-target wildlife exposed to baits. T3327 is not approved for use in the UK.

10) There are no currently available poisons that would be effective against badgers without causing deaths that would be considered markedly inhumane and/or pose significant risks to non-target wildlife. Although some poisons are currently used against rodents, despite having a mode of action that is considered markedly inhumane, this use reflects the absence of effective alternative approaches that would be more humane.

6. Shooting free-moving badgers

6.1 Generic issues

Shooting is widely used in wildlife management and was accepted, under certain conditions, to be the most humane way of killing foxes (Burns et al., 2000). There is much common ground between the shooting of badgers and of foxes. Both species will often present as relatively small targets requiring some skill on the part of the shooter to place a shot in the right place for a clean kill. Thus, the humaneness and effectiveness of shooting are almost entirely dependent on the competence and skill of the shooter and, in the absence of a standard competence test, there is a risk that shooting by less skilful people will risk increased levels of wounding. Shooting will be more time consuming during the winter when the badgers spend more time within their setts. Shooting is also likely to be more labour intensive than fumigation, poisoning, snaring or cage trapping, but could be conducted as part of other activities, such as gamekeeping or fox control, which would reduce the time required.

Although shooting *per se* is not a prohibited method under the Wildlife and Countryside Act (1981), the actual technique that would be employed to shoot many badgers would be prohibited under this Act. As badgers are mainly active at night, shooting would mainly take place during hours of darkness. In order to shoot under such conditions the badger would usually have to be illuminated (i.e. so-called ‘lamping’) and/or a night sight would have to be employed. Both of these are prohibited methods when used for the killing or taking of the species, including the badger, listed in Schedule 6 of the Wildlife and Countryside Act (1981). However, the Wildlife and Countryside Act (1981) does allow licences to be granted for night sights and illuminating devices to be used to kill badgers for various purposes; these include preventing the spread of disease (Section 16(3)). Full discussion of the legal position is contained in other EWD documents.

Under the Protection of Badgers Act (1992), a shotgun of not less than 20-bore and a rifle firing ammunition with a bullet weight of not less than 38 gr and generating a muzzle energy of not less than 160 ft lbs must be used. While 12-bore shotguns may be commonly held by farmers to control various bird and mammal pests, a suitable rifle may not be: a rimfire rifle firing the .22 long rifle cartridge, as may be used to shoot rabbits, does not meet the provisions of the Act. Calibres of .222in or larger (i.e. centre-fire rifles, although a .22in rimfire magnum may be suitable) will be required, such as those likely to be held by gamekeepers or deerstalkers, people who should be competent to use them. There is no calibre that is specially recommended for shooting foxes, thus neither will there be for shooting badgers.

6.1.1 Humaneness

There is no reliable information on the wounding rates in other species (Burns et al., 2000) that could be extrapolated to badgers. Indeed wounding rates in relation to shooting as a wildlife management technique have not been comprehensively studied in any species. Fox et al (2005) have conducted trials involving the shooting at life-sized effigies of foxes. Although some consider the study to be seriously flawed, this research is useful in as much as it provides the only published data relevant to wounding rates. Shotguns and rifles using various types of ammunition were fired at moving and stationary fox targets by shooters, who differed in skill, from a range of distances. Depending upon where each target was hit, it was scored as ‘killed’, ‘seriously wounded’, ‘lightly wounded’ or ‘missed’. Rifles ‘killed’ better than shotguns and ‘wounded’ less. Also as the shooter’s skill level increased the ‘killed’ category increased and the ‘missed’ category decreased, although the ‘wounded’ categories changed relatively little. It should be remembered that the seriousness of a wound is not necessarily proportional to the presumed suffering: a seriously wounded animal might die within an hour whilst a lightly wounded animal might take days or weeks to either recover or die. It was not possible from these trials to predict the time a real fox would have taken to die or recover from the wounds. Nor was it possible to predict the levels of pain and suffering it might have experienced in the process. The authors concluded, not surprisingly, that there was no regime that had a zero probability of wounding and that wounding rates varied widely across trials with the different types of gun, ammunition and levels of skill. It should be noted that in real life many wounded foxes would be promptly killed by a second or third shot: for example about 33% of shots fired by Scottish Gun Packs shooters were repeat shots (Fox et al. 2003).

Badgers are not apparently disturbed when spotlighted and can be shot with a rifle. Although some shooters may feel confident enough about their ability and equipment to shoot at longer distances, given the size of the target the maximum effective distance for use of a rifle is likely to be around 150m, and a range of not more than 100m may be preferred (Fox et al., 2005) given that it may be difficult to judge distances at night and thus adjust the aim for the fall of shot. There is no ideal firearm/ammunition combination, but a suitable calibre rifle should be .223in firing, for example, a 55gr (3.56g) expanding bullet. The bullet generates muzzle energy in excess of 1000 ft lbs. The rifle can be fitted with a telescopic sight (or image intensifier) to improve shot placement and may also be sound moderated, but the loud crack as the bullet travels at supersonic speeds cannot be suppressed. Such a noise may disturb other animals nearby and restrict the shooter to killing only one individual in a group, It is unlikely that a shotgun will be efficient at killing a badger beyond 30-40 m, as individual pellets carry relatively low energies and shot patterns can be variable. The use of shotguns was thought likely to compromise the welfare of foxes (Burns et al., 2000). Again, individual shooters may feel that they have the skill to kill at longer ranges and that the use of chokes, large shot sizes and magnum cartridges extends the effective range, but the risks of injury invariably increase under these circumstances. In relation to deer stalking, Burns suggested that to minimise badly placed shots, stalkers should prove their competence before being permitted to shoot.

6.1.2 Environmental impact

The environmental impact of shooting is likely to be low unless many non-target animals are shot; there are no species of high conservation value that could easily be confused with a badger by a competent person. Lead shot can be replaced with alternative materials e.g. bismuth, tungsten matrix; although these materials may not retain kinetic energy as well as lead and this could influence the humaneness of the technique. At present there is no regulatory requirement to use these substitutes except over water. Shooting incurs the risk of human casualties from misidentification of the target and from ricochet; a number of human injuries or deaths occur each year as a result of ‘lamping’ operations to shoot foxes. Shooting from a high seat constructed close to the sett, or to an area baited to attract badgers, would reduce these risks.

6.1.3 Effectiveness & feasibility

Given that shooting is likely to take one animal at a time the approach is more suited for use by those who are regularly patrolling the ground for other purposes. Effectiveness is almost entirely dependent on the competence and skill of the shooter to kill the animal as quickly as possible. The shooter therefore requires a clear view of the animal, assure that it is in range, and it remains stationary long enough to take aim and fire.

Lamping (i.e. using spotlights to illuminate badgers at night) and shooting will be most cost-effective when vegetative cover is minimal e.g. in early spring or post harvest, rather than at other times of the year when the animals are hidden by growing crops or other vegetation. An alternative approach to 'lamping' that may be feasible when vegetation is high is to use bait, such as peanuts, to attract badgers to a cleared area near the sett and then shoot them from a high seat. In this case the use of a night-sight (e.g. image intensifier) might preclude the need for a spotlamp, and firing the shot from a high seat would increase safety by ensuring that any off-target bullet went into the ground. This method could, therefore, be preferable to lamping and shooting, particularly if vehicular access is difficult, safety is uncertain, and/or powerful spotlamps sweeping across the landscape would disturb too many people. However, the logistics of moving and securing high seats may be problematic.

