The Old School, Brewhouse Hill, Wheathampstead,
Hertfordshire AL4 8AN, UK

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Evaluation of the tranquilliser trap device (TTD) for improving the humaneness of dingo trapping

CA Marks*[†‡], L Allen[§], F Gigliotti[†‡], F Busana[†‡], T Gonzalez[§], M Lindeman[†‡] and PM Fisher[#]

[†] Vertebrate Pest Research Department, Victorian Institute of Animal Science, PO Box 48 Frankston, Victoria 3199, Australia
[‡] Animal Welfare Centre, Department of Natural Resources and Environment, Sneydes Road, Werribee, Victoria 3030, Australia
[§] Robert Wicks Pest Animal Research Centre, Milmerran Road, via Inglewood, Queensland, Australia
* Contact for correspondence: Nocturnal Wildlife Research Pty Ltd, PO Box 2126, Wattletree Road Post Office, East Malvern 3145, Australia; camarks@attglobal.net
[#] Requests for reprints: Vertebrate Pest Research Department, Victorian Institute of Animal Science, PO Box 48 Frankston, Victoria 3199, Australia: vertebrate.pests@dpi.vic.gov.au

Abstract

*Predation of sheep and cattle by the dingo (*Canis lupus dingo*) is implicated in significant stock losses throughout much of mainland Australia. Leg-hold traps are commonly used for dingo control and ways are sought to improve the humaneness of these devices. We evaluated the performance of the tranquilliser trap device (TTD) attached to Victor Soft-Catch® traps for their ability to deliver a sedative and anxiolytic drug to trapped dingoes. A trapping programme was conducted in south-west Queensland where traps were set alternatively with a TTD containing either 800 mg of diazepam (drug TTD) or a placebo (placebo TTD). All TTDs included 20 mg of the bait marker iophenoxic acid (IPA) to ascertain dosing success. Each trap was fitted with an activity-monitoring data logger that recorded time of capture and subsequent dingo activity. In 41 out of 48 (85.4%) captures the TTD was ruptured and released its contents. No elevation in serum iodine levels above 1 mg ml^{-1} resulting from the ingestion of IPA occurred in 8 out of 36 (22.2%) captures, which suggests a higher rate of dosage failure. Dingo activity was highest in both groups immediately after capture, but declined after the first hour in each. The activity of dingoes that accepted a drug TTD was significantly reduced compared to those that took the placebo. However, tooth and limb damage scores did not differ significantly between the drug and placebo group. Much of the physical trauma may have occurred within the first hour of capture when activity was intense and before drug onset in the TTD drug group. The use of TTDs containing sedative and anxiolytic drugs has the potential to reduce anxiety and distress associated with prolonged captivity, but the delivery of a lethal agent that is rapidly acting and humane may result in better welfare outcomes.*

Keywords: *animal welfare, dingo, humaneness, trapping, vertebrate pest, wild dog*

Introduction

In a significant portion of the current distribution of sheep and cattle in Australia the dingo (*Canis lupus dingo*) is implicated as a predator of livestock (Fleming *et al* 2001). Steel-jawed leg-hold traps were once used widely in Australia for dingo control; however, these traps have the potential to inflict severe limb injury (Fleming *et al* 1998). The humaneness of leg-hold traps has received much attention from animal welfare and anti-trapping lobby groups worldwide (Gentile 1987). Padding of the steel jaws and the use of alternative devices such as the Victor Soft-Catch® trap have been shown to reduce trap-related injuries sustained by captured animals (Meek *et al* 1995; Fleming *et al* 1998). Padded traps were found to produce less physical trauma to canids such as red foxes (*Vulpes vulpes*) (Olsen *et al* 1986, 1988; Liscombe & Wright 1988) and coyotes (*Canis latrans*) (Olsen *et al* 1986; Linhart *et al* 1988; Hubert *et al* 1997). Dorner *et al* (1974) and Kreeger *et al* (1990) determined that a range of biochemical, serological and endocrinological changes indicative of stress and injury were reduced when padded leg-hold traps were used instead of steel-jawed traps. Despite this, some trappers have resisted the use of padded traps since they are perceived to be less effective than unpadded devices (Warburton 1982; Linhart *et al* 1986). Moreover, padded traps do not prevent the tooth damage, exertional myopathy, and anxiety (Rowan 1988) associated with prolonged capture.

