THE HUMANENESS OF RODENT PEST CONTROL

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Abstract *Animal Welfare 2003, 12*: 1-37

Rat and mouse control methods potentially affect the welfare of many millions of animals every year. Here, the humaneness of the methods used in the UK and the USA is assessed in terms of their speed and mode of action, the appearance and behaviour of affected animals, experiences of human victims, long-term effects on animals that survive exposure, and welfare risks to non-target animals. Several methods emerge as relatively humane: cyanide, alpha-chloralose, electrocution traps and well-designed snap traps all usually kill swiftly and with little distress. Preventative methods such as rodent-proofing are also humane, as well as an essential — and probably under-used — component of effective control. However, anticoagulant poisons, the most common means of controlling rodents, generally take several days to kill, during which time they cause distress, disability and/or pain. Sub-lethally affected animals are also likely to experience haemorrhages and their sequelae, and carnivores feeding on affected rodents may be secondarily poisoned. The acute rodenticides zinc phosphide and calciferol are also generally inhumane, the former typically causing severe pain for several hours, and the latter, pain and illness for several days. Sticky boards, to which rodents become adhered by the feet and fur until they are killed or simply eventually die, also raise very serious welfare concerns. This evidence highlights remarkable paradoxes in the way society treats different classes of animal, and argues for more education, legislation and research targeted at reducing the vast numbers of rodents currently killed inhumanely.

Keywords: *animal welfare, alpha-chloralose, anticoagulants, cyanide, humane rodent pest control, sticky boards, zinc phosphide*

Introduction

Many millions of rodents are killed each year as pests, yet the humaneness of the methods used receives little attention. Data do exist, however, on the likely suffering caused by pest control methods, collected from studies of animals poisoned under laboratory conditions, from clinical accounts of human accidental poisonings or suicides, and from the applied pest control literature itself. This review therefore aims to evaluate the welfare problems caused by rodent control, make recommendations for best practice, and identify future research priorities.

The need for rodent control is unquestionable. Rodents have long been commensal with humans, and they are enormously successful because of their impressive reproductive rates, omnivory, and specialised adaptations for gnawing (eg Meehan 1984). Annually, they spoil or destroy billions of dollars' worth of crops, as well as eggs, hatchlings and stored

foodstuffs (eg MAFF 1996; McDonald & Harris 2000). Thus annual losses in the USA alone were once put at $900 million, and now may well be higher (Meehan 1984). It has even been estimated that between a fifth and a third of the world's food supply never reaches the table because of losses to rodents (Corrigan 1995). This is the main reason for rodent control, and it can be extremely effective: in the Philippines, for example, rat control reduced annual rice losses from $36 million to $3.5 million (Proctor 1994). Rodents are also controlled to prevent damage to buildings and to inhibit the spread of diseases such as salmonellosis, fowl cholera, Weil's disease, bubonic plague, and many others (eg Meehan 1984; MAFF 1996; Macdonald *et al* 1999; Randall 1999). In addition, in New Zealand, the Seychelles, the Galapagos islands and other locations, controlling rodents is vital for the protection of indigenous flora and fauna (eg Eason & Spurr 1995; Gillies & Pierce 1999; Macdonald *et al* 1999; Thorsen *et al* 2000).

Rodent control is thus a vast and important exercise, and in the USA alone its annual cost has been estimated at over $300 million (Corrigan 1995). By far the most important targets are the house mouse (*Mus musculus domesticus*), Norway rat (*Rattus norvegicus*), and ship or roof rat (*Rattus rattus*) (eg Meehan 1984). House mice and Norway rats are found on every continent except Antarctica (UCMP 2001), and the house mouse is said to be the most widespread mammal on earth (Meehan 1984; DellPest 2001). One can appreciate the resultant scale of rat and mouse control from some recent estimates: several million (perhaps as many as twenty million) are killed annually in the UK alone (Fox & Macdonald 1999); over 1.7 million house mice were killed in twelve months in just one Asian city, Hanoi (Reuters 2000); and Australian farmers may kill as many as 70 000 mice in a single afternoon during mouse 'plagues' (Corrigan 1995). Rat and mouse control thus potentially affects the welfare of many millions of animals.

Assessing humaneness

Many different techniques are used to control rats and mice (see eg Meehan 1984), but this review considers only those legal in both the UK and USA. These are, first, several ingested bait poisons, or 'contact powders' ingested on grooming (anticoagulants, zinc phosphide, calciferol and related compounds, and alpha-chloralose); second, the fumigant poisons sulphur dioxide, carbon dioxide (CO_2), phosphine, and cyanide gas; and, following this, several non-poison systems — sticky boards, live traps, snap traps, electrocution traps, cellulose-based lethal feeds, and various techniques for repelling or excluding pests. To assess the humaneness of each of these methods, we considered the degree of pain, discomfort or distress caused, the length of time for which rodents are conscious and displaying clinical signs of poisoning, and the effect on any individual that escapes and survives. A method that causes the minimum number of symptoms before rapidly inducing unconsciousness or death, with no lasting ill effects on surviving animals, would thus be humane; in contrast, a method that causes severe and/or prolonged pain or distress, and leaves surviving animals ill or disabled, would be judged inhumane. We also consider the risk of poisoning of non-target animals, since this could obviously influence their welfare, and we summarise the main practical pros and cons of different approaches, as humane methods are unlikely to be widely adopted if impractical or ineffective.

Welfare can be assessed using measures derived from animals unanimously held to be experiencing stress or pain and/or from humans experiencing strong negative emotions (eg Mason & Mendl 1993; Mason 2001). In the current study, the evidence for pain or discomfort was of three types. Humans and rodents are likely to feel pain and anxiety in a broadly similar way (eg Bateson 1991; see also rodent-based pharmacological research on

anxiolytics and analgesics). The first type of evidence used in this review therefore comprised reports from poisoned humans such as suicide victims (eg Kruse & Carlson 1992), cases associated with Munchausen syndrome (eg Chua & Friedenberg 1998), and people exposed accidentally through industrial malpractice and the like (eg Inoue 1993; Lam & Lau 2000). The second type of evidence used here was the nature of the lesions or pathologies induced in rodents by the agent, from which clinicians can judge the degree of associated pain (eg Scott 1969; Kirkwood *et al* 1994; Broom 1999; Littin *et al* 2000). The third type was evidence from experimentally poisoned rodents, such as changes in behaviour and reactivity. Such studies typically catalogue inactivity, listlessness, depressed reactivity, altered appearance (eg unfocussed staring, spiky coat, hunched posture), resting outside the nest, abnormal breathing, and reduced eating and drinking leading to weight loss and dehydration (eg Rowsell *et al* 1979; Desheesh 1983; Cox & Smith 1992; Littin *et al* 2000) — behavioural changes validated by comparison with diseased or injured conspecifics, or analgesic-treated controls (eg Kirkwood *et al* 1994), and which in laboratory-research rodents would be held to indicate moderate to substantial pain (eg FELASA 1994; Wolfensohn & Lloyd 1998).

1) Ingested poisons

All ingested poisons have several welfare issues in common. First, individual animals ingest the poison while foraging, and thus when adult females are killed any dependent pups in the nest will be left to die of dehydration and starvation. Second, accidental poisoning of non-target animals can occur, although this can be reduced to a minimum with well-designed bait stations, appropriate dyes and lures, and bitter substances that deter non-rodents. Third, secondary poisoning can affect animals that eat dead or dying rodents, although the risks of this differ enormously between compounds. Fourth, the mode of action, the dose consumed, and the way the poison is absorbed, distributed, metabolised and excreted (ie its toxicokinetics) will all influence the intensity and/or duration of suffering. Anything that influences these features can therefore affect humaneness. Potential factors here include species, age, diet, health and the duration of exposure to the poison (eg Clarke & Clarke 1967; Brown 1980), plus various characteristics of the bait itself. Variation in such factors can help to explain variation between studies, including those we cite below. They also mean that one cannot directly extrapolate from one species to another: data are always needed from the specific species of interest, although insights from other species can still be revealing. Finally, it should be assumed that for most or all poisons, the least compromise in welfare results when as high a lethal dose as possible is consumed.

Anticoagulants

Anticoagulant poisoning is by far the most common means of rodent control (eg PSD 1997), being the basis of about 95% of rat and mouse control in the USA (Timm 1994a) and 92% of rodent control on UK arable farms (Thomas & Wild 1996, cited by McDonald & Harris 2000). Warfarin was the first important anticoagulant, but because of genetic resistance (eg Quy *et al* 1992; Smith *et al* 1994b) this is now supplemented by 'second generation' compounds such as brodifacoum, difenacoum and bromadiolone. All act by interfering with Vitamin K-1 metabolism and hence prothrombin formation and platelet-mediated clotting. The normal daily damage to blood vessels is then no longer repaired (eg Meehan 1984; Thijssen 1995) so that animals die principally from blood loss and its sequelae (eg cardiac, respiratory or kidney failure; Anderson 1980; Radostits *et al* 1999).

Anticoagulants are extremely effective and easy to use, although some protocols require repeated baiting and are therefore quite labour-intensive (eg Meehan 1984; Forage

Information System 1997; Weile 2001). Used correctly, they cause little bait shyness (eg Proctor 1994; Hyngstrom & Virchow 1996), and are also relatively cheap, a particularly important issue in the developing world (eg Kumar *et al* 1997). Furthermore, they are relatively safe if accidental poisoning occurs: their slowness of action allows several days for medical intervention (eg PSD 1997), and Vitamin K-1 and blood products are effective remedies (eg Padgett *et al* 1998; Sheafor & Couto 1999).

Humaneness

The nature, degree and duration of any suffering caused by anticoagulants depend on the site and severity of haemorrhages. This is influenced by the dose received and the exact nature of the compound, but individual predispositions also play a major role.

Mildly poisoned humans show increased bruising rates and bleeding from cuts, occasional nose and gum bleeds, blood in the faeces or urine, a pale mouth and cold gums, and general weakness (Sorex Ltd 1992; Killgerm Chemicals Ltd 1994; WHO 1995). More severe cases involve widespread haemorrhaging, usually internal (Sorex Ltd 1992; Killgerm Chemicals Ltd 1994; WHO 1995); autopsies reveal, for example, pulmonary and sub-dural haemorrhages, ovarian haematomas, multiple bleeding sites on the skin, and sub-mucosal bleeding into the lips (Palmer *et al* 1999). Medical case reports further describe bleeding from the urethra, intra-abdominal haemorrhaging, mesenteric haematomas, pleural effusions, acute renal failure, pericardial haemorrhages, haemoarthrosis, blood in the gastrointestinal tract, intra-cerebral haemorrhages, and other lesions (eg Kruse & Carlson 1992; Corke 1997).