Farmers, landowners or their agents will require appropriate authority from their local police force to possess and use firearms to kill badgers. Those who already possess Firearm Certificates for Section 1 firearms (rifles, some types of semi-automatic shotguns) will need an amendment to either include badgers as a target species for a particular firearm, or to acquire a specific firearm to kill badgers. Local Police Firearms Licensing Departments may have resource issues if there is a significant rise in applications for, or amendments to, firearm certificates, a problem that concerned ACPO during the FMD outbreak. In the absence of national guidelines, individual Police Forces may or may not allow certain large calibres to be used, such as .30-06in or .308in, that some may regard as an 'overkill' and more risky than use of smaller guns. Current licensing arrangements for shotguns are less restrictive and amendments may not be required. However, given the short range over which shotgun use will reliably kill badgers there will in general be a low likelihood of regularly encountering badgers at this distance, unless the animals have been attracted to a baited area beneath a high seat.

6.2 Summary & conclusions

1) Given that shooting is likely to take only one animal at a time this approach is more suited for use by those who are regularly patrolling the ground for other purposes. Shooting will be less effective during the winter when the badgers spend more time within their setts.

2) Badgers are not apparently disturbed when spotlighted and can be shot with a rifle. Although some shooters may feel confident enough about their ability and equipment to shoot at longer distances, given the size of the target, the maximum effective distance of rifles is around 150m, and a range of approximately 100m is preferred.

3) It is unlikely that a shotgun will be efficient at killing a badger except at very close range, as individual pellets carry relatively low energies and shot patterns can be variable. Again, individual shooters may feel that they have the skill to kill at longer ranges and that the use of chokes, large shot sizes and magnum cartridges extends the effective range. There is no information on the wounding rates that might occur when shooting at badgers.

4) Lamping and shooting is most effective when vegetative cover is minimal and thus visibility is relatively good, e.g. in early spring or post harvest, rather than when crops and other vegetation are high. However, if badgers can be enticed into a cleared area by an attractive bait it should be possible to shoot them from a high seat even during summer. This could reduce seasonal constraints on this method of culling.

5) Effectiveness and potential for avoiding wounding will depend on the training and competence of the shooter.

7. Use of snares and/or cage traps followed by shooting

7.1 Generic issues

Trapping badgers in cage traps (UK) or body snares (Ireland) has been used to reduce badger numbers in recent field experiments. For badgers, shooting appears to be the most widely accepted method of despatch for restrained animals. There are few data on the effectiveness of trapping badgers as a control technique, but information contained in the 2004 Independent Scientific Review of the Randomised Badger Culling Trial and Associated Epidemiological Research (available on the Defra website) suggests that trapping is unlikely to remove more than 80% of a population. A study of trapping feral cats in Australia found that young adults and kittens were more likely to be caught in cage traps and experienced adults in foot-hold traps (Short et al., 2002). The capture rate increased when young cats were becoming independent, population density was high and trapping effort was maximized. Conversely, trapping success declined when only a few experienced adults remained.

Under Section 11(2) of the Wildlife and Countryside Act (1981) it is an offence to set any type of snare (i.e. “any article”) “calculated to cause bodily injury to any wild animal in Schedule 6”, which includes the badger. However the Wildlife and Countryside Act (1981) allows licenses to be issued to use prohibited methods (e.g. snaring) to take and/or kill of species listed in Schedule 6 for a range of purposes including preventing the spread of disease. A licence under the Wildlife and Countryside Act (1981) would be required in order to cage trap badgers. However, snaring or cage trapping of badgers would require separate licensing under the Protection of Badgers Act (1992), as would the subsequent killing of badgers restrained by these means. Full discussion of the legal position is contained in other EWD documents.

The Agreement on International Humane Trapping Standards, as incorporated into the draft EU Humane Trapping Standards Directive (COM (2004) 532), proposes a legal requirement to assess the humaneness of those killing and restraining ‘mechanical’ traps that are used to kill or capture the 16 species listed in the Agreement. The badger is one of the species listed and, therefore, snares and cage traps would be covered by the Agreement. Therefore when the Directive comes into force, the humaneness of any type of snare or cage trap used to capture badgers will have to be assessed using the protocols for restraining traps outlined in the Agreement. These protocols involve testing the restraining mechanism in both on captive animals and free ranging animals in field trials. The trap will pass if at each stage of testing; a) the number of specimens of the target species from which data are derived is at least 20, and b) at least 80% of the animals under test show none of the adverse welfare indicators listed in Annex II of the Directive. As well as death, these indicators include such injuries as fracture, severance of ligament or tendon, amputation, breakage of tooth exposing pulp cavity, ocular damage including corneal laceration, and damage to spinal cord. In addition the Directive details additional behavioural and physiological measures to be carried out upon a sub-set of the test animals, but it is unclear if these additional tests are mandatory at this time.

Section 11(3) of the Wildlife and Countryside Act 1981 states that it is an offence if, whilst a cage trap or snare is in position, the trapper "fails, without reasonable excuse, to inspect it, or cause it to be inspected, at least once every day". Unfortunately this means that if inspected before dawn one day and after dusk the next there could be a period of approaching 48 hours between inspections. There are no data on how the probability of occurrence of the type of injury listed in Annex 2 of the Directive (see above) changes with the period of restraint within a cage trap or by a snare. However, in order to minimise the risk of adverse welfare consequences, animals should be dealt with as soon as possible after they are caught. The Independent Working Group on Snares (2005) has drawn up a Code of Good Practice for the use of fox and rabbit snares and its recommendations on inspection times are: "It is desirable that animals are dealt with as soon as possible after they are caught. During winter snares must be inspected as soon after sunrise as is practicable, and should again be inspected near dusk. In summer snares must be inspected before 9 am, and a further inspection should be conducted in the evening". These recommendations should be the minimal requirements for the inspection periods permitted when cage trapping or snaring badgers. Badgers caught at the beginning of a cold night would be susceptible to hypothermia, particularly if these animals had just emerged from the sett and had yet to find food. This risk would increase the longer the badger is left and conversely be minimised by frequent inspection. During cage trapping the majority of badgers are caught between dusk and midnight (CSL, 2005) and therefore inspection at the end of this period should not decrease the chance of catching but would minimise the time badgers were held in either a cage trap or snare. Additionally capture of non-targets could be reduced by restricting the time that the snare or cage trap were active to the times most likely to result in capture of a badger. In summary, the snare or cage trap ideally should a) not be set before dusk, b) be inspected late evening i.e. 23:00 to 24:00 and c) be inspected again at dawn.

7.2 Restraint with snares

7.2.1 Humaneness

The vast majority of snares used in the UK are set to restrain foxes or rabbits around the neck. Snares may be divided into free-running and self-locking types. A 'free-running' snare is a wire loop that relaxes when the animal stops pulling, whilst a 'self-locking' snare is a wire loop that tightens progressively by a ratchet action as the animal struggles. Under Section 11(1) of the Wildlife and Countryside Act 1981 it is an offence to kill or take, or to knowingly cause or permit the killing or taking of any wild animal using a self-locking snare. The Notes on Clauses to Section 11 of the Act state: "Under subsection 1(a) it would be an offence to set a self-locking snare in position with the intention of causing injury to any wild animal. The self-locking snare is regarded as a cruel method of taking wild animals". Thus it would seem that the ban on self-locking snares arose because of their perceived cruelty. However no experimental studies to assess the humaneness of snares, similar to those performed to assess the humaneness of mechanical traps, have been conducted, and it has been suggested that some designs of self-locking neck snares may be more humane than free-running neck snares (see below). As well as neck snares, there are also body snares designed to restrain the trapped animal around the body, and foot snares designed to hold one limb.