Tranquilliser trap (or tag) devices (TTDs) were first produced by attaching rolled cloth containing tranquillisers to the jaws of traps (Balser 1965). Modern TTDs consist of a moulded rubber tube containing a tranquilliser, which is capped at the distal end and affixed to the trap jaw by two metal rings at the base. After capture, canids bite at the tube (tag) and ingest a proportion of the drug. Delivery of diazepam (Balser 1965) and propiopromazine (PPZH) (Linhart *et al* 1981) by TTDs has been shown to reduce the extent of foot injuries received by coyotes captured in leg-hold traps. Additional trials showed that PPZH delivered by TTDs reduced the severity of injuries sustained by grey

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wolves (*Canis lupus*) (Sahr & Knowlton 2000). Appropriately selected drugs have the potential to depress the activity of captive animals and reduce tooth damage and limb injury that may be a consequence of repeated pulling and biting at traps. Drugs that reduce anxiety may also mitigate distress associated with capture. We sought to assess the success of TTDs for the delivery of a sedative and anxiolytic drug to dingoes captured by padded leg-hold traps. While physical damage, such as limb and tooth injury, was seen as an important criterion to assess the humaneness of trapping, the overall exertion of trapped animals has not been investigated. Consequently, we developed a new technique in order to measure the relative activity of captive dingoes whilst held in leg-hold traps fitted with a placebo TTD or drug TTD.

Materials and methods

Study area

All field trials were conducted on the Bulloo Downs Station located in south-west Queensland's Channel Country, 100 km west of Thargomindah (28.5°S, 143.5°E). The Bulloo Downs site was largely flat and semi-arid, receiving an average of 205 mm of rainfall per annum and natural irrigation resulting from the periodic flooding of Cooper Creek. The 10 770 km^2 property contained a combination of mulga-covered sand ridges, alluvial grasslands and lignum swamp and was used for beef cattle fattening. All experiments were conducted in October and the highest daytime temperature rarely exceeded 25°C.

TTD preparation

McBride TTDs (Ranchers Supply Company, Alpine, Texas, USA) were filled with either 2.3 ml of a water-based gel (K-Y® Lubricating Jelly: Johnson & Johnson Ltd, Maidenhead, UK) and 800 mg of diazepam (Hoffmann-La Roche AG, Basel, Switzerland), or gel only for the placebo TTDs. Drug and placebo TTDs were dosed with 20 mg of finely ground iophenoxic acid (IPA) (Aldrich Chemicals, USA), which was mixed evenly into the gel. IPA is a quantitative biomarker that causes elevated levels of plasma iodine consistent with the amount of IPA ingested (Saunders *et al* 1993). It has been successfully used in studies of bait acceptance by wild canids (Larson *et al* 1981; Saunders *et al* 1993; Fleming *et al* 1998; Marks & Bloomfield 1999) with no reported palatability problems. Saunders *et al* (1993) proposed that bait consumption in red foxes could be quantified using standard bait doses of IPA and measuring the subsequent plasma iodine concentration after bait ingestion. Although Sahr and Knowlton (2000) reported successful use of TTDs containing 500–1000 mg of PPZH when used on traps set for coyotes, PPZH is not available in Australia. Diazepam was selected for this study because of its availability and because it has been shown to produce profound sedation in red foxes for over 8 h at 10 mg kg^{-1} (Marks *et al* 2000). Given the large body of data concerning its veterinary use and its existing Australian registration as a therapeutic agent it was considered to be a better candidate for immediate regulatory approval and potential registration, and therefore to be the most expedient means to assess the efficacy of the TTD. The sedative and anxiolytic properties of the benzodiazepines are usually less prominent in domestic dogs (Rehm & Schatzmann 1985, pp 13–23) and their half-lives in domestic dogs are typically 2–20 times shorter than in humans (Boxenbaum 1982). However, diazepam has been shown to have a longer plasma half-life (dog $t_{1/2}$ = 7.62 h) than other drugs in this group such as lorazepam (dog $t_{1/2}$ = 1.98 h) (Boxenbaum 1982). Diazepam is a relatively inexpensive drug which has a good safety profile and a reported oral LD_{50} of >1000 mg kg^{-1} in mice and 325 mg kg^{-1} in rats (Anon 1996), suggesting a low potential to be toxic in non-target species.