Bleeding *per se* is not painful, but the accumulation of blood in enclosed spaces generally is (eg Yates & Smith 1989; PSD 1997). Thus, poisoned humans can experience localised muscle pain (Morgan *et al* 1996), joint pain (Kruse & Carlson 1992) and potentially severe abdominal pain caused by intra-peritoneal, mesenteric or ovarian bleeding (eg Macon *et al* 1970; Stanton *et al* 1974; Waxman & Baird 1978; Scott *et al* 1984; Kruse & Carlson 1992; Morgan *et al* 1996). Haemorrhages within the lungs, kidneys, spinal cord, orbits of eyes and gonads are also painful (reviewed in Broom 1999). Bleeding into lungs or airways can cause further distress by making breathing difficult (Broom 1999), and poisoned humans may also experience dizziness, localised reduced motor strength, the inability to urinate, and sometimes even paraplegia (eg Kruse & Carlson 1992; Morgan *et al* 1996).

Rodent data reveal similar clinical effects. Poisoned rats show external bleeding and pale extremities (Littin *et al* 2000), along with bloody diarrhoea (Scott 1969; Meehan 1984; PSD 1997). Internally, there can be multiple haemorrhages throughout the muscles and intestinal tract (Scott 1969), bleeding into the body cavities and epididymis (Rowsell *et al* 1979), and haemorrhages into the joints, lungs, other viscera and skeletal muscle (Meehan 1984). Timm (1994a) also states that subcutaneous haematomas are common. Detailed necropsies of rats poisoned with brodifacoum (Littin *et al* 2000) also revealed the following spectrum of haemorrhage sites: subcutaneous and deep tissues of the thorax (10/12 animals), limb musculature (7/12), testes (5/6 males), and stifle joints (2/12). The Pesticide Safety Directorate (PSD 1997) reports similar findings, plus gastrointestinal, orbital, intra-cranial and a variety of other haemorrhages judged "capable of producing severe pain".

Unsurprisingly, such lesions cause signs indicative of the moderate to severe pain and distress reported by humans. These include anorexia, laboured breathing, struggling movements, reduced activity, poor condition, and sometimes paralysis (eg Rowsell *et al* 1979; Meehan 1984; Cox & Smith 1992; Berny *et al* 1997; PSD 1997; Littin *et al* 2000). For example, four days after ingesting brodifacoum, rats showed reduced activity levels,

anorexia, and less use of their normal curled sleeping posture. Instead, they were frequently seen lying, or standing in a hunched posture with the abdomen tucked up and head lowered (Littin *et al* 2000). In addition, one third of the rats developed paresis and then paralysis two days before death (K E Littin, personal observation; Littin *et al* 2000). Mice also show evidence of altered behaviour: in the wild, brodifacoum-poisoned mice have been observed above ground during the day and, in one study, around 25% died above ground or half-submerged in their burrows (Brown & Singleton 1998; see also Cox & Smith 1992), indicating abnormal activity patterns consistent with illness. Anticoagulant-poisoned rodents in the wild also apparently find it more difficult to escape from predation (Berny *et al* 1997).

The symptomatic period ranges — depending on the individual, the particular anticoagulant and, to some extent, the dose — from just a few hours (in some difenacoum and brodifacoum studies) to, more commonly, an average of one to three days, with a maximum of four to five days of clinical signs (for a review, see PSD 1997). In rats poisoned with brodifacoum, for example, Littin *et al* (2000) found a mean of three days between the onset of clinical signs and death (which occurred at a mean of 7.2 days). Animals typically remain conscious during this time (eg PSD 1997): electroencephalograms (EEGs) remain normal until just prior to death (Rowsell *et al* 1979) and, in the study by Littin *et al* (2000), although even unparalysed animals lay prostrate for a mean of 11.4 h prior to death, they stayed conscious and occasionally pushed or pulled themselves along the floor. The symptomatic period is presumably reduced when times to death are shorter; in one study, for example, most poisoned rats took just one to three days to die, and some, less than 24 hours (Rowsell *et al* 1979; see also PSD 1997). However, times to death are longer than this in all other studies, being typically in the region of four to eight days (eg Gill *et al* 1994; PSD 1997; Littin *et al* 2000). Furthermore, animals ingesting lower doses can take longer still to die; for example, mice can take up to eleven days (Newton *et al* 1990), although the length of the symptomatic period has not been reported here. Thus, overall, although there can be variation, the norm is for clinical symptoms to last for several days.

For humans or companion animals that have received sub-lethal or near-lethal doses, medical care is generally required because of internal damage (eg to liver and kidneys; Meehan 1984), blood loss, and anaemia (eg Robben *et al* 1997; Sheafor & Couto 1999). Clotting times also often remain sub-optimal for weeks or even months (eg WHO 1995; Morgan *et al* 1996; Corke 1997). This suggests that sub-lethally poisoned rodents could be ill or disabled for a considerable period. Learned avoidance to the bait will also occur, although only if there is a short enough interval between intake and symptom-onset (eg Brunton *et al* 1993; Smith *et al* 1994b), further indicating the aversiveness of the poison's effects.

Risks to non-target animals

Predators generally have to eat several poisoned rodents before experiencing ill effects (eg Meehan 1984; Newton *et al* 1990; Gray *et al* 1994), and secondary poisoning is therefore said to be unlikely (eg Meehan 1984; Proctor 1994). However, the prolonged persistence of most second generation anticoagulants makes the risk a real one (eg Carter & Burn 2000; McDonald & Harris 2000; Eason & Wickstrom 2001). Dead or dying rodents found outside the nest are dangerous for a relatively long period (Cox & Smith 1992) unless they are safely removed. For example, in Norway rats, brodifacoum has a half-life in the serum of over 6.5 days (Bachmann & Sullivan 1983) and, in the liver, of 130 days (Parmar *et al* 1987). Furthermore, ingested poison can progressively accumulate in the livers of predators and scavengers (eg Eason & Spurr 1995; Shore *et al* 1999). Thus some anticoagulants reach dangerous levels even if poisoned rodents are eaten only once every few days (Timm 1994a);

buzzards, for example, can succumb if experimentally fed bromadiolone-poisoned rodents eight or even ten days apart (Berny *et al* 1997, citing Grolleau *et al* 1985). Brodifacoum is particularly dangerous as it has both a very long biological half-life and a very low LD_{50} (eg Meehan 1984; Newton *et al* 1990; Eason *et al* 1996a; Meyer 2000; Stone *et al* 1999).

Accumulated anticoagulants have been found in the stomachs and livers of many wild carnivore species, including polecats, barn owls, and red kites (Newton *et al* 1990; Shore *et al* 1996; Gillies & Pierce 1999; Shore *et al* 1999; Carter & Burn 2000; Carter & Grice 2000). Furthermore, fatal secondary anticoagulant poisoning has been implicated in the deaths of red foxes, owls, buzzards, kites, corvids and many others (Newton *et al* 1990; Proctor 1994; Berny *et al* 1997; Shore *et al* 1999; Stephenson *et al* 1999). Dogs and cats have also been secondarily poisoned, often fatally (eg Du Vall *et al* 1989; Martin *et al* 1994; Proctor 1994; Timm 1994a; Robben *et al* 1997; Padgett *et al* 1998). Brodifacoum is sometimes the culprit (eg Carter & Grice 2000) even in the UK where it should not be used outdoors (eg Meyer 2000). In addition, although the doses that cause damage are still being debated (eg Kaukeinen *et al* 2000; Littin *et al* 2002), sub-lethal secondary poisoning has been implicated in the reduced breeding success of some New Zealand owls (Stephenson *et al* 1999); and even predators that are not made ill may show increased clotting times for days, or even weeks, after ingesting a poisoned rodent (Newton *et al* 1990).

Overall, secondary anticoagulant poisoning is rare and, in general, is not a major conservation issue (eg Newton *et al* 1990; Berny *et al* 1997). However, it clearly raises ethical and welfare questions, because accidentally affected animals that receive a high enough dose are likely to experience the same symptoms as target rodents. Thus secondarily poisoned dogs display physical weakness and lethargy, coughing and respiratory distress, pallor, anorexia, and ventral haematomas as well as many internal haemorrhages (eg Du Vall *et al* 1989; Robben *et al* 1997; Sheafor & Couto 1999), while wild animals can develop haemorrhages in a range of potentially painful or distressing sites (Stone *et al* 1999; Carter & Burn 2000), such as subcutaneously and within the lungs, muscle tissue, brain and pericardial sac.

Zinc phosphide

Although used relatively little in the UK (eg PSD 1997), zinc phosphide is, worldwide, the most commonly used rodenticide after the anticoagulants, particularly in developing countries (Meehan 1984). An acute poison, it kills after a single dose (eg Meehan 1984; MAFF 1996; Forage Information System 1997) and acts by producing phosphine gas in the stomach, which upon absorption is a potent inhibitor of cytochrome oxidase (eg Meehan 1984; Timm 1994a) with additional direct cytotoxic effects (eg Rowsell *et al* 1979; Rodenberg *et al* 1989). Organs with the greatest oxygen requirements, such as the heart and brain, are thus particularly sensitive to damage (Guale *et al* 1994). Death usually occurs as a result of cardiac and respiratory failure, preceded by pulmonary oedema and hypotension (eg Rodenberg *et al* 1989; Gupta *et al* 1995; PSD 1997). The phosphine can also damage tissue in other organs such as the liver and kidneys (Rodenberg *et al* 1989; Timm 1994a; Siwach *et al* 1995; Guale *et al* 1994).

Zinc phosphide is one of the more effective acute rodenticides; it is particularly useful when a rapid population reduction is required (eg Meehan 1984; Pathak & Saxena 1997) and, experimentally, can cause higher death rates than bromadiolone (Malhi *et al* 1994). It does have some practical disadvantages, however. Bait shyness is a problem (eg Timm 1994b), and pre-baiting is therefore needed (eg Meehan 1984; Sugihara *et al* 1995; MAFF 1996; Forage Information System 1997). Also, zinc phosphide becomes less effective if weathered,

especially if damp (eg Meehan 1984; Koehler *et al* 1995). It can therefore sometimes be less useful in practice than anticoagulants (eg Sugihara *et al* 1995; Amjad *et al* 1999; Mathur 1997). An additional disadvantage is that zinc phosphide has no antidote (Meehan 1984), treatment for accidental poisoning being primarily supportive (eg Rodenberg *et al* 1989; Andersen *et al* 1996).

Humaneness

Poisoned humans experience liver, kidney and heart damage (eg Timm 1994a), death resulting from cardiac failure (eg Andersen *et al* 1996), circulatory shock (eg Misra *et al* 1988; Chugh *et al* 1989, 1998), pulmonary oedema (eg Rodenberg *et al* 1989; Gupta *et al* 1995) and/or renal failure (eg Misra *et al* 1988). Autopsies generally further reveal cardiac and pulmonary congestion, hepatic engorgement and gastrointestinal mucosal congestion (eg Misra *et al* 1988; PSD 1997). Organ damage is seen in some cases, such as petechial haemorrhaging (Misra *et al* 1988; Siwach *et al* 1995). Early symptoms in humans include diarrhoea and vomiting (eg Timm 1994a), both often very severe, black and smelling of phosphorus (Timm 1994a; Andersen *et al* 1996; Chugh *et al* 1998). 'Excitement' (Timm 1994a) and respiratory distress are also common (eg Chugh *et al* 1989, 1998; Gupta *et al* 1995; PSD 1997). Victims report experiencing nausea, headaches, vertigo, a feeling of coldness, chest tightness and abdominal or stomach pain (Misra *et al* 1988; Rodenberg *et al* 1989; Timm 1994a; PSD 1997). As the poisoning develops, this abdominal or retrosternal pain tends to become burning and very severe (eg Misra *et al* 1988; Gupta *et al* 1995; Andersen *et al* 1996; PSD 1997; Chugh *et al* 1998).