From field observations derived mainly from the snaring of foxes, it is clear that snares can have a range of welfare impacts on animals caught in them. At one extreme, snares have been the preferred method of biologists for the capture of foxes for radio-tagging in every UK study in a rural area (Broom, 1999; Lloyd, 1980;

Macdonald, 1987; Hewson, 1990; Reynolds & Tapper, 1995) and Broom (1999) reported that well-designed stopped snares can hold an animal without exerting painful pressure on it. Anecdotal accounts of the behaviour of radio-tagged animals before and after capture, has led to the consensus that any impact of capture is short-lived. However, the use of snares by research biologists may not generally be representative of the use of snares by other users because of more frequent snare inspection periods. At the other extreme there is no doubt that snares can cause severe injuries (e.g. NFBG, 2002) – several photographs of badgers severely injured by snares have been published. In some cases, snares have cut deeply into skin and underlying tissues causing wounds whose welfare effects can be conservatively inferred to be extremely severe, in that they were consistent with causing severe pain (in a vet's opinion) of prolonged duration (days in some cases), with no alleviation. It should be noted, however, that the snares in these cases would have been set to catch foxes rather than badgers and that the adverse effects are likely to have been caused by their manner of use and/or specific design. Both of these could be moderated to minimise, but not eliminate, the risk of adverse effects.

It has been argued that locking neck snares are humane as they kill rapidly. A pressure of only 4 pounds is required to cause ligature strangulation in humans and unconsciousness occurs between 10 and 15 seconds later. Death follows within a few minutes, as a result of blocking the jugular vein and/or carotid arteries and preventing oxygen supply to the brain rather than asphyxiation (DiMaio & DiMaio, 1998). However, in the two trials that have reported results from the use of locking neck snares it is clear that they do not cause a rapid death as a result of strangulation. In the Humane Traps Panel trial (1969) only 7 out of 88 foxes caught with self-locking snares were dead at the morning inspection, although many were moribund. The second study looked at self-locking snares to capture coyotes (Guthery & Beasom, 1978) and found 48% alive the morning after capture. The anatomy of a badger is different to both coyotes and foxes in that they have thicker and possibly stronger neck muscles; making it even more unlikely that a self-locking neck snare would kill the animal quickly.

Mechanisms that restrain the animal around the body rather than around the neck were used to capture badgers that would not enter cage traps during MAFF badger control operations in Devon (MAFF, 1984). The body snares used were free running and had stops, i.e. a crimp in the wire that prevents the noose of the body snare going below a set diameter. These body snares were set at ground level, or a few cm above, on active badger runs and were inspected every two hours. (Snares may also be set for badgers in the tunnel entrances to their setts but this raises the issue of trapped animals trying to retreat down their burrows). Of the 36 badgers restrained no injuries were reported, and only a few of the captured badgers were not caught around the body. Similarly Cheeseman & Mallinson (1980) used body snares to catch badgers for a radio-tracking study and stated that, when properly set, body snares cause less stress to restrained badgers than cage traps; they found no injuries in more than 50 captures using body snares whereas badgers sometimes damaged their teeth and claws in cage traps. However it must be emphasised that in these two studies the body snares were set by skilled personnel and checked every couple of hours. Setting a body snare for a badger is different from the placement of a neck snare for a fox. Hence, the competence of those using the technique is likely to affect capture rates and the welfare of the trapped badgers.

Currently there is only one foot snare, the Aldrich, licensed for use in the UK and this can only be used to catch large non-indigenous mammals, i.e. for the recapture of escaped zoo animals. Another foot snare, the padded Rose Leg Cuff, is being developed for the capture of foxes and badgers. Pen trials have been carried out of the padded Rose leg cuff against badgers and there was no evidence that restraint, for up to 8 hours, by the padded Rose leg cuff severely stressed or injured the captive badgers. The animals spent approximately 20 minutes of the first hour of restraint attempting to pull the trapped limb from the padded foot snare. Thereafter this activity decreased to an average of only 5 minutes per hour for the remainder of the restraint period. The leg cuff caused no skin abrasions or limb injuries. The trapped paw showed some swelling but this decreased swiftly (within 30 minutes) after release from the leg cuff, and additional veterinary examinations 24 hours later found that all the trapped limbs were completely normal. Field trials of this device against badgers are currently in progress but it is too early to judge its efficacy under field conditions.

Shooting restrained badgers can be carried out using firearms and ammunition that comply with the Protection of Badgers Act (1992); i.e. a shotgun of not less than 20-

bore, and a rifle firing ammunition with a bullet weight of not less than 38 gm and generating a muzzle energy of not less than 160 ft lbs must be used. A 12-bore shotgun, a type many farmers already possess, firing a cartridge loaded with BB shot should be effective. Badgers may be shot at distances that avoid any risk of splashback and, provided a suitable backstop is present, less risk of ricochet, yet close enough to ensure that a competent shooter will kill the animal humanely, i.e. within 30m. Any incidental damage to the snare is of little consequence as replacements can be easily obtained or made.

7.2.2 Environmental impact

The major environmental impact of snares arises from the accidental capture of non-target species. During MAFF badger control operations in Devon, over a total of 1,805 body snare-nights, only two non-target animals were captured (0.001%). Both non-targets were foxes and their health status is unknown. However, in the fox snaring trials conducted in the Humane Traps Panel Trial (1969) similar rates of capture were recorded for both the target and non-target species. This is in agreement with more recent reports showing that non-target captures are between 40 and 48% of total number of captures in snares set for foxes (IWGS, 2005). The use of stops will help limit non-target captures of smaller species and accidental capture by limbs of all species. Non-target captures should also be reduced by the implementation of best practice whilst setting snares; in particular giving due consideration to the size of the noose, the height off the ground and the type of run it is placed on. Body snares for badgers should be set either on or just above the ground and therefore a “jump bar” can be used to prevent deer and other livestock from being caught by a leg.

7.2.3 Effectiveness & feasibility

The average capture rate achieved by MAFF personnel during the badger control operations in Devon was only approximately 1 badger per 50 badger snare-nights. However, it should be emphasised that here the majority of the badgers had already been removed by cage traps before the body snares were deployed. Of some concern from this report is the fact that around 33% of captured badgers escaped. The report argues that this was probably due to the free running body snare becoming too loose and suggests that a better design of body snare would include a ‘one-way’ stop to prevent the wire loosening too much, in addition to a stop to prevent the wire becoming too tight. The one-way stop would allow the eye of the noose to pass over it in one direction but not back again whilst the traditional stop would prevent all passage of the eye. The eye of the noose would therefore freely move between the traditional stop which sets the smallest diameter possible for the noose, and the one-way stop which sets the largest possible diameter. A much higher capture rate of 1 badger per 3 body snare-nights was achieved when catching badgers for a radio-tracking study (Cheeseman & Mallinson, 1980) but as no previous badger removal had occurred in this case the rates are not comparable. More data are required before an accurate assessment of the efficacy of body snares in a variety of habitats can be made. Capture rates for foxes in neck snares are very dependent on the skill of the operator. Capture rates of 48 foxes per 1000 snare days can be achieved, though if set in a bad place are as low as 5 captures per 1000 snare days (IWGS, 2005). These rates are much lower than the reported capture rate achieved for badgers by Cheeseman and Mallinson (1980).

Foot snares are powered by springs and hence have to be granted Spring Traps Approval Orders before they can be used in the UK. No suitable foot snare is currently approved in the UK and there are no relevant efficacy data.