Trapping, recovery and specimen collection

All trapping was undertaken with modified #3 Victor Soft-Catch® leg-hold traps (Woodstream Corporation, Lititz, PA, USA). Trap modifications included: #11 PIT Pan Tension Kit; #4 Montgomery coil springs; D-ring base plate; 1.2 m chain containing double swivels and a #19 PIT Cushion Spring attached midway on the chain (Minnesota Trapline Company, USA). Traps were dyed and waxed (de-scented) prior to use and placed in areas where substantial dingo signs existed along vehicle tracks and around watering points. Before setting the traps, each was fitted alternatively with either a drug or a placebo TTD to one of the jaws only. Trap pans were either covered with gauze cotton cloth or supported by foam rubber and the chain secured by a 0.5 m steel stake. Trap sites were lured either with a commercial canid attractant (Canine Call: Magna Glan or Final Touch, Minnesota Trapline Company, USA) or with fermented meat preparations. Traps were inspected at approximately 24 h intervals as this is the legal requirement and common field practice that we wished to follow in order to make assessment of the TTD realistic. Dingoes were killed with a .222 calibre rifle by a shot to the heart from an approximate distance of 20 m as soon as the trap site was approached. A head shot was not appropriate as we wished to examine dentition for evidence of trapping-related damage, which may be destroyed by massive head trauma. Shooting was conducted by a trained person from a distance that allowed a consistently humane death to be effected. Immediately afterwards blood samples were taken from the heart or body cavity. Two 10 ml blood samples were stored in vacutainers without preservative and a single 10 ml sample was stored in a lithium heparin vacutainer (Becton, Dickson and Company, Franklin Lakes, USA). Dingo serum was submitted to the Alan Fletcher Research Station (Queensland Department of Natural Resources and Mines, Sherwood, Queensland, Australia) where total iodine was determined to within 1 mg l^{-1} by a calorimetric method after iodine had been catalysed to iodide in a caesium–arsenic reaction. Body mass was determined to the nearest 0.5 kg with a spring balance and the gender and coat colour were recorded. A search was made in the immediate area of the trap and any remnants of the TTD were collected and stored.

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The limb that had been held by the trap was severed from the carcass and individually tagged and frozen. Frozen limbs were transported to the Victorian Institute of Animal Science where they were thawed, X-rayed and then dissected and inspected for gross abnormalities. The pathologist who necropsied and assessed limb damage was not informed of the experimental treatment associated with each limb. Damage to soft tissue, bone, tendon, and cartilage was assessed using the scoring method described by Onderka *et al* (1990). Heads were removed immediately after death and later assessed for tooth damage. Teeth were removed from the skull after de-fleshing and age was determined by tooth density and pulp cavity methods described by Ellerton (2002) and Thomson and Rose (1992). The stomach and small intestines were inspected and any fragments of the TTD were recovered and stored.

Activity monitoring

Pulse count data loggers (Tiny-tag OEM 0-255: Gemini Data Loggers, Sydney, Australia) measuring 33 × 56 × 50 mm were modified so that they contained an internal non-position-sensitive CM4400-1 vibration and motion transducer (Assemtech, Essex, UK), which changes contact state when subjected to vibration and motion. Unlike mercury tilt-switches commonly used in activity-monitoring radio collars (Kenward 1987), the CM4400-1 transducer does not require large movements biased in one plane to achieve a change in contact state (Marks *et al* 2000). A box to house the data logger was constructed from 3 mm thick plate aluminium measuring 39 × 39 × 59 mm, which was sealed with a welded plate at one end. The activity logger was placed inside the box and enclosed with an end cap held in place by two screws. A metal eyelet, welded to the top of the housing, allowed it to be shackled to the chain 30 cm from each trap. Immediately before setting each trap the activity logger was programmed to continuously count total contact rate changes during each 30 s period (GLM Version 2.2: Gemini Data Loggers, Sydney, Australia). The activity logger and housing were then buried with each trap so that no parts were visible above ground level. Upon recovery, the data logger was removed from the housing and activity data were downloaded to a laptop computer. Activity data revealed the time at which the trap was sprung and a continuous record of the frequency and duration of movement of the tethered data logger until the trapped animal was euthanased.

Statistical analysis

Statistical analysis was conducted with Systat® Version 7 (SPSS Inc, Chicago, USA). The mean activity per min and weight of dingoes in the placebo and drug TTD groups were compared with Student's *t*-test. Total activity each hour was calculated as the total area under the curve (AUC) and a repeated measures ANOVA was used on transformed data to test the contribution of group, time of captivity and interactions of each on mean activity. Correlations with activity and weight were undertaken with Pearson's product moment correlation so that any effects of a relatively higher dose rate of diazepam received by smaller animals could be assessed. Tooth and limb damage scores for each group were compared using the non-parametric Mann-Whitney *U* test.