Animal studies indicate similar effects. Death is generally due to heart failure, with internal organ congestion (Meehan 1984). Necropsies of poisoned rodents have shown acute catarrhal enteritis in the duodenum (Rowsell *et al* 1979) and gastric ulcers consistent with chemical corrosion, along with blood in the trachea and lungs, and coronary and hepatic congestion (PSD 1997). Signs of poisoning are also similar to those of humans (except that rodents cannot vomit), and include respiratory distress (eg Meehan 1984; Guale *et al* 1994; Sterner & Mauldin 1995; PSD 1997), diarrhoea (Scott 1969), excitation (Guale *et al* 1994), and lassitude and depression (Meehan 1984; Guale *et al* 1994; Sterner & Mauldin 1995; PSD 1997). Poisoned rodents may kick at their abdomens with their hind feet (Rowsell *et al* 1979) and show postural changes indicative of pain (PSD 1997). In one study of rats housed in pens, poisoned rats were located in open as well as concealed areas (Malhi *et al* 1994), another sign of altered activity prior to death. Final symptoms can include convulsions and paralysis (Timm 1994a; PSD 1997) and rodents typically die in a prone position, legs and tails outstretched (Timm 1994a).

Times taken to die vary between studies, with an almost bimodal distribution of death times that is presumably dose-related and perhaps reflects the poison's two main actions. Deaths seem to be either rapid (ie well under 24 h) or more prolonged (eg 24–72 h). Rowsell *et al* (1979) report that rats generally die within 8 h of ingestion, the Pesticide Safety Directorate (PSD 1997) that death could occur within 5 h, and Sterner and Mauldin (1995) report that voles die within 4–12 h of baiting. The FAO (1999) similarly reports that most rodents die 8–24 h after ingestion, but also that a few may die on the second day of baiting. Meehan (1984) and Timm (1994a) also report that most die within 24 h or even 'overnight', but that some deaths can be delayed for several days, in which case liver damage occurs. For example, Malhi *et al* (1994) report that death occurs in rats in one to three days.

Clinical signs generally appear rapidly (eg Rowsell *et al* 1979). For example, a reduction in feeding may be apparent in 15 min or less (Meehan 1984), reduced activity within 1 h

(Sterner & Mauldin 1995), and behaviours such as abdomen-kicking 3–6 h after ingestion (Rowsell *et al* 1979). Thus, although some experimental studies suggest no signs of distress until what is described as a short, terminal "death agony" (Timm 1994a), most studies indicate a symptomatic period of several hours (eg Rowsell *et al* 1979; PSD 1997), with intoxication occurring over several days in those rodents that do not die overnight (Timm 1994a).

The poison may cause little lasting harm to sub-lethally dosed subjects. Human cases generally involve medical interventions that may prevent early deaths and allow humans to survive tissue damage (eg Misra *et al* 1988; Siwach *et al* 1995) that would presumably slowly kill untreated animals (cf eg Timm 1994a). Hence, in humans, toxic effects may last for several days (PSD 1997). However, victims who are alive after three days are said to recover completely (Timm 1994a), as they eliminate the phosphine via the lungs or kidneys (Gupta *et al* 1995; Chugh *et al* 1996). Likewise, in animals without medical support, there is evidence that those that manage to survive the illness period have no long-term sequelae (PSD 1997). However, for as long as six months afterwards, these animals will display a powerful learned aversion to foodstuffs associated with the poison (Shepherd & Inglis 1993).

Risks to non-target animals

There is some potential for secondary poisoning because of the compound's persistence for several days in poisoned rodents' guts (Guale *et al* 1994; Timm 1994a; MAFF 1996; Wildlife Damage Review 2001). However, because it does not accumulate in their muscles or other tissue (Timm 1994a; Sterner & Mauldin 1995), nor within the predators themselves, the risk is low. Thus as long as any single dose eaten is not too great, predators will experience no ill effects even if fed poisoned rodents over several days (Meehan 1984).

Calciferol and cholecalciferol

A form of Vitamin D, calciferol (also called ergocalciferol) interferes with calcium homeostasis, causing the mobilisation of calcium from the bone matrix and increased uptake in the gut (Meehan 1984; Timm 1994a; PSD 1997). Victims usually die from hypercalcaemia, kidney failure, and/or the side-effects of soft-tissue calcification, particularly metastatic calcification of the blood vessels and nephrocalcinosis (Meehan 1984; MAFF 1996; PSD 1997). Cholecalciferol (Vitamin D_3) acts in the same way, so will not be discussed separately.

Calciferol is an acute poison, and can be readily formulated as a one-feed bait requiring no pre-baiting (eg Eason & Wickstrom 2001; Feral Control 2000). It is also potentially very effective (Meehan 1984; Brunton *et al* 1993; Timm 1994b). This makes it a fairly common rodenticide in some countries; for example, it is used on 3–4% of UK arable farms (Thomas & Wild 1996, cited by McDonald & Harris 2000). However, palatability problems and degradation can reduce its effectiveness (Meehan 1984; Brunton *et al* 1993), and it is therefore less suitable for outdoor use, especially when damp (MAFF 1996). It is also not recommended for use against rats (MAFF 1996) and is relatively expensive (Eason & Wickstrom 2001). A final disadvantage is that when treating accidental poisoning, the hypercalcaemia and other symptoms are often difficult to reverse (eg Hatch & Laflamme 1989; Fooshee & Forrester 1990; Eason & Wickstrom 2001).

Humaneness

Human data tend to come from chronic low-dose poisoning, such as side-effects from medicinal uses, rather than acute high-dose poisoning. As with all pesticides, dose can affect

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symptomology (especially progressive calcification in this case) and hence the humaneness implications of such data for lethally poisoned rodents. However, they are presented here because chronic low-dose human poisoning may be at least illustrative of the welfare impact of sub-lethal poisoning. The primary cause of illness or death in these cases is kidney failure, a secondary outcome being haemorrhage following the rupture of calcified blood vessels (eg PSD 1997). For example, a woman who took calciferol every day for two months developed mental and renal impairment (Meehan 1984), and another patient, permanent renal damage (Paterson 1981). Fatal cases have also involved the calcification of heart and lung tissue, as well as the arteries and renal tubules (PSD 1997). Victims typically show vomiting, anorexia, weight loss, irritability and depression (Meehan 1984; PSD 1997), and experience severe, frequent (if transient) headaches, nausea, and pain and intense discomfort in other parts of the body (PSD 1997).

The effects of acute poisoning are better documented in companion and pest animals than in humans, but appear similar. In cats and dogs, clinical signs of poisoning include lethargy and severe depression, anorexia, vomiting and polydipsia (Moore *et al* 1988; Talcott *et al* 1991). Internally, poisoned dogs show gastrointestinal haemorrhage, myocardial necrosis, and calcification of vascular walls (Gunther *et al* 1988), while those with the most severe clinical signs also show calcification of the kidneys and stomach (Rumbeiha *et al* 1999). Poisoned horses show leg stiffness, recumbency, weakness, anorexia and substantial weight loss and, internally, extensive mineralisation of cardiovascular and other soft tissues (Harrington & Page 1983). Eason (1993) reported that rabbits receiving a lethal dose of cholecalciferol lost their appetite and about 10–20% of their body weight before dying two to four days after dosing. Brushtail possums dosed with cholecalciferol experienced widespread mineralisation and died four to seven days after dosing, probably from heart failure, while sub-lethally affected animals stopped eating and became constipated three or four days after dosing (Jolly *et al* 1993).

Poisoned rodents display similar lesions and signs of pain and dysfunction. For example, in mice given intra-peritoneal cholecalciferol (Hatch & Laflamme 1989), these included ocular squinting, a reluctance to move, lethargy, weakness, anorexia, hunched posture, rough coat, and dehydration, followed at larger doses by tremors and coma. In another study, high doses led to appetite loss, listlessness, piloerection, hunched posture, lack of reaction to external stimuli, weight loss, priapism, and frequent micturition (PSD 1997). Anorexia is also described in much of the rodent control literature (eg Meehan 1984; Timm 1994a; Feral Control 2000). Internally, blood vessel calcification can also be seen in poisoned rodents (Meehan 1984), along with calcification of internal organs (Brunton *et al* 1993, citing Sebrell & Harris 1971). In the laboratory, this poison can therefore result in learned avoidance, although this is not seen in field conditions because of the relatively long delay (see below) between ingestion and the onset of clinical signs (reviewed in Brunton *et al* 1993).

Some workers state that rodents usually experience acute symptoms within 14 h (Feral Control 2000); however, others put the onset period at a little later, most rodents becoming ill and ceasing to eat after 24–48 h (Meehan 1984; Saini & Parshad 1992; Brunton *et al* 1993; Sheikher & Jain 1995; PSD 1997). In a study of mice by Hatch and Laflamme (1989), the time to the onset of illness after a lethal calciferol injection was two to four days. Meehan (1984) states that tissue calcification can be seen from two days onward. Death, however, usually takes a few days longer. For example, in laboratory bait studies, times until death range from three to eleven days in mice, two to ten days in black rats, and one to thirteen days in Norway rats (Meehan 1984; Hatch & Laflamme 1989; Saini & Parshad 1992; Sheikher & Jain 1995), with four to five days an approximate average (PSD 1997). In field

conditions, typical times to death are also three to five days, sometimes longer (Timm 1994a; PSD 1997). Thus the period for which animals show clinical signs is several days long. For instance, in one study of house mice, the symptomatic period for lethally poisoned animals averaged four to six days depending on dose (Hatch & Laflamme 1989), and in another it ranged from two to seven days (Sheikher & Jain 1995). More recently, the mean symptomatic period has been put at two days for rats and three for mice (with a maximum of 10 days; PSD 1997).

For victims of accidental poisoning, medical care is often needed, particularly because of kidney failure. Although animals ingesting low levels may recover fully (Moore *et al* 1988; Hatch & Laflamme 1989), this is often a gradual process (cholecalciferol-poisoned rabbits take up to two weeks to recover, for example; Eason 1993), and in many individuals it only occurs with intervention (eg Fooshee & Forrestor 1990; Scheftel *et al* 1991; Rumbeiha *et al* 1999). Even then, companion animal cases show that serum calcium may not stabilise for weeks (Fooshee & Forrestor 1990), and long-term effects (for example in human cases) are known to include permanent renal damage (eg Paterson 1981). Thus sub-lethally poisoned rodents are likely to be ill and anorexic for several days at least (some studies suggest 12–14 days [Hatch & Laflamme 1989; Brunton *et al* 1993, citing Sebrell & Harris 1971]), and very possibly left with longer-term sequelae.