7.3 Restraint with cage traps

7.3.1 Humaneness

In the RBCT cage traps were set in the countryside near to occupied setts and normally pre-baited for several days. The traps were constructed from wire mesh that may be coated in plastic to reduce injuries to the captured animal. Much information is available on injuries caused by cage traps to badgers as a result of the Krebs trial (Woodroffe et al., 2005b). Injuries to badgers caused by cage traps have also been assessed by wildlife biologists (CSL, 2005). From the data collected during the Krebs trial 88.5% of 6000 captured badgers had no detectable injuries. This was significantly different from that found by the wildlife biologists (CSL, 2005) who found that 51% of 902 captured badgers had some form of injury. This discrepancy may be due to the wildlife biologists making a more detailed examination of the badgers. In both studies the vast majority of the injuries found were of a minor nature, e.g. abrasions and hair loss on the limbs and snout. Although a small proportion of the badgers did sustain serious injuries in both studies the incidence of such injuries is not sufficient to cause the cage trap to fail the requirements set out in the draft EU Humane Trapping Standards Directive (COM (2004) 532). These more serious injuries included tooth breakage and jaw damage (1% CSL, 2005; 1.8% Woodroffe et al., 2005b) and claw and pad injuries (2.8% CSL, 2005), and were most likely inflicted while the badgers were trying to escape from the cage traps. Data on time spent in the cage trap indicate that injuries are most likely to be obtained at the start of the capture period and may not accumulate during the restraint period; hence reducing the time in the cage trap by a couple of hours is unlikely to have an impact on severity of injuries. However, it cannot be assumed that because the animal is not trying to escape it is experiencing less fear. Many animals adopt an apathetic response to stressful situations even though their physiological state implies that they are distressed. Some indication as to the relative distress caused by cage trapping can be gauged from re-capture incidences during field studies. High re-capture rates were reported in a longitudinal study of badgers (Tuyttens et al., 1999) suggesting that the distress caused by the initial capture was not great and/or not lasting.

To cause immediate unconsciousness and then death by shooting, a bullet or shotgun pellet should destroy the respiratory centres in the brain stem. In order to achieve this with a badger restrained in a cage trap it is desirable to fire the shot within 2-15cm of the top of the badger’s head. This means placing the muzzle of the barrel (or moderator if fitted) within the cage, which in turn has implications for the minimum mesh size of the trap. Defra officials, operating under Crown exemption, use a single-shot pistol firing a .22in subsonic, hollow-nose bullet weighing 40gr and generating muzzle energy of 84-86 ft lbs. To meet the provisions of the Protection of Badgers Act, more powerful bullets, generating 160 ft lbs or more, fired from a rifle would have to be used. This is likely to result in the use of centre-fire cartridges, with bullets that often generate in excess of 1000 ft lbs. Shooting at such close range with this type of ammunition poses significant risks to the operator from bullets passing through the badger and ricocheting off the cage mesh or hard surfaces lying underneath the trap. The use of a shotgun of 20-bore or larger presents similar risks. The disruption caused by more destructive ammunition may also result in splashback of potentially infected tissue onto the operator, a risk that led MAFF to reject the use of a .410 shotgun (which is less powerful than a 12 bore). Firearms with barrels or moderators too thick to pass through the mesh should not be used, as firing into the cage from outside risks deflecting the shot as well as damaging the trap. Farmers or landowners or their agents would require a Section 5 permit issued by the Home Office to possess and use a pistol, but the wording in the Protection of Badgers Act would seem to exclude pistols, even if ammunition that met the muzzle energy criterion could be obtained. It thus may not be possible for farmers to obtain a suitable firearm/ammunition combination to despatch badgers in cage traps humanely and safely.

7.3.2 Environmental impact

Uninjured non-target species can be released as soon as they are found. However they could be injured (particularly tooth and claw damage) as a result of escape attempts, and they will also experience stress and fear as a result of being captured.

7.3.3 Effectiveness & feasibility

Cage traps are more cumbersome to transport and manoeuvre on site, and are more easily located by animal rights protesters than body snares. Furthermore, as badgers can be initially wary of cage traps, a common practice is to leave traps unset, but baited, for a number of days until the animals begin entering without hesitation. This approach will often maximise the number of individuals caught on the first trap night, but necessarily delay the control operation and increase the costs due to more inspections. If the cage traps are too closely spaced, dominant individuals may monopolise the bait and effectively exclude others. For badgers, cage traps can be set near active setts and seem to be particularly successful in the summer when food is short, whereas body snares set along major pathways can be effective all year round. When using cage traps to catch badgers for a radio-tracking study Cheeseman & Mallinson (1980) found that, with pre-baiting, the average success rate in their study area was one capture per six trap-nights, but that in mid-summer when food was scarce it became as high as one capture per two trap-nights.

7.4 Summary & conclusions

1) Although the adverse welfare status of badgers caught in neck snares set for foxes has been publicised, very few data are available on the humaneness of body snares specifically set for the capture of badgers. From what little information there is it

appears that, when correctly set for badgers, both body and padded foot snares cause few injuries (although the humaneness of padded foot snares has so far only been tested in pen trials and effectiveness has yet to be assessed). Setting a body snare for a badger is different from the placement of a neck snare for a fox and, therefore, training in this specialised technique would be required; there would be a serious risk of both injury to badgers and non-target capture if used by persons without the appropriate competencies.

2) Snares should only be used to restrain, not kill, badgers.

3) No suitable foot snare is currently approved in the UK. Also the humaneness of s used on badgers will need to be assessed as required by the Agreement on International Humane Trapping Standards, as incorporated into the draft EU Humane Trapping Standards Directive (COM (2004) 532),

4) Cage traps cause very few major injuries to badgers but can result in a significant number of minor injuries.

5) Cage traps are far more cumbersome to transport and manoeuvre on site than snares and are easier for animal rights protesters to locate. Furthermore, as badgers can be initially wary of cage traps, a common practice is to leave traps unset, but baited, for a number of days until the animals begin entering without hesitation. Whilst this approach maximises the number of individuals caught on the first trap night, it necessarily delays the control operation and increases costs.

6) Cage traps are positioned near active setts and are particularly successful in the summer when food is short; whereas snares are set along major pathways and can be effective all year round. Where both cage traps and body snares were used to capture badgers in the same study area, the average capture rate for cage traps was one badger per six trap-nights, and for body snares one badger per three body snare-nights.

6) Shooting restrained badgers can be carried out using firearms and ammunition that comply with the Protection of Badgers Act 1992. A 12-bore shotgun, a type many

farmers already possess, firing a cartridge loaded with BB shot should be effective, safe and legal.

7) To despatch badgers in cage traps Defra officials, operating under Crown exemption, use a single-shot pistol firing a .22in subsonic, hollow-nose bullet weighing 40g and generating muzzle energy of 84-86 ft lbs. To meet the provisions of the Protection of Badgers Act, more powerful bullets fired from a rifle would have to be used and this would pose significant risks to the operator from bullets passing through the badger and ricocheting off the cage mesh or hard surfaces lying underneath the trap. The use of a shotgun of 20-bore or larger presents similar risks. It may not be possible for farmers to obtain a suitable firearm/ammunition combination to despatch trapped badgers humanely and safely.

8. Overview

1) It is recommended that the following approaches are not given further consideration:

Fumigation with:

a) Phosphine –inhumane (4.2.1)

b) Hydrogen cyanide – less humane (4.3.1) and less feasible than other potential fumigants (4.3.3)

b) Carbon dioxide – less humane than other potential fumigants (4.4.1) and unfeasible (4.4.3)

c) Carbon dioxide with argon – unfeasible (4.4.3)

e) Carbon monoxide generated by diesel engine - less humane than other potential fumigants (4.5.1) and unfeasible (4.5.3)

Poisons

There are no currently available poisons that would be effective without causing deaths that would be considered markedly inhumane and/or risks to non-target wildlife (5).

2) It is recommended that the following approaches are not given further consideration at this time:

Fumigation with CO generated by portable methanol engine

Not currently available for field use and possible humaneness concerns (4.5.3); and potentially suitable alternatives already exist

Use of padded foot snare followed by shooting

Currently no information on effectiveness (7.2.3)

3) The following approaches have potential for further consideration:

Fumigation of setts with:

a) Carbon monoxide generated by cartridges – could be effective for small setts, gap in knowledge regarding potential sub-lethal effects (4.5.3), currently CO is not registered as an approved vertebrate control agent in the UK.

b) Carbon monoxide generated by an idling, badly tuned petrol engine without catalytic converter - gap in knowledge with regard to concentrations attainable in large setts particularly in relation to blind tunnels, gap in knowledge with regard to potential sub-lethal effects and adverse effects of non-CO components of exhaust gases (4.5.3), currently CO is not registered as an approved vertebrate control agent in the UK.