Results

Recovery and study population

In total, 48 dingoes (males = 24; females = 24) were captured (drug TTD = 25; placebo TTD = 23), predominantly during the periods 2000h–2300h and 0300h–0500h. No species other than dingoes and dingo hybrids were captured. Capture times between 1600h and 0600h (between the first and last capture) significantly departed from a random distribution ($\chi^2 = 38.0$; $df = 12$; $P < 0.001$) indicating that a significant bias existed in the time at which dingoes were captured. Overall there was a significant difference between the weight of males (mean = 18.8 kg; standard deviation [SD] = 2.47) and females (mean = 14.5 kg; SD = 2.27) ($t_{46} = 6.16$; $P < 0.001$). However, no significant difference existed in male and female weights between the placebo and drug groups.

Success of TTDs for drug delivery

Serum iodine concentrations were obtained for 36/48 dingoes, as 12 samples were not suitable for iodine analysis because of high levels of haemolysis. Mean serum concentration was 22.58 mg l^{-1} (SD = 23.6 mg l^{-1}; range = 0–69.2 mg l^{-1}). Serum iodine concentration was positively related to duration of captivity ($r = 0.52$; $F_1 = 9.67$; $P < 0.005$), indicating that iodine levels were continuing to climb and maximal iodine concentrations were unlikely to have been achieved in many of the samples. In 85% (n = 41) of all captures, fragments of the TTD from the gut or ground surrounding the point of capture confirmed that the TTD had been ruptured and its contents at least partially discharged. Overall, 8/36 (22.2%) dingoes were found to have serum iodine concentrations of <1 mg l^{-1} and another three of <3.75 mg l^{-1}. There was no significant difference in body mass ($t_{34} = -1.27$; $P = 0.21$) or time spent in captivity ($t_{34} = -0.26$; $P = 0.8$) for these 11 dingoes compared to the remaining sample. Dingoes captured with either placebo or drug TTDs where the TTD did not rupture (n = 7) were not considered in the analysis comparing activity, limb or tooth damage.

Activity of drug and placebo groups

There was no significant difference in the mean duration of captivity for trapped dingoes in the drug (mean = 703.5 min; SD = 237.9) and placebo (mean = 774.4 min; SD = 226.5) groups ($t_{45} = -1.04$; $P = 0.303$). Data from two activity-monitoring devices were discarded as one device had become wedged underneath the trap stake and another had been incorrectly attached to the trap. Combined with the seven dingoes that failed to liberate the contents of the TTD, comparative activity-monitoring data were analysed for 39 dingoes (drug TTD = 19; placebo TTD = 20).

Mean AUC activity per min was significantly different between the drug (mean = 14.20; SD = 12.47) and placebo

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(mean = 48.61; SD = 27.78) groups (t_{36} = –5.47; $P < 0.001$). There was no difference in overall mean activity attributable to the gender of the dingoes (t_{18} = –0.44; P = 0.66). A moderate positive correlation existed between body mass and mean activity per min in the placebo (r = 0.526; F = 6.88; df = 1; P = 0.017) and drug groups (r = 0.55; F = 6.9; df = 1; P = 0.018). The mean hourly activity of dingoes that took drug TTDs was significantly lower than the activity of those that accepted the placebo (F_1= 53.1; $P < 0.001$). Activity declined over time in both groups (F_{11} = 16.03; $P < 0.001$), but this was significantly enhanced by the drug TTD (F_{11} = 30.44; $P < 0.002$). There was no significant relationship between serum iodine level and mean activity per min (F_1 = 1.15; P = 0.303). Activity in the second hour of captivity in the drug TTD group declined to 24% of its mean first hour value (t_{18} = 6.59; $P < 0.001$). Activity in the placebo TTD group also revealed a significant decline to 52% of its mean value in the first hour (t_{19} = 5.15; $P < 0.001$). Thereafter, mean drug TTD activity scores were less than mean placebo TTD scores for each hour with the exception of hours 9 and 12. In both instances, 95% and 89% of the activity in both hours is attributed to four dingoes in the drug TTD group (Figure 1). Dingo activity in the 5 min prior to destruction was not significantly different between both groups (t_{38} = –1.221; P = 0.230).