Risks to non-target animals

Calciferol brings low secondary poisoning risks, as rodents tend to cease eating after consuming relatively small amounts (eg Zeinelabdin & Marsh 1991); it is also quickly metabolised within the rodent's body (Proctor 1994; Stone *et al* 1999; Eason & Wickstrom 2001; Feral Control 2000; Wildlife Damage Review 2001). For example, Eason *et al* (1996b) found that cats eating the carcasses of possums poisoned with cholecalciferol experienced only slightly elevated serum calcium concentrations, and no changes in appetite or body weight. Thus secondary toxicity from feeding on poisoned rodents has not been demonstrated (Timm 1994a).

Alpha-chloralose

Alpha-chloralose is a narcotic previously used as a hypnotic, sedative and general anaesthetic in human and animal medicine (eg Scott 1969; Meehan 1984). It is now used only as an anaesthetic in some research work, because it causes unconsciousness without baroreceptor depression (eg Talman *et al* 1981; Holzgrefe *et al* 1987; McKenzie *et al* 1996). It is used more commonly as a hypnotic and immobilising agent for wild birds (eg Timm 1994a; Broom 1999), and also as a rodenticide. In rodents, it differs from the previous poisons in acting centrally: it depresses brain activity, thereby retarding metabolism, slowing the heart and respiration, and lowering the body temperature so that hypothermia results (Meehan 1984; Timm 1994a; MAFF 1996; Pest Control Portal 2001). The body temperature of an unconscious mouse can fall by as much as 20°C (Meehan 1984), and this is primarily what kills poisoned rodents, although respiratory failure can also occur (Timm 1994a).

Alpha-chloralose is potentially highly effective and efficient, and can give excellent, rapid results without pre-baiting, even in the presence of alternative food (eg Meehan 1984). It has been widely used in the UK (Meehan 1984), although not on largely outdoor sites such as arable farms (eg McDonald & Harris 2000). Accidental poisoning is easy to treat, especially in small animals, which primarily need simply to be kept warm (eg Meehan 1984; PSD 1997). Although poisoned and unconscious humans need medical attention to ensure airways stay clear (PSD 1997; Tox-In 2000; Mallinckrodt Baker Inc 2000), the risks to relatively

large species are generally low because of their slow rate of heat loss (Pest Control Portal 2001). However, alpha-chloralose is said to be more expensive than anticoagulants; and mice can also build up tolerance after several days' exposure (Meehan 1984). Its reliance on hypothermia also restricts its use to ambient temperatures below 15–16°C (Meehan 1984; Timm 1994a; Pest Control Portal 2001), with best results occurring below 13–14°C (MAFF 1996). Its reliance on hypothermia also makes it unsuitable for rats because of their smaller surface area:volume ratio (eg MAFF 1996). Furthermore, because of perceived risks of accidental or secondary poisoning (although some sources describe this rodenticide as being comparatively safe; eg PSD 1997; Pest Control Portal 2001), in the UK it is classified as unsuitable for outdoor use (MAFF 1996).

Humaneness

In humans, this agent causes no pain, although a little physical discomfort. At low doses it causes inebriation (Thomas *et al* 1988; PSD 1997; Tox-In 2000), and at higher doses it may cause bronchial hypersecretion (Tox-In 2000), coughing and shortness of breath, headache, nausea, vomiting and dizziness (PSD 1997; Kintz *et al* 1999; Mallinckrodt Baker Inc 2000). In poisoned humans and anaesthetised animals, muscle twitches and convulsions may occur, but these generally happen during, not prior to, coma (Manzo *et al* 1979; Thomas *et al* 1988; PSD 1997; Wolfensohn & Lloyd 1998).

In rodents, there are similarly few signs of real distress. Symptoms include inebriation, hyperactivity, and a staggering or uncoordinated gait (Scott 1969; Meehan 1984; Timm 1994a). There can also be posterior weakness, prostration, increased salivation, an increased sensitivity to touch or sound (Timm 1994a; Pest Control Portal 2001), and myoclonic twitches (Timm 1994a; PSD 1997). This symptomatic period progresses rapidly (eg feeding ceases 10–15 min after the onset of ataxia; Timm 1994a), and is also generally very short (eg Scott 1969): indeed, mice can be unconscious within 15 min of eating the poison (Meehan 1984). Full unconsciousness is also preceded by sleepiness (PSD 1997) and an apparently reduced sensitivity to pain (Pest Control Portal 2001). The general non-aversiveness of this agent is further suggested by observations that after recovering, mice often return to the bait (Scott 1969). In rats, however, the poison may be less humane, especially if used at higher ambient temperatures, as it can then cause convulsions while the animals are still conscious; non-lethally poisoned rats can thus develop learned aversion to the bait (PSD 1997).

Animals ingesting non-fatal levels rapidly recover to full normal functioning (Manzo *et al* 1979; Meehan 1984; Wolfensohn & Lloyd 1998). For example, in humans, neurological disturbances generally resolve within 24–36 h or less (Thomas *et al* 1988; Tox-In 2000; PSD 1997). Thus, at air temperatures above 15°C, poisoned mice may simply regain consciousness and recover (Timm 1994a).

Risks to non-target animals

Secondary poisoning is possible because rodents become unconscious rapidly and so remain above ground. Secondary poisoning can also be rapid if no intervention occurs (eg Wildlife Damage Review 2001). Thus, secondary poisoning has killed buzzards (Van Nie 1975) and red kites (Carter & Burn 2000, citing Sharp & Hunter 1999). However, the risks are low for larger predators, as the compound is non-cumulative (Pest Control Portal 2001) and such animals have relatively low rates of heat loss even if they become unconscious (Pest Control Portal 2001). Thus overall, such risks appear low, and are potentially soluble by collecting rodent carcasses (an easier task for this poison than for others, as the rodents die within a fairly short time window, and the most dangerous carcasses are those above ground and

uncovered, ie visible to avian scavengers). Also, as discussed above, the treatment for accidental poisoning is fairly simple.

2) Fumigant poisons

Correctly carried out, fumigation is the most efficient rodent control method (Meehan 1984). In welfare terms, it also has two particular advantages: first, affected animals are not dangerous to predators, so that secondary poisoning risks are negligible (eg Wildlife Damage Review 2001); and second, all animals within a burrow system are poisoned simultaneously so that dependent young are killed with their mothers, instead of being left to die in the nest. However, some animals may be exposed to sub-lethal doses, with immediate and long-term harmful consequences. In addition, fumigation is usually expensive (requiring the use of special equipment by a licensed operator), burrow systems need to be found, and for safety reasons this method cannot be used in domestic settings, near livestock, or when soil is sandy or loose, as accidental poisoning is a real risk (eg Meehan 1984; Proctor 1994; MAFF 1996; Wildlife Damage Review 2001).

Sulphur dioxide

The burning of sulphur to produce sulphur dioxide has not been recommended in the UK for decades (Meehan 1984), but one sulphur-containing product does remain provisionally approved (PSD 1997), and in other countries, such as Canada, 'sulphur dioxide bombs' are still used (eg Saskatchewan Agriculture and Food 1998).

Sulphur dioxide is converted into sulphurous and sulphuric acid in contact with the mucous membranes. It causes a range of damage to the airways and lungs, including changes to the structure of the epithelium, laryngeal spasm, bronchoconstriction, haemorrhage, oedema and accumulation of blood and fluid in the airways and lungs, collapsed lungs, emphysema, and eventually respiratory arrest (Miller *et al* 1981; Osweiler *et al* 1985; Sittig 1991; Budavari *et al* 1996; Drazen *et al* 1999). Hence, death is likely to be due to asphyxia (Rowsell *et al* 1979).

Humaneness

Although data are limited, the likelihood of pain is very high because of the conversion of the gas into acid on the mucous membranes of the eyes, mouth and respiratory tract (eg Rowsell *et al* 1979). Even low concentrations still cause minor lesions in the respiratory tract and behavioural signs of distress (PSD 1997). Bleeding and secretions into lungs or airways can cause further distress by making breathing difficult (Osweiler *et al* 1985). Death, probably by asphyxiation as discussed above, occurs between 20 min and 5 h after exposure. It is not preceded by unconsciousness.

This gas is presumably also distressing to non-lethally affected animals, but does not cause lasting harm: they may suffer damage to their mucous membranes, but no long-term pathology (PSD 1997).

Carbon dioxide

Carbon dioxide (CO_2) is used in some enclosed indoor sites, especially cold stores (eg Meehan 1984). At concentrations above 40%, it kills by causing a lack of oxygen (anoxia), leading to the loss of normal brain function and eventually respiratory failure, and also via metabolic effects mediated by acidosis, including acidosis of the cerebrospinal fluid (eg Danneman *et al* 1997; Raj *et al* 1997; HSUS 2002).

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Humaneness

As with most agents, the humaneness of CO_2 depends on its concentration. High concentrations have rapid effects; for example, cats lose consciousness in 60% CO_2 within 45 s and experience respiratory arrest within 5 min (AVMA 2000), while rats become unconscious within 2–3 min if the gas is at 100% concentration (PSD 1997; Danneman *et al* 1997). Hackbarth *et al* (2000) also report tachypnoea, but no hormonal or behavioural changes suggestive of distress when rats are exposed to increasing CO_2 (a flow rate of 6 l min^{-1} leading to 55.5% CO_2 after 2 min of exposure). Similarly, Smith and Harrap (1997) saw dyspnoea, but no behavioural evidence of distress in rats introduced into 75% CO_2, or exposed to increasing CO_2 (10 l min^{-1} leading to 45% CO_2 after 2 min and peaking at 80% after 7 min). Reports such as these underlie the use of CO_2 as a recommended means of killing laboratory animals, especially when used at concentrations of 70% or greater (eg AVMA 2000).

However, death can be far more prolonged at low concentrations or flow rates, for example taking 16 min to induce unconsciousness at a concentration of 50% (Danneman *et al* 1997) and 2–24 h to kill rats in pest control situations (Meehan 1984). Furthermore, in a study using concentrations of 50–100%, behavioural signs of aversion, and oedema and haemorrhaging into the lungs, were more severe at these lower concentrations (Danneman *et al* 1997), perhaps because the gas was inhaled for longer before death. CO_2 is in fact strongly aversive to a range of species including humans, pigs, chickens and mink (Raj & Gregory 1995a,b; Danneman *et al* 1997; Cooper *et al* 1998) — as well as rats and mice themselves (Leach *et al* 2001). This may be due to the production of carbonic acid when CO_2 contacts the mucous membranes (AVMA 2000), irritating them and causing discomfort (Lucke 1979). To humans, high concentrations of CO_2 thus cause a burning, choking sensation that is highly unpleasant (Raj & Gregory 1995a; Danneman *et al* 1997). However, even concentrations of 35–40% can be painful on the human mucosa (HSUS 2002, citing Anton *et al* 1992), while levels as low as 25% are aversive to laboratory rats, stimulating rapid avoidance (Leach *et al* 2001). A further possible reason for such avoidance is that even low concentrations of CO_2 act as a potent stimulus of breathing and, as a result, cause hyperventilation and feelings of breathlessness (eg HSUS 2002, citing CCAC 1993).