Shooting of free-running badgers:

Requires access to a rifle of at least .222 bore. While appropriate shotguns may produce a humane kill at short ranges, at longer ranges wounding is likely to be a significant issue, this severely compromises their practical use in most circumstances. Humaneness with respect to rifles and shotguns will depend on training and competence of operator. Current gap in knowledge regarding wounding rates of badgers (6.1.1).

Restraint followed by shooting:

a) Body snares – may be effective for use by trained personnel of proven competence but current information gap regarding injuries (7.2.1) and non-target captures (7.2.2)

b) Cage traps – unfeasible for use by non-Crown employees due to type of firearms required for safe and humane despatch (7.3.1)

9. References

Andrews, E. J., Bennett, B. T., Clark, J. D., Houpt, K. A., Pascoe, P. J., Robinson, G. W. & Boyce, J. R. 1993. 1993 Report of the Avma Panel on Euthanasia. *Journal of the American Veterinary Medical Association*, **202,** 230-249.

Anning, S. T. 1948. Calciferol in the Treatment of Chilblains. *Lancet*, **254,** 573-573.

Anonymous. 2004. Material Safety Data Sheet: Carbon Monoxide. In: *Document number 001013*, pp. 12: Airgas Inc.

Barnett, E. A., Fletcher, M. R., Hunter, K. & Sharp, E. A. 2002a. Pesticide poisoning of animals 2000: Investigations of suspected incidents in the united kingdom. A report of the Environmental Panel of the Advisory Committee on Pesticides.

Barnett, E. A., Fletcher, M. R., Hunter, K. & Sharp, E. A. 2002b. Pesticide poisoning of animals 2001: Investigations of suspected incidents in the united kingdom. A report of the Environmental Panel of the Advisory Committee on Pesticides.

Bateson, P. 1991. Assessment of Pain in Animals. *Animal Behaviour*, **42,** 827-839.

Bateson, P. 1992. Do Animals Feel Pain. *New Scientist*, **134,** 30-33.

Birks, J. D. S. & Kitchener, A. C. 1999. The distribution and status of the polecat Mustela putorius in Britain in the 1990s. London: The Vincent Wildlife Trust.

Bourne, J., Donnelly, C., Cox, D., Gettinby, G., McInerney, J., Morrison, I. & Woodroffe, R. 1998. Towards a sustainable policy to control TB in cattle. First Annual Report of the Independent Scientific Group on Cattle TB. London: MAFF Publications.

Bowman, R. G. 1999. Fate of sodium monofluroacetate (1080) following disposal of pest bait to a landfill. *New Zealand Journal of Ecology*, **23,** 193-197.

Britt, D. 1987. Humaneness of carbon dioxide as an agent of euthanasia for laboratory rodents. In: *Euthanasia of Unwanted, Injured or Diseased Animals or for Educational or Scientific Purposes*, pp. 19-31. Potters Bar: UFAW.

Brockmann, J. L., McDowell, A. V. & Leeds, W. G. 1955. Fatal Poisoning with Sodium Fluoroacetate - Report of a Case. *Journal of the American Medical Association*, **159,** 1529-1532.

Broom, D. 1999. The welfare of vertebrate pests in relation to their management. In: *Advances in Vertebrate Pest Management* (Ed. by Cowan, P. D. & Feare, C. J.), pp. 309-329. Fürth: Filander Verlag.

Burns, L., Edwards, V., Marsh, J., Soulsby, L. & Winter, M. 2000. Final Report of the Committee of Inquiry into Hunting with Dogs in England and Wales. London: HMSO.

Burrell, G., Seibert, F. & Robertson, I. 1914. Relative effects of carbon monoxide on small animals. pp. 23. Washington: US Bureau of Mines.

Carter, I. & Grice, P. 2000. Studies of re-established Red Kites in England. *British Birds*, **93,** 304-322.

Casteel, S. W. & Bailey, E. M. 1986. A Review of Zinc Phosphide Poisoning. *Veterinary and Human Toxicology*, **28,** 151-154.

Chapman, R. C. & Bennett, A. F. 1975. Physiological Correlates of Burrowing in Rodents. *Comparative Biochemistry and Physiology a-Physiology*, **51,** 599-603.

Cheeseman, C. & Mallinson, P. 1980. Radio tracking in the study of Bovine Tuberculosis in badgers. In: *A handbook on Biotelemetry and Radio Tracking. Proceedings of an International Conference on Telemetry and Radio tracking*

in Biology and Medicine, Oxford, 20-22 March 1979 (Ed. by Amlaner, C. J. & Macdonald, D. W.), pp. 649-686. Oxford: Pergamon Press.
Chenoweth, M. B. & Stjohn, E. F. 1947. Studies on the Pharmacology of Fluoroacetate. 3. Effects on the Central Nervous Systems of Dogs and Rabbits. *Journal of Pharmacology and Experimental Therapeutics*, **90,** 76-82.
Chi, C. H., Chen, K. W., Chan, S. H., Wu, M. H. & Huang, J. J. 1996. Clinical presentation and prognostic factors in sodium monofluoroacetate intoxication. *Journal of Toxicology-Clinical Toxicology*, **34,** 707-712.
Chi, C. H., Lin, T. K. & Chen, K. W. 1999. Hemodynamic abnormalities in sodium monofluoroacetate intoxication. *Human & Experimental Toxicology*, **18,** 351-353.
Chung, H. M. 1984. Acute-Renal-Failure Caused by Acute Sodium Monofluoroacetate Poisoning. *Veterinary and Human Toxicology*, **26,** 414-414.
Clarke, E. G. C. & Clarke, M. L. 1975. *Veterinary Toxicology*. London: Baillière Tindall.
Close, B., Banister, K., Baumans, V., Bernoth, E. M., Bromage, N., Bunyan, J., Erhardt, W., Flecknell, P., Gregory, N., Hackbarth, H., Morton, D. & Warwick, C. 1996. Recommendations for euthanasia of experimental animals .1. *Laboratory Animals*, **30,** 293-316.
Colvin, B. A., Hegdal, P.L. and Jackson, W.B. 1988. Review of non-target hazards associated with rodenticide use in the USA. *EPPO Bulletin*, **18,** 301-308.
Compressed Gas Association. 2001. Oxygen-Deficient Atmospheres. *Safety Bulletin*, **SB-2, 4th edition**.
Conlee, K. M., Stephens, M. L., Rowan, A. N. & King, L. A. 2005. Carbon dioxide for euthanasia: concerns regarding pain and distress, with special reference to mice and rats. *Laboratory Animals*, **39,** 137-161.
Cooper, J., Mason, G. & Raj, M. 1998. Determination of the aversion of farmed mink (Mustela vison) to carbon dioxide. *Veterinary Record*, **143,** 359-361.
Cornwell, P. B. 1969. Alphakil—a new rodenticide for mouse control. *Pharmaceutical Journal*, **202,** 74-75.
Cowan, D. P., Vaughan, J. A., Prout, K. J. & Christer, W. G. 1984. Markers for measuring bait consumption by the European wild rabbit. *Journal of Wildlife Management.*, **48,** 1403-1409.
Cowlin, R. 1967. An excavation of a badger sett in Southend. *The Essex Naturalist*, **32,** 70-72.
Cox, P. R. & Smith, R. H. 1990. Rodenticide ecotoxicology: assessing non-target population effects. *Functional Ecology*, **4,** 315-320.
CSL. 1991. Assessment of the relative humaneness of phosphine and hydrogen cyanide in rabbit control. Unpublished internal report: Central Science Laboratory.
CSL. 1993a. Convulsions and insensibility - a literature review: implications for the assessment of the humaneness of vertebrate pesticides. Unpublished report to the Pesticide Safety Division: Central Science Laboratory.
CSL. 1993b. Development of carbon monoxide as a means of rabbit control. Unpublished internal report: Central Science Laboratory.
CSL. 1993c. Review of the toxicology of phosphine in relation to its use in relation of to its use in rabbit control. pp. 24. Unpublished internal report: Central Science Laboratory.