Tooth and limb damage

Damage to the premolar teeth was frequent, with chipped or broken teeth common in both groups. Total tooth damage scores were similar for the drug (range = 0–26.5) and placebo TTD groups (range = 0–25); no significant difference between the median tooth damage scores was observed (T = 311.5; P = 0.254). No correlation was found between tooth damage score and mean activity per min (r = 0.12; df = 38; P = 0.231), or tooth damage score and duration of capture in the placebo group (r = –0.15; df = 20; P = 0.55). Limb damage was limited in its extent and frequency, with 13/20 and 16/19 dingo limbs having no visible injury in the placebo and drug groups respectively. Simple or compound fractures did not occur; fractures were limited to a single case of a bone chip on a digit. Superficial damage was generally limited to small cutaneous lacerations and subcutaneous haemorrhage. In the drug group, the single dingo that had a limb damage score of >1 (score = 10) was also observed to have the highest mean activity per min for the group, which was more than three times the group average activity score. However, there was no significant difference in the median limb damage scores for both groups ($T_{19,20}$ = 339.0; P = 0.253).

Discussion

A dose of 800 mg diazepam in the TTD group produced a highly significant difference in activity compared to the placebo group after the first hour of capture. However, 15% of dingoes did not rupture the TTD and levels of the serum marker of <1 mg l^{-1} in 22.2% of dingoes sampled suggests that TTD rupture alone did not guarantee that its contents would be ingested. In five cases the complete removal of the TTD may have been related to its inadequate fixture to the trap, which may be addressed by improving attachment procedures. Moreover, the use of a TTD attached to each of the trap jaws may greatly increase the likelihood of successful dosing. Serum iodine levels were found to increase significantly with time in captivity, but the time period between dosing and achieving maximal levels is unknown for the dingo. Conclusions about the relative quantities of IPA ingested by dingoes cannot be made without calibration of a dose–response curve during a 24 h period.

Dingoes in the placebo group showed reduced activity after the first hour of capture, possibly as a result of exhaustion or acceptance of capture. No paralytic sedation was observed upon recovery in the drug group, although symptoms sometimes included a noticeable loss of coordination. There was no difference in the activity of dingoes in either group during the period immediately preceding recovery. Sahr and Knowlton (2000) found that the degree of sedation is often difficult to objectively assess from cursory observations prior to recovery. Drug effects may abate over time and the efficacy of drugs delivered by the TTD cannot be objectively assessed by *post hoc* observations.

Dingoes caught in drug TTD traps were found to have tooth damage that was not significantly different from that of dingoes in the placebo group. Neither the duration of capture nor the mean activity was related to the tooth damage sustained by each dingo. Surprisingly, the majority of dingoes in the placebo group showed no apparent limb injury. Dingoes from the drug TTD group had less limb damage and a lower injury score overall compared to the placebo group, but this difference was not significant. These data suggest that much of the tooth damage and limb injury sustained by trapped dingoes occurs immediately after capture when activity levels (in the placebo group) are 2–4 times greater than during subsequent hours. From the time of capture, the onset of sedation is unlikely to be rapid enough to prevent tooth damage unless drug onset can be greatly accelerated.

Physical injury and pain comprise only one facet of the distress associated with trapping and confinement, as physical exertion and anxiety will also affect the welfare of the captured animal. Anxiety may be a major factor that promotes attempted flight, and exertion may aggravate injury. Whilst drug doses that result in profound tranquillisation, paralytic sedation, and central nervous system depression may be used in order to manage anxiety, this may prevent the trapped animal from defending itself from attack by insects, rivals of the same species, or predators (Sahr & Knowlton 2000). In our study, we discovered a fresh scrotum injury sustained by a dingo, which was apparently inflicted by another dingo. Moreover, heavy tranquillisation and sedation may also affect an animal's ability to thermoregulate (Wixson *et al* 1987), which may be fatal in very hot or cold climates. Alternative approaches may include the selection of drugs that have potent anxiolytic