CO_2 is thus probably acutely distressing for pest rodents. It may be rapidly acting if used at sufficiently high doses, but it is probably difficult or impossible to achieve these rapidly in a real pest control situation. On the positive side, however, there are unlikely to be long-term effects on rodents that survive because the gas is eliminated quickly via the lungs (Danneman *et al* 1997; HSUS 2002, citing Kohler 1999) — although the brain may be left damaged by prolonged anoxia, an issue not considered in these texts.

Phosphine

Aluminium phosphide is similar to zinc phosphide in that it produces phosphine on contact with water. However, this compound is used as a fumigant rather than bait, one or two tablets being placed per burrow or several placed under gas-proof sheets when fumigating specific structures (eg Meehan 1984). In practice, this gas has been used very successfully against infestation, being potently toxic (Meehan 1984; Timm 1994a). The action and toxicity of phosphine have already been discussed above, although the mode of delivery is clearly different here.

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Humaneness

In humans, inhaling phosphine typically causes coughing, choking, breathlessness and pressure in the chest, nausea and vomiting, lung and abdominal pain, headache and a buzzing in the ears, jaundice, intense thirst, and also ataxia, paraesthesias, intention tremors and convulsions, before leading to coma (eg Meehan 1984; Marks 1996; PSD 1997). In terms of pathologies, it causes pulmonary oedema (Wilson *et al* 1980; Garry *et al* 1993), and autopsy may also reveal myocardial damage (Wilson *et al* 1980).

In poisoned rodents, it gives rise to similar signs of respiratory irritation and pain and other forms of discomfort (Meehan 1984). For example, in one study, rats exposed to phosphine gas showed "clinical signs indicative of mild respiratory irritation" such as salivation, lacrimation, face-pawing and dyspnoea (Waritz & Brown 1975). A review by the Pesticide Safety Directorate (PSD 1997) also showed that rats and mice exposed to phosphine gas display face-washing movements suggestive of eye and respiratory irritation, shivering, piloerection, clinging to the walls of the cage, exophthalmos (protruding eyeballs), convulsions, and hindlimb paralysis followed by full paralysis and death. Animals may not start being symptomatic until 30 min after exposure, and die usually within 2 h (the range being 50 min to 3 h, depending on dose) (PSD 1997). The symptomatic period is thus a few hours at maximum.

There appears to be little lasting harm to subjects exposed to non-fatal levels. Although tissue damage has been reported in human fatalities (Wilson *et al* 1980), rats exposed to lethal levels showed no histopathological changes (Waritz & Brown 1975). Meehan (1984) also reports negligible *post mortem* findings. Similar results were found in another study: there were no necroses in rats killed by the gas, along with no apparent ill effects in animals that recovered (PSD 1997).

Cyanide gas

Cyanide gas is generated within a burrow system via calcium or magnesium cyanide powder which releases hydrogen cyanide (HCN) gas on contact with water, or via discs of cardboard soaked in HCN which are packed in airtight tins until needed (Meehan 1984). A pump may be used to propel the gas throughout the system (PSD 1997). Cyanide is primarily a centrally acting toxin which inhibits the cytochrome oxidase system of all cells (Timm 1994a) and suppresses CNS activity, leading to respiratory suppression and cardiac arrest (Bonsall 1984; Anonymous 1993; Gregory *et al* 1998). It also combines with haemoglobin, destroying the oxygen-carrying capacity of erythrocytes to cause cyanosis and tissue anoxia, the brain again being the most affected organ (Meehan 1984; Krishna & Katoch 1989; PSD 1997). Overall, these effects rapidly lead to coma and death (Bonsall 1984; Anonymous 1993; Gregory *et al* 1998). Cyanide seems as effective as phosphine gas in practice and, as a result, is used worldwide (Meehan 1984). However, although antidotes such as amyl nitrate can be very effective (eg Nagler *et al* 1978; Krishna & Katoch 1989; Timm 1994a; Lam & Lau 2000), this poison's rapid speed of action makes it highly dangerous in case of accident.

Humaneness

In humans, cyanide's effects once again depend on dose. Low doses of cyanide cause dyspnoea, sharp headaches, salivation, weakness and convulsions. There can also be nausea and giddiness, vomiting, breathlessness and a feeling of pressure, and anxiety, but no pain (Meehan 1984; PSD 1997; Suchard *et al* 1998; Gregory *et al* 1998). Symptoms also include irritation of the mucous membranes of the eyes, nose, mouth and throat (Meehan 1984). Higher doses lead to transient respiratory and cardiac stimulation before loss of

consciousness, convulsions, respiratory failure and death (PSD 1997; Gregory *et al* 1998). Such loss of consciousness is generally rapid (eg Gregory *et al* 1998); the acute inhalation of cyanide gas can kill humans within minutes if not seconds (Anonymous 1993; Timm 1994a). Laboratory studies of primates show that the gas leads to hyperventilation, followed by loss of consciousness after 1–5 min (Purser *et al* 1984).

For rodents, too, cyanide gas is said to be a quick and relatively untraumatic cause of death (eg Scott 1969; Rowsell *et al* 1979), although there are few data on its clinical signs and speed of action. Concentrations of 1 mg l^{-1} will kill rabbits in under 1 min (mice reported as being more sensitive and rats less so), while 0.22 mg l^{-1} kills rabbits in 18 min on average (PSD 1997). In this cited study, animals generally 'collapsed' in about a third of the time taken to die, while in another cited study, death occurred a minute or less after onset of symptoms, even at concentrations that took 29 min to kill (PSD 1997). Other studies of cyanide's humaneness focus on cyanide in baits, such as those used for possums in New Zealand (eg O'Connor *et al* 1998; Gregory *et al* 1998; Feral Control 2000). Here, cyanide causes some signs of discomfort but again only briefly, these being rapidly followed by unconsciousness. Signs of poisoning include short episodes of hyperpnoea or dyspnoea, uncoordinated and abnormal body movements for about 1 min, and prostration with spasms and a growing lack of responsiveness to external stimuli for a further 3–4 min (Gregory *et al* 1998). However, there is no retching, vomiting or evidence of pain (Gregory *et al* 1998; Feral Control 2000). Convulsions occur, but as cyanide causes a rapid loss of cortical EEG activity (Burrows *et al* 1973 and Brierley *et al* 1977, cited by Gregory *et al* 1998) and as the convulsions occur after the start of the progressive loss of reactivity to external stimuli, they are believed not to be distressing (Gregory *et al* 1998). In this study, possums thus showed clinical signs for about 5 min, being unconscious 6–7 min after ingestion until death 7–10 min after onset of unconsciousness (Gregory *et al* 1998). Another report states that ingested cyanide can cause possums to be unconscious in just 60–90 s, and dead in 2–5 min, animals being symptomatic for just 40–70 s (Feral Control 2000). In addition, although cyanide-shyness can occur, it does not seem to result from learned aversion (Warburton & Drew 1994). Studies of rabbits and possums therefore suggest that cyanide will kill rodents rapidly, render them unconsciousness even more rapidly, and cause some brief, mild to moderate distress, but no pain.

Cyanide appears, however, to be the only fumigant with a risk of long-term sequelae. Sub-lethal doses in both humans and dogs can cause Parkinsonism (eg Schmidt *et al* 1978; Inoue 1993), particularly if these doses are high (reviewed in Gregory *et al* 1998). This is because cyanide can damage central dopaminergic systems (Kanthasamy *et al* 1994). However, healthy survivors of cyanide exposure have also been reported, both in human studies (eg Bonsall 1984; Lam & Lau 2000) (although interpretation is more difficult here because of antidote-use; Gregory *et al* 1998), and in animal studies (eg Schmidt *et al* 1978; Purser *et al* 1984; Gregory *et al* 1998). Thus, long-term disability is a risk of surviving this gas, but appears far from inevitable.

3) Traps

Trapping rodents is generally considered labour-intensive because large numbers of traps are usually needed, they can be bulky to carry, and they require regular checking (eg Meehan 1984; Smith 1994; Killgerm 2000). However, many sectors of the food industry (eg those seeking organic status, or requiring approval from the American Institute of Baking) rely on non-toxic control (eg Hughes 1998), and trapping can be very successful (eg Proctor 1994;

Killgerm 2000). For example, intensive trapping has significantly reduced rat damage to paddy fields (Islam & Karim 1995). Thus, good success rates can be achieved, providing that high numbers of traps are used (eg a dozen in a house, and a hundred or so in a small warehouse; Randall 1999). Trapping also has the practical advantage that bodies are collected, allowing the simultaneous monitoring of rodent populations, and also preventing the unpleasant smell of decomposing corpses (eg Corrigan 1998b; Weile 2001).

As with poisons, nestlings are not killed but adult females are, with obvious welfare consequences, and the accidental trapping of non-target animals can occur, although not if traps are well-designed and appropriately located (eg Morriss *et al* 2000); however, the bodies of trapped rodents are obviously harmless to predators.

Sticky boards

Sticky boards are squares of wood, plastic or stiff cardboard coated with highly adhesive 'rodent glue'. They are placed on rodent runways, and when an animal crosses the boards it becomes stuck by the feet and fur. How the animal then dies varies. In the UK, where boards are used by professional pest controllers only (eg Allen 1999; Network Pest Control Systems 2001), they must be checked at least daily and live animals "humanely killed" (eg MAFF 1996; Randall 1999). However, in countries where these traps can be bought by the general public, rodents may be killed in a variety of unregulated ways, or even left on the boards to die. For example, Meehan (1984) says "they do not kill the animal immediately", Potter (1994), that "mice become entangled ... soon dying of suffocation", and Gilkeson and Adams (1996), that "there is some controversy ... because of the length of time it takes for captured rodents to die".

In practical terms, sticky boards can catch many animals at once, but are not suitable for damp or dusty environments (eg Meehan 1984; Saskatchewan Agriculture and Food 1998). In some cases, they are stipulated for mice only (eg Killgerm 2000; Network Pest Control Systems 2001), but in the USA and other countries, versions are also produced for rats (eg Galaxymall 2001).

Humaneness

The humaneness of sticky boards depends on the length of time for which the animal is trapped and on the manner of death (eg Frantz & Padula 1983). In the UK, if checked daily as recommended (eg MAFF 1996), rodents may be stuck for up to 24 h, although some in the pest control industry recommend that the traps are checked more frequently, for example, every 8 or 12 h (Hughes 1998; Allen 1999). However, when used by the general public, as in the USA, the length of time is unregulated and may be several days.

During this time, rodents are likely to experience pain and distress through being trapped, the physical effects of the adhesive on functioning (eg suffocation; Potter 1994), and trauma resulting from panic and attempts to escape, such as forceful hair removal, torn skin and broken limbs (Frantz & Padula 1983). After 3–5 h, animals have been reported as covered in their own faeces and urine (Franz & Padula 1983). When boards are collected, animals are also often squealing (Howard 1996; Agrizap 2000); one pest control operative even described them to us as "screaming their heads off". Some rodents also bite through their own limbs to escape (eg Frantz & Padula 1983; Helst 2002). Sticky boards would thus seem to have the same major welfare costs as leghold traps: instant and prolonged distress and trauma, followed by dehydration, hunger and sometimes self-mutilation when animals are held trapped for long periods.