CSL. 1994. The Humaneness of Calciferol: A Literature Review. Unpublished internal report: Central Science Laboratory.

CSL. 2001. Use of a humane method for fox control. Unpublished internal report: Central Science Laboratory.

CSL. 2005. Injuries in cage trapped badgers (Meles meles L). Unpublished internal report: Central Science Laboratory.

Danneman, P. J., Stein, S. & Walshaw, S. O. 1997. Humane and practical implications of using carbon dioxide mixed with oxygen for anesthesia or euthanasia of rats. *Laboratory Animal Science*, **47,** 376-385.

Darden, T. R. 1972. Respiratory Adaptations of a Fossorial Mammal, Pocket Gopher (Thomomys-Bottae). *Journal of Comparative Physiology*, **78,** 121-&.

Defra. 2005. A Computational Fluid Dynamics assessment of factors which will effect carbon monoxide dispersion within a simplified badger sett. pp. 56. Internal report to Defra.

De Vries, H. W., Zimmerman, A. N. E., Storm- van Leeuwen, W., Maas, A. H. J., Douze, J. M. C. & De Leeuw, R. J. M. 1977. Experimental Study of Acute Carbon Monoxide Intoxication in Dogs. *Acta Pharmacologica Et Toxicologica*, **41,** 374-392.

Deng, Z. & Chang, Z. 1986. Efficacy of a carbon monoxide gas cartridge against field rodents. In: *Twelfth Vertebrate Pest Conference* (Ed. by Salmon, T.), pp. 163-165. University of California, Davis, California.

DiMaio, V. & DiMaio, D. 1998. *Forensic Pathology*. New York: Elsevier.

Eason, C. 1997. Sodium monofluroacetate toxicology in relation to its use in New Zealand. *Australisian Journal of Ecotoxicology*, **31,** 57-64.

Eason, C., Morgan, A. & Wright, G. 1994. The fate of sodium monofluroacetate (1080) in stream water, and risks to humans. *Human and Experimental Toxicology*, **13,** 640.

Egekeze, J. & Oehme, F. 1980. Cyanides and their toxicity: a literature review. *Veterinary Quarterly*, **2,** 104-114.

Foss, G. L. 1948. Some Effects of an Acute Toxic Substance Formed by Solution of Methyl-Bis (B-Chloroethyl) Amine in Water after Injection in Animals. *Quarterly Journal of Experimental Physiology and Cognate Medical Sciences*, **34,** 279-284.

Fox, N. C., Blay, N., Greenwood, A. G., Wise, D. & Potapov, E. 2005. Wounding rates in shooting foxes (Vulpes vulpes). *Animal Welfare*, **14,** 93-102.

Fuhr, I., Bransford, A. & Silver, S. 1948. Sorption of fumigant vapors by soil. *Science*, **107,** 274-275.

Gajdusek, D. C. & Luther, G. 1950. Fluoroacetate Poisoning - a Review and Report of a Case. *American Journal of Diseases of Children*, **79,** 310-320.

Gigliotti, F., Marks, C. & Busana, F. 2001. Development of a Carbon Monoxide Fumigation Technique of the control of European rabbits (Oryctolagus cuniculus). In: *12th Australian Vertebrate Pest Conference*. Melbourne.

Ginsberg, M. D. 1985. Carbon-Monoxide Intoxication - Clinical-Features, Neuropathology and Mechanisms of Injury. *Journal of Toxicology-Clinical Toxicology*, **23,** 281-288.

Ginsberg, M. D. & Myers, R. E. 1974. Fetal Brain-Damage Following Maternal Carbon-Monoxide Intoxication - Experimental Study. *Acta Obstetricia Et Gynecologica Scandinavica*, **53,** 309-317.

Grant, W. 1986. *Toxicology of the eye*. Springfield (IL).

Green, C. 1987. *Animal anaesthesia*. London: Laboratory Animals LTD.

Gregory, N. G., Milne, L. M., Rhodes, A. T., Littin, K. E., Wickstrom, M. & Eason, C. T. 1998. Effect of potassium cyanide on behaviour and title to death in possums. *New Zealand Veterinary Journal*, **46,** 60-64.
Guale, F. G., Stair, E. L., B.W., J., Edwards, W. C. & Haliburton, J. C. 1994. Laboratory diagnosis of zinc phosphine poisoning. *Veterinary and Human Toxicology*, **36,** 517-519.
Guthery, F. S. & Beasom, S. L. 1978. Effectiveness and Selectivity of Neck Snares in Predator Control. *Journal of Wildlife Management*, **42,** 457-459.
Hall, A. & Rumuck, B. 1986. Clinical toxicology of cyanide. *Annuals of Emergancy Medicine*, **15,** 1067.
Hanriot, M. & Gautier, A. 1897. Les chloraloses. *Archives Internationales de Pharmacodynamie et de Therapie*, **3,** 191-211.
Hansen, N. E., Creutzberg, A. & Simonsen, H. B. 1991. Euthanasia of Mink (Mustela Vison) by Means of Carbon-Dioxide (CO2), Carbon-Monoxide (CO) and Nitrogen N-2. *British Veterinary Journal*, **147,** 140-146.
Harikrishna, M. & Arun, C. 2003. Stochastical analysis for vehicular emissions on urban roads - a case study of Chennai. In: *Third International Conference on Environment and Health, Chennai, India,15-17 December, 2003.* (Ed. by Bunch, m., Madha Suresh, v. & Vasantha Kumaran, t.), pp. 158 – 163. Chennai, India: Chennai: Department of Geography, University of Madras and Faculty
of Environmental Studies.
Hart, S., Marks, C. & Staples, L. 1996a. DEN-CO-FUME® - Humane Control of foxes (Vulpes vulpes) in natal dens. In: *Humaneness and Vertebrate Pest Control* (Ed. by Marks, C. A.), pp. 58-61. Ropet Printing: Tynong North, Victoria.
Hart, S., Marks, C. A. & Staples, L. 1996b. Den-Co-Fume – Humane control of foxes (Vulpes vulpes) in natal dens. In: *Humaneness and Vertebrate Pest Control.* (Ed. by Fisher, P. M. M., C. A.), pp. 58-61. Tynong North: Ropet Printing.
Hass, G. 1956. Pathological calicification. In: *The biochemistry and physiology of bone* (Ed. by Bourne, G.), pp. 767-810. New York: Academic Press.
Hayward, J. S. & Lisson, P. A. 1978. Carbon-Dioxide Tolerance of Rabbits and Its Relation to Burrow Fumigation. *Australian Wildlife Research*, **5,** 253-261.
Hewson, R. 1990. Victim of Myth. In: *Predation upon lambs by foxes in the absence of control*. London.: League Against Cruel Sports.
HSUS. 2004. Carbon Dioxide for Euthanasia and Anesthesia: Concerns Regarding Pain and Distress. pp. 29: The Humane Society of the United States.
IWGS. 2005. Report of the Independent Working Group on Snares. London: Defra.
King, D., Kirkpatrick, W., Wong, D. & Kinnear, J. 1994. Degradation of 1080 in Australian soil. In: *Proceeds of the science workshop on 1080* (Ed. by Seawright, A. & Eason, C.): The Royal Society of New Zealand Miscellaneous series 28.
Kirkwood, J. 2000. Humaneness of MAFF's Badger Despatch procedures. London: MAFF.
Klaasen, C. 1985. Non-metallic environmental toxicants: air pollutants, solvents and vapors, pesticides. In: *The pharmacological basis of therapeutics* (Ed. by Goodman, L., Gilman, A., Rall, T. & Murad, F.), pp. 1628-1650. New York: MacMillan.
Klemm, W. 1964. Carbon dioxide anaesthesia in cats. *American Journal of Veterinary Research*, **25,** 1201.