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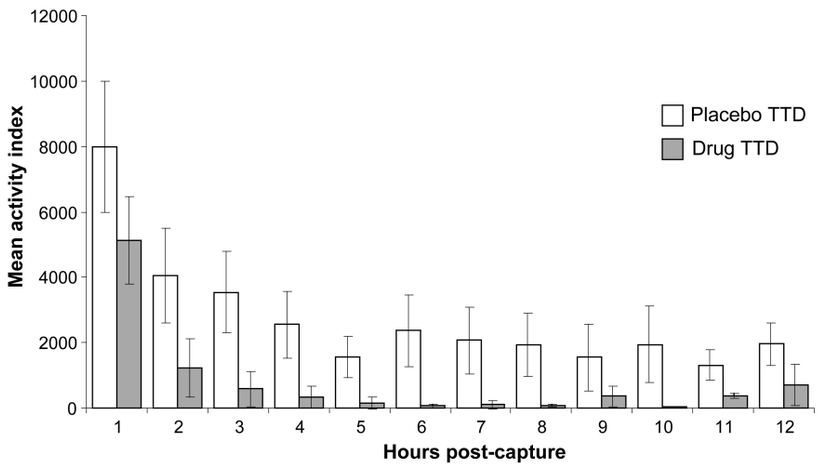
Figure 1

Mean hourly activity measure, AUC (Area Under Curve), for dingoes captured in traps with attached placebo TTDs or drug TTDs ($P < 0.05$). Post-capture activity in the drug TTD group falls significantly below that in the placebo TTD group after the first hour.



and analgesic properties at doses that do not produce profound sedation. Diazepam was used successfully to treat stress-induced hyperthermia in silver foxes (*Vulpes vulpes*) without major sedation, supporting its effectiveness as an anxiolytic in this canid (Moe & Bakken 1998). It is thought that all vertebrate species possess specific receptor sites for benzodiazepine drugs, which influence states of anxiety (Rowan 1988). Diazepam receptor affinity correlates well with the drug's behavioural potency, which includes anxiolytic, sedative-hypnotic, muscle-relaxant and anti-convulsant effects (Feldman *et al* 1996). Given that the drug doses received significantly reduced activity, anxiolysis would be another effect that could be expected.

These data suggest that the onset of anxiolysis and sedation occurred within the first hour of capture and that physical injuries may have been sustained during this time. The use of rapidly acting and humane toxicants in TTDs may be an alternative strategy to produce a more desirable animal welfare outcome through elimination of injury and anxiety. Agents that cause a rapid loss of consciousness and rapid death, typically sought during euthanasia, would be required. Presently, there is no ideal canid toxicant registered in Australia that would be suitable for TTD delivery. Sodium fluoroacetate (1080), the toxicant commonly used for wild dog control in Australia (Fleming *et al* 2001), would be unsuitable because it has a latent period of some hours before the onset of symptoms and death (Chenoweth & Gilman 1946; Chenoweth & St John 1947). The perceived humaneness of 1080 is controversial, especially with respect to its use in the control of carnivores (Gregory 1996; Oogjes 1996; Marks *et al* 2000). Strychnine-impregnated cloth attached to traps has been shown to be lethal to dingoes (Fleming *et al* 2001), yet, while relatively rapid in its action, strychnine toxicosis is associated with significant pain and suffering (Seawright 1989; Fleming *et al* 2001). Alternatively, sodium cyanide has been shown to be a very rapidly acting toxicant for canids (Connolly 1988; Algar & Kinnear 1990; Marks & Gigliotti 1996; Bubela *et al* 1998; Busana *et al* 1998; Allen & Gonzalez 2001; Marks *et al* 2002). Although cyanide is considered to be a humane means of euthanasia for dogs (Carding 1977), it is a non-selective mammalian toxicant (Marks & Gigliotti 1996; Marks *et al* 2002). Its use in TTDs may be problematic, especially if used with toxicant carriers that may enhance the persistence of cyanide or promote the contamination of traps and the area surrounding the trap site. We suggest that priority should be given to the selection of an appropriate TTD toxicant that causes rapid and humane euthanasia and is of low risk to field staff.

Animal welfare implications

The TTD appears to be a largely effective means of delivering chemical agents to dingoes caught in leg-hold traps, although efficacious delivery cannot always be guaranteed. In most states of Australia, field staff are required to inspect trap sites at least every 48 h. Potentially this may still result in substantial periods of confinement for dingoes before euthanasia, and tooth injury and distress may be experienced. In many instances, more frequent inspections are not practical in areas where trapping is conducted over large areas by a single operator. The use of anxiolytic and sedative drugs in TTDs may improve the humaneness of trapping by reducing overall activity and distress experienced over this period. However, the use of sedative/anxiolytic drugs may not eliminate tooth damage unless drug onset can be accelerated.

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