The mode of death and its welfare consequences vary. In the UK, the animals may be killed with CO_2, by neck dislocation, or sometimes by striking (Timm 1994b; Allen 1999). However, in other countries, they may be unspecifically "hit with a stick" (FAO 1999), and some literature, including web-sites advertising these products to the USA public, simply does not specify how the animals should be killed (eg Randall 1999; Galaxymall 2001), raising the possibility that people may use drowning, incineration, or other convenient methods. All of these killing methods, even the humane ones, also involve the potential welfare problem of fear at the approach and proximity of humans. Simply leaving rodents to die, in contrast, does not raise this issue, but it clearly brings many problems of its own, as animals will die more slowly from dehydration, starvation or exhaustion (eg Agrizap 2000). Exhausted animals can also fall face down into the glue and suffocate (Frantz & Padula 1983). When left to die like this, one study showed that the shortest recorded death time was 3 h, but some animals were still alive 24 h after being trapped (Frantz & Padula 1983).

Live traps

Live box traps may be baited, or unbaited but placed on runs where the animals travel into them. Good traps can catch several animals at once (eg Potter 1994; Killgerm 2001), and sometimes can be more effective than other forms of trapping (Islam & Karim 1995), though they are said to be more effective for mice than for rats (Corrigan 1997b). Checking is least labour-intensive for forms with transparent tops, or those that produce a signal (allowing remote monitoring) when a rodent has been caught (eg Natrocell 2001b). Trapped rodents can then be released off-site, or humanely destroyed (Killgerm 2001).

Humaneness

Live traps need not injure or harm the animal, although the restraint itself may cause stress, as may trapping several live animals together (which can sometimes even result in cannibalism; Agrizap 2000). Humaneness also depends on whether the traps contain sufficient bait to prevent starvation and nesting material to prevent cold stress (Cleminson 1969; Corrigan 1997a), on how often they are checked and thus how long animals are left there, and also on whether, and how, animals are killed. Releasing live animals to a new location also raises potentially serious welfare issues (eg Broom 1999; Letty *et al* 2000). For example, both dormice (Bright & Morris 1994) and red squirrels (Kenward & Hodder 1998) show high mortality rates when translocated to an unfamiliar area, finding it more difficult to forage and evade predation. The same is therefore probable for rats and mice — especially mice, which are very likely to experience high aggression from local territory-holders (eg van Zeegeren 1980). Also, in some countries such as the USA, it is not uncommon to use such traps to kill, simply by leaving animals trapped until they die (eg Corrigan 1998b), presumably of starvation or dehydration, and raising obvious welfare issues.

Snap traps

Snap traps are spring-based devices which kill by means of a rapidly descending bar. They are baited, with chocolate, fruits, peanut butter and cooked meats all being effective lures (Allen 1999; Randall 1999; Killgerm 2000). A practical disadvantage is that they need to be re-set each time they catch a rodent. However, they are potentially easier to monitor than live traps and, in the field at least, they can also be more effective (eg Woodman *et al* 1996; Stanko *et al* 1999) giving potentially excellent control. For example, in one New Zealand national park, 'Victor Professional' snap traps successfully and cost-effectively reduced black rat populations by about 90%, and were significantly more effective than

anticoagulants (Burns *et al* 2000). They are also the recommended means of control in some indoor situations, such as office infestations (Corrigan 1997b).

Humaneness

The best snap traps kill instantaneously, and are thus good from a welfare perspective (Nutman *et al* 1998; Broom 1999). Draft New Zealand National Animal Welfare Advisory Committee Trapping Guidelines, for example, require traps to cause a loss of palpebral reflexes in under three minutes (see eg Warburton *et al* 2000). Forms designed to crush the skull are said to be most efficient and humane; in mink, all good snap traps were found to cause irreversible loss of consciousness within 2 min, and within 1 min when the skull was damaged rather than the neck (Proulx & Barrett 1991).

However, welfare problems can occur if the traps cause injury rather than death, and this makes it absolutely essential to check them at least daily. For example, in one study of squirrel traps, 5% of animals were still alive when traps were checked (Cleminson 1969), and other surveys indicate that 7–14% of wild rodents caught by snap traps may be injured without being instantly killed (T Sainsbury, personal communication 2001). In a range of mammals including rodents, this can occur if the trap design is incorrect (Proulx & Barrett 1991; Drickamer & Mikesic 1993; Warburton *et al* 2000). For example, the 'Museum Special' trap has been found to catch mice by the legs or tail 57% of the time, compared with just 4% for the 'Victor', the latter also having a much higher kill rate (99% compared to 74%) (Drickamer & Mikesic 1993). Non-fatal injury can also occur if the lack of pre-baiting leads to tentative approach movements from the target animal rather than a confident reaching towards the bait (C Booty, personal communication 2001), or if a sensitive trap (eg one for a mouse) is placed such that larger non-target animals can trigger it. Rats caught in mouse-traps, for example, tend to be injured rather than killed, although this risk could be reduced by enclosing the trap so as to allow access only to mice.

Electrocution traps

Traps that kill by electrocution are a relatively recent innovation. With the trade-name 'Zapper', these devices consist of an open-ended box baited with dry food. The floor is made of two plates which are terminals; a rodent bridging these two plates receives a 2 min-long shock, transmitted via the feet, of around 2000V (Agrizap 2000; Weingarden 2000; S Griffiths, personal communication 2002; M Weingarden, personal communication 2002). This causes the heart to fibrillate and the respiratory muscles to become unable to function, the failure of these organs then causing death (Agrizap 2000; Weingarden 2000).

The practical advantages of such traps are numerous. They can work outdoors as well as indoors (although they need to be covered with plastic or similar in case of rain; Agrizap 2000; Victor 2001). They are battery-driven, hence easy to power, and also portable. Disposing of bodies is aesthetically less unpleasant than with snap traps; and whether or not they have been triggered can be assessed easily, by means either of a light signal or of radio signals allowing up to 16 traps to be simultaneously remotely monitored (eg Agrizap 2000). Finally, they can also be used effectively on large, complex sites such as farms (Helst 2002; Trap-Man 2002), and in domestic settings they seem to work as well as poisons and faster than snap traps (Weile 2001), although not all agree that they would work well against large populations (Weile 2001). Their disadvantages are that, like many other traps, they work best after a period of pre-baiting, especially to catch rats; they also have to be reset between kills (Agrizap 2000) (although note that this is not true for similar traps used on possums; Dix *et al* 1994); and small pets may potentially receive a shock if entering them (Bugspray 2002),

although these traps are very unlikely to do pets (or children) any real harm (Agrizap 2000). Mice may also sometimes move too fast to make a good contact between the plates (Agrizap 2000), and tests on rats in New Zealand found that three out of five rats fell over when shocked, broke the contact, and so failed to be killed (Warburton 2002). Finally, they are expensive — more than ten times the price of a typical snap trap.

Humaneness

For humans, the experience of receiving a shock depends on the voltage, current and waveform of the electricity involved (eg Stratbucker 1984; Taser International 2002c), and so, without detailed technical data from the Zapper, it is difficult to judge exactly which human accounts are most relevant. The manufacturers state that the Zapper works like a police 'stun-gun', which is similarly a high-voltage, low-current apparatus (Agrizap 2000; Weingarden 2000; M Weingarden, personal communication 2002). Stun-guns apply frequent pulses of high voltage that spread over the body from the point of electrode contact (Burdett-Smith 1997; Harris 2001; Taser International 1996, 2002a). They can sometimes cause surface contusions or lesions (eg Ordog *et al* 1987; Ikeda *et al* 1992; Burdett-Smith 1997) and also, later, pain from muscle-stiffness (Burdett-Smith 1997). The shock itself is also generally painful and aversive (Kornblum & Reddy 1991; Fish & Geddes 2001; Harris 2001; Wright 2001); livestock immobilisers, which work in a similar way, are likewise known to be aversive to sheep (Rushen 1986). However, there are no long-term effects of being shocked with a stun-gun (eg Ordog *et al* 1987; Fish & Geddes 2001; Taser International 2002b). In some cases, human targets even retain no clear memory of the experience (eg Taser International 2002b,c) — although the study showing this to the greatest extent was largely based on drug addicts, many part-way through psychotic episodes (Ordog *et al* 1987), and post-shock amnesia is not generally the norm (S Tuttle, personal communication 2002).

However, in one important way the Zapper is not like a stun-gun: it aims to kill. One reason that stun-guns are not lethal is that their rapidly pulsatile waveforms have minimal effects on heart and lung tissues (Stratbucker 1984; Taser International 2002a), while the Zapper, in contrast, *does* cause these muscles to constrict (Agrizap 2000). The induction of ventricular fibrillation is also the way in which electrocution is used to slaughter sheep, cattle and other livestock (eg HSA 2000a). However this process is believed to be intensely painful (HSA 2000a; C Mason, personal communication 2002); thus when meat animals are slaughtered, electrocution across the thorax must be preceded or accompanied by stunning, for example by also passing a current across the head to induce rapid unconsciousness (HSA 2000a,b; see also Close *et al* 1996 on laboratory rabbits). The crucial issue for the Zapper is, therefore, does it cause unconsciousness before the animal can experience the painful muscle constriction, ventricular fibrillation and respiratory distress caused by being shocked? At the moment, data are not available to assess this. For example, although there are accounts that shocked rodents show no behavioural signs of pain (Weile 2001) and lose their palpebral reflexes within 30 s (B Warburton, personal communication 2002), such observations can tell us nothing about the animals' real experiences because of the paralysing effects that the shocks have on muscle activity (C Mason and M Raj, personal communications 2002).

However, whatever the rodent experiences prior to death, the time taken to die is very brief (Weile 2001), for example under 2 min (M Weingarden, personal communication 2002), with some commercial websites suggesting that this can be further reduced with a greater power supply (eg Bugspray 2002). These results on time taken to die are not dissimilar to those concerning snap traps. Furthermore, any rodents that escape being shocked are not left with burns (a happy contrast with similar traps for possums; Dix *et al*

1994) because of the current's very low amperage (Weingarden 2000; Weile 2001; M Weingarden, personal communication 2002), although the animal may perhaps experience muscle weakness or loss of function for a short period (eg as felt for up to 15 min by humans shocked with stun-guns; Burdett-Smith 1997). Mice exposed to such non-lethal shocks are also said to return readily to the trap (Agrizap 2000), although it is unclear whether this claim is backed by data.

4) Non-toxic lethal feeds

Cellulose-based lethal feed pellets are another relatively new product (Natrocell 2001a; Pest Control Direct 2001). The pellets consist of plant-based material, primarily cellulose, flavoured for palatability. They are non-toxic and hence safe for larger species to ingest; and they also have no secondary poisoning risks (Natrocell 2001a). They can be used on both outdoor and indoor sites (although they lose effectiveness if damp), and by industries unable to tolerate toxins on site. However, how they kill is difficult to ascertain. The pellets interfere with the normal functioning of the gut (Natrocell 2001a) and seem to kill by encouraging the proliferation of gastrointestinal pathogens (Brennan 2001), thus perhaps causing illness or toxic shock.