Klimmer, O. 1969. Contributions to the study of the action of phosphine (PHs). The question of the so-called chronic phosphine poisoning. *Archives of Toxicology*, **24,** 164-167.

Krebs, J., Anderson, R., Clutton-Brock, T., Morrison, I., Young, D. and Donnelly, C. 1997. Bovine Tuberculosis in Cattle and Badgers. Report to The Rt Hon Dr Jack Cunningham MP. London: Maff Publications.

Kurata, M., Suzuki, M. & Agar, N. S. 1993. Antioxidant Systems and Erythrocyte Life-Span in Mammals. *Comparative Biochemistry and Physiology B-Biochemistry & Molecular Biology*, **106,** 477-487.

Lambooy, E. 1990. Use of Carbon-Dioxide for Slaughtering Hogs - Report on a Conference Held in Heeze on January 26-27, 1990. *Fleischwirtschaft*, **70,** 1161-1166.

Lambooy, E., Roelofs, J. A. & Vanvoorst, N. 1985. Euthanasia of Mink with Carbon-Monoxide. *Veterinary Record*, **116,** 416-416.

Lambooy, E. & Spanjaard, W. 1980. Euthanasia of Young-Pigs with Carbon-Monoxide. *Veterinary Record*, **107,** 59-61.

Leach, M., Bowell, V., Morton, D. & Allen, T. 2001. Aversion to inhalation anaesthesia and euthanasia in laboratory rodents. In: *35th International Congress of the International Society for Applied Ethology* (Ed. by Garner, J., Mench, J. & Heekin, S.), pp. 159. Center for Animal Welfare: Davis, USA.

Leeson, R. & Mills, B. 1977a. Unpublished report on a survey of badger sett A.

Leeson, R. & Mills, B. 1977b. Unpublished report on a survey of badger sett B.

Lindgren, M. & Hansson, P. A. 2004. Effects of transient conditions on exhaust emissions from two non-road diesel engines. *Biosystems Engineering*, **87,** 57-66.

Littlewood, S. 1965. Report of the departmental committee on experiments in animals. London: HMSO.

Lloyd, G. 1980. *The red fox*. London.: Batsford,.

Lund, M. 1974. Calciferol as a rodenticide. *International Pest Control*, **16,** 10-11.

Macdonald, D. 1987. *Running with the fox*. London: Unwin Hyman.

MAFF. 1981. Research and Development Report: Mammal and Bird Pests 1981. In: *Reference Book 255(81)*. London: HMSO.

MAFF. 1984. Eighth report on Bovine Tuberculosis in Badgers. London: MAFF.

Magnusson, R., Nilsson, C. & Andersson, B. 2002. Emissions of aldehydes and ketones from a two-stroke engine using ethanol and ethanol-blended gasoline as fuel. *Environmental Science & Technology*, **36,** 1656-1664.

Marks, C. A., Gigliotti, F., Busana, F., Johnston, M. & Marks, C. 2004. Fox control using a para-aminopropiophenone formulation with the M-44 ejector. *Animal Welfare*, **13,** 401-407.

Marks, C. A., Hackman, C., Busana, F. & Gigliotti, F. 2000. Assuring that 1080 toxicosis in the red fox (Vulpes vulpes) is humane: fluoroacetic acid (1080) and drug combinations. *Wildlife Research*, **27,** 483-494.

Marrs, T. C. & Bright, J. E. 1986. Kinetics of Methemoglobin Production .1. Kinetics of Methemoglobinemia Induced by the Cyanide Antidotes, Para-Aminopropiophenone, Para-Hydroxyaminopropiophenone or Para-Dimethylaminophenol after Intravenous Administration. *Human Toxicology*, **5,** 295-301.

Mason, G. & Littin, K. E. 2003. The humaneness of rodent pest control. *Animal Welfare*, **12,** 1-37.

Mcdonald, R. & Harris, S. 2000. The use of fumigants and anticoagulant rodenticides on game estates in Great Britain. *Mammal Review*, **30,** 57-60.

McDonald, R. A. & Harris, S. 2000. The use of fumigants and anticoagulant rodenticides on game estates in Great Britain. *Mammal Review*, **30,** 57-64.

McIlroy, J. C. 1982. The Sensitivity of Australian Animals to 1080 Poison .4. Native and Introduced Rodents. *Australian Wildlife Research*, **9,** 505-517.

McTaggar, D. 1970. Poisoning Due to Sodium Fluoroacetate (1080). *Medical Journal of Australia*, **2,** 641-&.

Meehan, A. 1984. *Rats and Mice - Their biology and control*. East Grinstead: The Rentokil Library.

Moreland, A. F. 1974. Carbon-Monoxide Euthanasia - Chamber Concentrations Using Automobile Engine Exhaust for Euthanasia of Dogs. *Journal of the American Veterinary Medical Association*, **165,** 747-747.

Murphy, E. C., Robbins, L., Young, J. B. & Dowding, J. E. 1999. Secondary poisoning of stoats after an aerial 1080 poison operation in Pureora Forest, New Zealand. *New Zealand Journal of Ecology*, **23,** 175-182.

Neal, E. & Cheeseman, C. L. 1996. *Badgers*. London: Poyser.

Newton, I., Wyllie, I. & Freestone, P. 1990. Rodenticides in British barn owls. *Environmental Pollution*, **68,** 101-117.

NFBG. 1999. Badgers and Bovine Tuberculosis. A paper submitted to the Agriculture Select Committee by the National Federation of Badger Groups. London: National Federation of Badger Groups.

NFBG. 2002. The case for a ban on snares. A report by the National Federation of Badger Groups. London: National Federation of Badger Groups.

Ntampakis, D. & Carter, I. 2005. Red Kites and rodenticides - a feeding experiment. *British Birds*, **98,** 411-416.

Ogilvie, S. C., Hetzel, F. & Eason, C. T. 1996. Effect of temperature on the biodegradation of sodium monofluoroacetate (1080) water and in Elodea canadensis. *Bulletin of Environmental Contamination and Toxicology*, **56,** 942-947.

Oliver, A. & Blackshaw, D. 1979. The dispersal of fumigant gases in warrens of the European rabbit Oryctolagus cuniculus. *Australian Wildlife Research*, **6,** 39-55.

Pattle, R., Stretch, H., Burgess, F., Sinclair, K. & Edginton, J. 1957. The Toxicity of Fumes from Diesel Engine under Four Different Running Conditions. *The British Journal of Industrial Medicine*, **14,** 47-55.

Pelz, H. & Gemmeke, H. 1988. Methods to control the two forms of Arvicola. *European Plant Protection Organisation Bulletin*, **18,** 435-439.

Peters, R. A. 1952. The Puzzle for Therapy in Fluoroacetate Poisoning. *British Medical Journal*, **2,** 1166-1170.

Peterson, E. N., Kirby, R., Sommer, M. & Bovee, K. C. 1991. Cholecalciferol Rodenticide Intoxication in a Cat. *Journal of the American Veterinary Medical Association*, **199,** 904-&.

PSD. 1997. Assessment of the humaneness of vertebrate control agents. York: MAFF.

Raj, A. B. M. 1999. Behaviour of pigs exposed to mixtures of gases and the time required to stun and kill them: welfare implications. *Veterinary Record*, **144,** 165-168.

Raj, M. & Gregory, N. 1995. Welfare implications of gas-stunning pigs. 1: Determination of the aversion to the initial inhalation of carbon dioxide or argon. *Animal Welfare*, **4,** 273-280.