Humaneness

The product takes four to ten days to work (Pest Control Direct 2001), with up to five days from pellet-acceptance until death (Natrocell 2001a). Animals become huddled and lethargic in the last few hours before dying (Natrocell 2001a), suggesting pain, discomfort or sickness, but for a relatively short symptomatic period. This may be behind claims that this product is more humane than conventional poisons (Brennan 2001; Natrocell 2001a). However, because the manner of death is unknown, it is currently difficult to truly assess humaneness. Potential causes for concern include distension of the gut leading to gastrointestinal pain and discomfort, energy deprivation leading to hunger and weakness, and the illness and distress that would follow septicaemia or toxic shock (see eg Gregory 1998 for an account of physiological mechanisms of sickness).

5) Deterrence and proofing

Removing rodents usually only leaves a temporary void, soon re-filled by immigrants and the rapidly proliferating descendants of surviving animals (eg Proctor 1994; Allen 1999). For example, in urban areas, reinfestation can follow elimination programmes within months (Lambropolous *et al* 1999), and in another study on an agricultural site, the rat population took only 2–8 weeks to recover after 70% were killed (Lu *et al* 1994). Therefore for long-term population reduction, unless control is very sustained or frequently repeated, other techniques need to be employed to reduce the carrying capacity of the site and surrounding areas, or to exclude or repel rodents (eg Meehan 1984; Macdonald *et al* 1999; Rentokil 2001; Hughes 1998).

The most obvious and important tactic is to minimise available food and water (eg Proctor 1994; Hyngstrom & Virchow 1996; MAFF 1996; Surgeoner 1996) by clearing surplus food, water and refuse away, keeping food in sealed rodent-proof containers, and dealing with leaky taps and open water troughs. Eliminating nest-sites and refuge areas is also vital (Hyngstrom & Virchow 1996; MAFF 1996; Ramsey & Wilson 2000; Surgeoner 1996) and can be very effective: in one urban rat control programme, it was estimated that up to 90% of burrows were successfully eliminated (Lambropoulos *et al* 1999). This so-called

'harbourage' is important around the site too; clearing surrounding vegetation and debris can be very effective, reducing local populations and creating clear regions that rodents are reluctant to cross (Proctor 1994; Lu *et al* 1994; Forage Information System 1997; Ramsey & Wilson 2000). To give one example, such techniques were shown to reduce rodent damage to Australian macadamia orchards by up to 65% (White *et al* 1998, cited by Horskins & Wilson 1999).

Other deterrents include predators. Cats will not eradicate an established colony, but they may deter new rodents from arriving (Meehan 1984; Timm 1994b; Allen 1999); for example, in one Burmese village, houses with cats were found to have no rats, in contrast to houses without such predators (Proctor 1994). Encouraging natural predators such as barn owls may also slow rodents' population growth rates (Nader 1969, cited by Meehan 1984; van Vuren *et al* 1998); for example, the provision of perches for raptors can effectively reduce mouse populations (Kay *et al* 1994). Some chemicals, including naphthalene (Randall 1999; Hughes 1998) and aluminium ammonium sulphate (PSD 1997; Broom 1999), may also act as more localised deterrents to protect specific areas or foodstuffs. Ultrasound-emitting devices, in contrast, seem to have little or no success (eg Meehan 1984; Timm 1994b; IRRI 2001; Federal Trade Commission 2001).

Finally, physical exclusion is also important: as Hyngstrom and Virchow (1996) put it, "the most successful and permanent form of rat control is to *build them out* by eliminating their access". Methods include sinking low foundations to prevent animals burrowing in (Surgeoner 1996), erecting surrounding walls topped with T-pieces (MAFF 1996), putting metal collars on pipes to stop them being climbed (Allen 1999), plugging gaps in buildings with wire wool or netting (Randall 1999; Network Pest Control Systems 2001), placing bristle-strips along the bottom of doors (Network Pest Control Systems 2001), screening windows with wire mesh (Proctor 1994; MAFF 1996), and edging door and window frames with metal to prevent rodents entering by gnawing (Proctor 1994; MAFF 1996).

Humaneness

Reducing the availability of foodstuffs may perhaps increase infant rodent mortality, and predators may cause fear. Chemical deterrents may also cause some temporary irritation (PSD 1997), possibly to non-target animals too (PSD 1997; Hughes 1998). However, overall, such effects are very minor compared to those of other control techniques (eg PSD 1997; Broom 1999).

Discussion

The relative humaneness of different rodent control methods

Assessing humaneness is complex, not least because it involves comparing durations and intensities of suffering, and making such judgements as "is extreme breathlessness worse than nausea?" and "is a few hours of intense pain better or worse than several days of milder distress?". Rodent control methods clearly have a range of welfare implications, and so drawing boundaries across such a continuum is difficult. This task is made even more difficult by the fact that a given method often has a range of effects, and so may be more or less humane depending on dose, environmental factors, and other variables. Rodent control is also a complex ethical issue as it is often essential, and thus factors such as efficacy, economic cost, and human safety usually have to be weighed against animal suffering.

However, bearing such difficulties in mind, we suggest five methods of rodent control that seem relatively humane. The first is deterrence and exclusion, by means of rodent-proofing,

good hygiene etc — a method which seems to have few welfare consequences (eg PSD 1997; Broom 1999). The second is the use of well-designed snap traps, which will kill extremely rapidly if set appropriately and of good quality (eg Cleminson 1969; Proulx & Barrett 1991; Nutman *et al* 1998; Broom 1999). The third is the use of electrocution traps. These are certainly marketed as humane (eg Agrizap 2000; Pest Control Direct 2001; Bugspray 2002; Helst 2002), and if it does cause instant stunning, as is claimed, then the Zapper would be one of the most humane means of killing rodents available. As discussed, there is a real danger that this product causes fibrillation of the heart plus respiratory paralysis without prior loss of consciousness, which would be very painful and distressing. Nevertheless, this lasts for under 2 min, making the product rather similar to snap traps: not ideal, but better than most of the alternatives on offer. Furthermore, animals that escape are likely to be undamaged. The fourth option is cyanide gas. This has been recognised for several decades as promisingly humane (eg Scott 1969; Rowsell *et al* 1979; Gregory *et al* 1998), despite being opposed by at least one UK animal welfare organisation (RSPCA 1997), and also by Close *et al* (1996) for laboratory rodent euthanasia. It was also judged as relatively humane for rabbits (or at least more humane than phosphine) by the Pesticide Safety Directorate (PSD 1997). Cyanide does cause some discomfort, but it induces a very rapid and painless loss of consciousness. Sub-lethal doses may leave some animals disabled, but this is arguably offset by other advantages: as with all fumigants, dependent young are not left to die in the nest because all animals in the burrow are killed at the same time and, additionally, there is no risk of secondary poisoning to non-target animals. The final relatively humane method is the bait poison, alpha-chloralose. Again, this may cause some discomfort, but it acts rapidly and causes no pain or serious distress. Overall, this has "obviously great possibilities for humane rodent control" (Scott 1969). The Pesticide Safety Directorate also considered it to be a relatively humane control agent, as long as it is used at dose rates and environmental conditions favouring a rapid loss of consciousness (PSD 1997).

In addition to these five options, live box-trapping may also be acceptable (eg Cleminson 1969), particularly if traps are well-monitored so that no animal is trapped for long, and the despatch of trapped animals is rapid and humane. Release is less favourable to welfare, however: the likely plight of animals set free into unfamiliar areas, especially those already inhabited by other rodents, must not be overlooked however tempting it is to do so (see eg Bright & Morris 1994; Kenward & Hodder 1998).

Three further methods are less humane still, but arguably not the worst of current methods. The first is CO_2, which was considered relatively humane by the Pesticide Safety Directorate (PSD 1997) and which can potentially kill within minutes. This gas is undoubtedly aversive, and can in some circumstances take far longer than this to kill. However, it never takes longer than several hours, and also causes unconsciousness some time before death; in addition, it has the various welfare advantages shared by all fumigants (see above). The second is phosphine gas. This does cause signs of pain for a few hours but no longer, and, along with the usual welfare advantages of fumigants, also seems to cause no serious long-term harm to animals that survive sub-lethal doses (eg PSD 1997). The third member of this group is cellulose-based lethal feedstuffs, as these are also reported to cause signs of pain or illness for just a few hours (Natrocell 2001a). However, such data urgently need to be corroborated with detailed, published studies, and it may well be that further research demotes this last technique to the 'least humane' group given below.

The remaining methods of rodent control are often or always inhumane, either acting in a few hours but with very severe effects, killing in around a day with less acute effects, or causing lower levels of pain and distress but taking several days to induce unconsciousness.

Sulphur dioxide and zinc phosphide are methods of the first type. Sulphur dioxide causes severe pain and discomfort for several hours, along with some minor long-term damage to surviving animals, and has therefore been classified as inhumane by others (Rowsell *et al* 1979; PSD 1997). Concerning zinc phosphide, the Pesticide Safety Directorate (1997) says: "assuming a relatively short duration of severe symptomology ... the phosphine-generating compounds cause suffering but at high doses are more humane ... than the anti-coagulants rodenticides or calciferol". However, the ingested form is arguably less humane than the inhaled one; thus Scott (1969) described it as "very cruel", Rowsell *et al* (1979) noted that it "caused distress", and acute rodenticides such as this were described as "inhumane" by Chambers *et al* (1999) — all conclusions more consistent with the agonising descriptions of human suicides (eg Andersen *et al* 1996). This poison can also cause physical damage that causes a longer illness period, lasting up to several days.

An inhumane method of the second type, taking longer than a few hours to kill, is the sticky board. This method has long caused concern because of the enormous distress that the boards cause (see eg Frantz & Padula 1983; Meehan 1984; Proctor 1994; Hughes 1998; Randall 1999), even if the trapped animals are found after just a few hours and then humanely despatched. In the UK, sticky boards therefore tend to be avoided by responsible pest control operatives; for example, Network Pest Control Systems (2001) describe them as a "last resort measure", and for welfare reasons they are also not recommended by UK governmental agencies (MAFF 1996). In the USA and other countries, however, their use is even more alarming as they can be bought by the general public. Here, how long rodents are trapped for, and how they die, must be left to the imagination.

The bait poisons calciferol and anticoagulants are control methods of the third variety. Calciferol generally results in a prolonged time to death — usually a few days. It also has toxic effects associated with severe discomfort in humans, and a long symptomatic period in rodents associated with anorexia which will also have secondary disabling effects. Furthermore, sub-lethally affected rodents are likely to be left with long-term damage. Suffering for several days would be terrible for humans, causing sleep deprivation and probably weight loss and dehydration, to compound the direct effects of the poison. However, it is arguably even worse for small mammals, for whom this represents a greater proportion of their total lifespans (Kirkwood *et al* 1994, citing Porter 1992), and which need to eat, drink and sleep more frequently than do larger animals to maintain normal functioning. Acute poisons such as this are therefore acknowledged by some, including certain sectors of the pest control industry, to be painful (Chambers *et al* 1999; Killgerm 2000), and the Pesticide Safety Directorate (1997) judged calciferol to be markedly inhumane.

Finally, the anticoagulants, the most common means of rodent control, also cause discomfort and pain which lasts several days. As with calciferol, not only is this inherently unpleasant, but it also interferes with abilities to forage, resulting in weight loss and dehydration, and hinders escape from predators. Anticoagulants can also leave surviving animals ill and internally damaged, and can bring relatively high risks of secondary poisoning to non-target animals. Thus although they are often classified as humane in the pest control literature (eg Timm 1994a; Killgerm 2000), only one scientific study even partially supports this view (Rowsell *et al* 1979), with more recent experimental work revealing anorexia, postural changes and other clinical signs that typically last several days (eg Cox & Smith 1992; PSD 1997; Littin *et al* 2002). As we have reviewed, data from human cases provide further evidence that anticoagulants can cause pain and distress. The Pesticide Safety Directorate (1997) therefore concluded that anticoagulants are "markedly inhumane",

Littin *et al* (2000) that they cause "a prolonged period of sickness ... when rat ... welfare may be compromised", Kirkwood *et al* (1994) that anticoagulants cause "severe distress and pain", and Chambers *et al* (1999), too, that they are inhumane.

Rodent control: a welfare anomaly?

From the evidence in this paper, we can see that rodents are routinely subject to cruelty. This highlights an interesting paradox in the way we treat different classes of animal. Animals killed for food, research or their fur are never legally permitted to suffer for hours, let alone for days. Indeed in laboratories, slaughterhouses and veterinary practices, acceptable killing methods usually have to act in seconds (eg Broom 1999). The situation for rodent pests is thus very anomalous. Furthermore, if pets or research animals are lethally poisoned with calciferol or anticoagulants, they are often euthanased rather than being left to die (eg Hatch & Laflamme 1989; Talcott *et al* 1991; Johnson & Prescott 1994; Rumbeiha *et al* 1999), even if they are wild rodents (eg Gill *et al* 1994; PSD 1997). Thus in some circumstances the inhumaneness of these rodenticides, *including to rodents themselves*, is acknowledged — and yet in pest control situations it is largely ignored. Such issues are particularly important because of the enormous scale of rodent control. Even thirty years ago, Scott (1969) lamented "the atrocities ... inflicted on many millions of animals", and little has changed today: annually, many millions of rodents are killed using inhumane methods.

So why are these inhumane methods allowed? The primary reason is the undoubted necessity for effective rodent control. A second reason is the public's generally unsympathetic attitude to 'vermin' (eg Rowsell *et al* 1979; Broom 1999). A third is probably the unobtrusive way in which these nocturnal, burrow-living animals usually die: rodents generally become ill and die hidden from human view, making their suffering easy to overlook. The control of brushtail possums in New Zealand highlights how important this can be; here, the very visible symptoms of these poisoned animals caused a national demand for more humane methods (eg Eason *et al* 1997; O'Connor *et al* 1998). The fourth likely reason is the lack of detailed data, to date, on the part of many pest control operatives and animal welfare charities, resulting in little drive to limit or replace the most inhumane methods.

However, if rodent control methods are now looked at more critically, a number of questions clearly need to be asked. Does the practical need for control fully justify the suffering currently caused? Are the practical problems with some humane methods really insoluble? And is humaneness currently a high enough priority in the development of new or refined techniques? Looking more specifically at legislation and licensing within the UK, should anticoagulants (currently even brodifacoum, with its elevated secondary risks) remain easy for the general public to buy, or instead become more controlled (restricted to professional pest controllers, and then used only as a last resort)? Conversely, given its great humaneness, should alpha-chloralose now become licensed for use outdoors? And finally, is it logical or ethical for rodent traps and trapping to be completely unregulated by the current legislation (the 1954 Pest Control Act), which only covers non-rodent vertebrates? These questions are important given the enormous scale of rodent control; if rethinking our techniques were to reduce the use of these methods even by as little 10 or 20%, the number of animals prevented from suffering would still be vast.

Best practice with current technologies

The concerned individual can make some contribution to humane rodent control by trying a variety of approaches before resorting to sticky boards or the inhumane rodenticides.

For rodents inside buildings, alpha-chloralose, well-designed snap traps or electrocution traps should be used wherever possible. Alpha-chloralose is the method of choice for mice, when overnight temperatures fall below 16°C (aided by turning off hot water systems etc overnight, to prevent any pockets of warmth that could save some animals). Since rodents are more of an indoor pest in the winter (Potter 1994; Hughes 1998), this may not be a great constraint. Then where pest controllers would normally supplement this acute control with sustained anticoagulant baiting, prolonged snap trapping (or electrocution trapping) can be used instead. 'Victor' models have performed particularly humanely and effectively in a number of studies. The traps need to be numerous, in suitable locations and sensibly baited (see Corrigan 1998a, Randall 1999 and Victor 2001 for excellent advice, and/or use a pest control firm accustomed to working in poison-free industries); and protect mouse-traps in tunnels, if there is a risk that rats will get injured in them. Traps designed for larger rodents are also the most humane way to control indoor rats. In addition, control will always need aiding with proper rodent-proofing, the proper enclosure of foodstuffs and so on. If these approaches fail or need supplementing, then we might tentatively recommend live trapping with rapid humane despatch (not release) of trapped animals, or even CO_2 gassing for enclosed spaces such as cold stores — although these do have welfare issues, the severity of which is still not fully researched.

For outdoor rodents in burrow systems, we would recommend cyanide gas, catching any remaining animals with electrocution traps or well-designed — and regularly monitored — snap traps. Once again, rodents should be prevented from gaining access to nest-sites, food, water or shelter through habitat management and proper rodent-proofing (for example, consider covering animal feed troughs at night), and predators such as cats, barn owls and other raptors should be encouraged (see Kay *et al* 1994 for an effective example of this practice). If cyanide is too dangerous for a site, extensive snap-trapping should be used. If this fails, then we provisionally recommend using phosphine gas, live trapping, and perhaps lethal cellulose feeds. Rodents in semi-open buildings such as barns are the most difficult to control humanely, as the lack of burrow systems precludes cyanide while the lack of site enclosure rules out alpha-chloralose. However, deterrence, proofing and the removal of harbourage should all reduce rodent populations, along with systematic and sustained electrocution trapping and/or snap trapping.

Future research

Completely humane rodent control with current methods is often going to be difficult or costly. The rodent control industry therefore needs to develop new approaches which are practical and cost-effective, but with humaneness now as a top priority.

One approach is to refine existing poisons to make them more humane. For example, anticoagulants could potentially be developed that cause a far quicker death, and with minimal pain, perhaps by incorporating drugs such as salicylates that potentiate their action (eg Timm 1994a; Littin *et al* 2000). Ideally, rapid blood loss would occur via the intestinal tract so that animals become unconscious without painful haemorrhages building up in internal organs, muscles and joints. Compounds can also be made more effective to reduce the risk of non-lethal doses and/or to enhance death times (adding calcium salts to calciferol and its allies may be one such avenue; see Jolly *et al* 1995). An alternative approach is to offset the worst effects by including in the bait analgesics (cf Littin & O'Connor 2000), anti-emetics, or other compounds to cause sedation or unconsciousness. For example, Marks *et al* (2000) found that red foxes dosed with the poison 1080 (sodium monofluoroacetate), plus the sedative anxiolytic diazepam, showed much less intense activity after poisoning than foxes

poisoned with 1080 alone. This approach may be difficult to implement for compounds that take days to kill, but is potentially fruitful for zinc phosphide which usually causes clinical signs within, and for, just a few hours. Trap design, too, and the design of 'bait station' type trap enclosures, could also be refined to minimise the risk of non-lethal injury to both target and non-target animals. Electrocution traps also need to be assessed to see if they stun before killing and, if they do not, electrode design needs to be rethought to increase current flow through the brain.

In addition to refining existing techniques, new agents need to be investigated. For example, new fumigant technologies need to be developed, such as carbon monoxide capsules. This gas is undetectable to most species, and the deprivation of oxygen it induces seems to be a humane means of killing (Raj & Gregory 1995a; Broom 1999), simply leading to unconsciousness and then death (although research should first confirm that rodents, as burrowing animals, are not able to detect hypoxia; cf eg Raj & Mason 1999).

Cyanide baits are not currently used against rodents, but they too could have enormous potential for humane control. As reviewed earlier, when used against possums in New Zealand, they do cause some signs of discomfort (eg mild breathlessness), but only very briefly, this being rapidly followed by unconsciousness. Furthermore, the risk of sub-lethal dosing is lower than with gassing, and the risk of secondary poisoning is negligible: a lethal dose as ingested by a rodent will generally be ineffective against larger predators (Feral Control 2000), and the compound breaks downs very rapidly with very limited assimilation into the victim's (or predator's) body (Feral Control 2000; Wildlife Damage Review 2001). Cyanide paste has some disadvantages: it can lead to bait shyness (Eason & Wickstrom 2001) and can also give off dangerous vapours (Gregory *et al* 1998), but these problems can be solved with encapsulation, pellets being coated so that the toxin is released only on crushing (Feral Control 2000). Designing encapsulated pellets that are effective for rodents still remains a challenge, however (B Warburton, personal communication 2002).

Anti-fertility compounds and a range of methods of reproductive suppression also have some potential as humane pest control agents (eg Broom 1999). They may be practical too — in the laboratory, some require ingestion only every two or three weeks (Gao & Short 1994). However, note that not all anti-fertility agents are automatically humane: some chemosterilants are toxic (eg Saini & Parshad 1993); substantial doses of hormone can have unpleasant effects such as gastrointestinal disturbance (eg Chambers *et al* 1999); some agents starve or abort foetuses (Chambers *et al* 1999); and others act to increase gestation lengths, causing female deaths during parturition (Gao & Short 1994).

Finally, repellent compounds may have real potential for humane rodent control in the future. Predator odours are known to be aversive to rodents, as are synthetic analogues of these compounds (eg Denver Wildlife Research Center 1995; Kemble & Bohlwahnn 1996). They can also cause long-lasting deterrence of wild rodents in field conditions (Sullivan and co-workers, cited by Kemble & Bohlwahnn 1996). Developing powerful repellents with low rates of habituation could thus lead to effective yet humane control.

Conclusions

The most common methods of rodent control are generally inhumane. Furthermore, they are applied with little consideration for the welfare of the affected animals. Indeed, some of the least humane methods can currently be used by members of the general public, and as a first measure rather than as a last resort. This is largely incompatible with the way we treat other animals, even rodents that are poisoned for research in the laboratory. It is also a serious

welfare issue, as it affects many millions of rodents each year. However, some more humane methods do exist, namely snap trapping (with well-designed traps), electrocution, cyanide gassing, and alpha-chloralose, along with rodent exclusion and elimination of food supplies and harbourage. These methods can all be extremely effective (although admittedly, sometimes in limited circumstances). New industry-led research also needs to be encouraged with humaneness as a top priority. Reducing the number of rodents killed with existing anticoagulant preparations (and other inhumane techniques), even by just 10 or 20%, would have significant welfare consequences because of the vast numbers of animals currently affected.

Permission

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