Raj, M. & Gregory, N. 1996. Welfare implications of gas-stunning pigs. 2: Stress of induction of anaesthesia. *Animal Welfare*, **5,** 71-78.

Raj, M. & Mason, G. 1999. Reaction of farmed mink (Mustela vison) to argon-induced hypoxia. *Veterinary Record*, **145,** 736-737.

Reigart, J. R., Brueggeman, J. L. & Keil, J. E. 1975. Sodium Fluoroacetate Poisoning. *American Journal of Diseases of Children*, **129,** 1224-1226.

Reynolds, J. & Tapper, S. 1995. The ecology of the red fox (Vulpes vulpes) in relation to small game in rural southern England. *Wildlife Biology*, **1,** 105-119.

Robinson, R. F., Griffith, J. R., Wolowich, W. R. & Nahata, M. C. 2002. Intoxication with sodium monofluoroacetate (compound 1080). *Veterinary and Human Toxicology*, **44,** 93-95.

Roper, T. J. & Kemenes, I. 1997. Effect of blocking of entrances on the internal environment of badger Meles meles setts. *Journal of Applied Ecology*, **34,** 1311-1319.

Roper, T. J., Tait, A. I., Fee, D. & Christian, S. F. 1991. Internal Structure and Contents of 3 Badger (Meles-Meles) Setts. *Journal of Zoology*, **225,** 115-124.

Ross, J., Page, R. J. C., Nadian, A. K. & Langton, S. D. 1998. The development of a carbon monoxide producing cartridge for rabbit control. *Wildlife Research*, **25,** 305-314.

Roughton, F. & Darling, R. 1944. The effect of carbon monoxide on the oxyhemoglobin dissociation curve. *American Journal of Physiology*, **141,** 17-31.

Savarie, P. J., Pan, H. P., Hayes, D. J., Roberts, J. D., Dasch, G. J., Felton, R. & Schafer, E. W. 1983. Comparative Acute Oral Toxicity of Para-Aminopropiophenone (Papp) in Mammals and Birds. *Bulletin of Environmental Contamination and Toxicology*, **30,** 122-126.

Schmidt, E., Newton, G., Sanders, S., Lewis, J. & Conn, E. 1978. Laetrile toxicity studies in dogs. *JAMA Journal of the American Medical Association*, **252,** 2053-2056.

Schwerma, A., Ivy, H., Friedman, H. & LaBrosse, E. 1948. A study of resuscitation from the juxtalethal effects of exposure to carbon monoxide. *Occupational medicine*, **5,** 24.

Scientific Committee on Animal Health and Welfare. 2001. The Welfare of Animals Kept for Fur Production. http://europa.eu.int/comm/food/animal/welfare/international/out67_en.pdf.

Scientific Panel on Animal Health and Welfare. 2004. Opinion of the Scientific Panel on Animal Health and Welfare on a request from the Commission related to welfare aspects of the main systems of stunning and killing the main commercial species of animals. *The EFSA Journal*, **45,** 1-29.

Semerak, C. & Bacon, L. 1930. Experimental lesions of the brain from carbon monoxide. *Archives of Pathology*, **10,** 823–839.

Sherley, M. 2004. The traditional categories of fluoroacetate poisoning signs and symptoms belie substantial underlying similarities. *Toxicology Letters*, **151,** 399-406.

Shita, A., Cornil, A. & Ectors, M. 1981. Clinical and Electroencephalographic Aspects of Acute Alphachloralose Poisoning. *Acta Clinica Belgica*, **36,** 20-24.

Shore, R. F., Birks, J. D. S. & Freestone, P. 1999. Exposure of non-target vertebrates to second-generation rodenticides in Britain, with particular reference to the polecat Mustella putorius. *New Zealand Journal of Ecology*, **23,** 199-206.

Short, J., Turner, B. & Risbey, D. 2002. Control of feral cats for nature conservation. III. Trapping. *Wildlife Research*, **29,** 475-487.

Smith, I. A. & Boyd, J. H. 1972. Another case of poisoning by alphachloralose. . *Veterinary Record*, **91,** 622.

Soholt, L. F., Yousef, M. K. & Dill, D. B. 1973. Responses of Merriams Kangaroo Rats, Dipodomys-Merriami, to Various Levels of Carbon-Dioxide Concentration. *Comparative Biochemistry and Physiology*, **45,** 455-462.

Spinelli, J. 1991. Preventing suffering in laboratory animals. *Scandinavian Journal of Laboratory Animal Science*, **18,** 159-164.

Spurr, E. B. 2000. Hen eggs poisoned with sodium monofluoroacetate (1080) for control of stoats (Mustela erminea) in New Zealand. *New Zealand Journal of Zoology*, **27,** 165-172.

Stewart, R. D. 1976. Effect of Carbon-Monoxide on Humans. *Journal of Occupational and Environmental Medicine*, **18,** 304-309.

Stone, W. B., Okoniewski, J.C. and Stedelin, J.R. 1999. Poisoning of wildlife with anticoagulant rodenticides in New York. *Journal of Wildlife Diseases*, **35,** 187-193.

Stupfel, M., Romary, F., Magnier, M. & Poliansk.J. 1971. Carbon dioxide tolerance after adaptation to hypercarbia. *Archives of Environmental Health*, **22,** 440-443.

Thompson, R. 1969. Tests with the Deckson Rabbit Fumigator. Unpublished report.

Tietz, N. 1976. *Fundamentals of clinical chemistry*. Philadelphia, Pennsylvania.

Timm, R. 1994. Norway Rats. In: *Prevention and Control of Wildlife Damage* (Ed. by Timm, R. & GE, L.), pp. B105-B120. University of Nebraska, Lincoln.

Trabes, J., Rason, N. & Avrahami, E. 1983. Computed-Tomography Demonstration of Brain-Damage Due to Acute Sodium Monofluoroacetate Poisoning. *Journal of Toxicology-Clinical Toxicology*, **20,** 85-92.

Tuyttens, F., Macdonald, D., Swait, E. & Cheeseman, C. 1999. Estimating population size of Eurasian badgers (Meles meles) using mark-recapture and mark-resight. *Journal of Mammalogy*, **80,** 950-960.

Van Oettingen, W. 1941. Studies on the mechanism of carbon monoxide poisoning as observed in dogs aneasthatized with sodium amytal. *US Public Health Bulletin*, **274,** 1-50.

Van Zutphen, L. F. M., Baumans, V. and Beynan, A.C. (eds.). 1993. *Principles of laboratory animal science: A contribution to the humane use and care of animals and to the quality of experimental results.* Amsterdam: Elsevier Science Publishers,.

Weinbroum, A. A. 2005. Pathophysiological and clinical aspects of combat anticholinesterase poisoning. *British Medical Bulletin*, **72,** 119-133.

Winston, J. M. & Roberts, R. J. 1978. Influence of Increasing Age on Lethality Induced by Carbon-Monoxide or Hypoxic Hypoxia. *Biology of the Neonate*, **34,** 199-202.

Woodroffe, R., Bourne, F. J., Cheeseman, C. L., Cox, D. R., Donnelly, C. A., Gettinby, G., McInerney, J. P. & Morrison, W. I. 2005a. Welfare of badgers (Meles meles) subjected to culling: development and evaluation of a closed season. *Animal Welfare*, **14,** 19-25.

Woodroffe, R., Bourne, F. J., Cox, D. R., Donnelly, C. A., Gettinby, G., McInerney, J. P. & Morrison, W. I. 2005b. Welfare of badgers (Meles meles) subjected to culling: patterns of trap-related injury. *Animal Welfare*, **14,** 11-17.

Zimmerman, M. 1983. Ethical guidelines for investigations of experimental pain in conscious animals. *Pain*, **16,** 109-110.