Optimization of Formulations for the Lethal Control of Feral Pigs

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ABSTRACT: Feral pig populations continue to increase and disperse into unoccupied habitats in North America. Associated control efforts cost U.S. taxpayers as much as $1.5 million/year. However, no toxicants are registered for feral pig control in the U.S. Development of sodium nitrite as an active ingredient in feral pig toxicants is ongoing in Australia, and a registration has been granted in New Zealand. Sodium nitrite is a strong oxidizer and is unpalatable to feral pigs, and thus it must be masked and stabilized to ensure effective dosing. We researched 3 different formulations of sodium nitrite loaded in a single bait matrix to evaluate mortality and acceptance in the context of U.S. registry requirements. Formulations were accepted by feral pigs but did not cause acceptable mortality rates in subjects. One formulation (TX1) produced mean mortality rates of 50%, which was well below our stated goal of 90%. Bait acceptance and mortality were diminished by insufficient masking of sodium nitrite. However, results indicate that our sodium nitrite formulation improved acceptance and mortality rates in feral pigs and could be the basis for improvements. Future investigations will focus on masking the taste of sodium nitrite.

KEY WORDS: control, control strategies, damages, feral swine, microencapsulation, sodium nitrite, Sus scrofa, toxic bait, wild pigs

INTRODUCTION
Feral pig (Sus scrofa) populations and range distribution continue to expand in North America (Bevins et al. 2014), leading to increased agricultural losses, property damage, negative impacts to native flora and fauna, and potential for transmission of disease to livestock and humans. Damage and control costs are estimated at $1.5 billion annually in the United States (Pimentel 2007). At the national scale, it will be challenging for the collective efforts of national, state, and private control practitioners to halt range expansion and population increase using available methods (i.e., hunting, trapping, specialized shooting, aerial gunning, dog hunting, and fencing). Annual damages and control costs alone justify the development of novel cost-effective methods for mitigating the problem. Human health risks and impacts to flora, fauna, and habitat, though difficult to quantify, provide further justification for development of new lethal control measures to be used in integrated management programs for feral pigs.

There are numerous lethal means for controlling feral pigs. However, cost-efficacy of these tools is highly variable and often expensive (Coblentz and Baber 1987, McCann and Garcelon 2008). Toxicants offer promise for cost-effective control because feral pig sows and their offspring frequently feed in groups (Eisenberg and Lockhart 1972) and can be attracted to artificial bait sites (Campbell et al. 2013a,b). Group feeding at artificial bait sights may facilitate delivery of baits to numerous individuals at a single location, reducing labor required to treat multiple individuals. Coblentz and Baber (1987) evaluated the economics of shooting, trapping, snaring, and poisoning and suggested that toxic baits were the most cost-effective means of control.

Feral pig toxicants are registered for use in Australia and New Zealand (APVMA 2013, NZEPA 2013). However, the products have not been evaluated in or are not suitable for registry in the U.S. due to concerns about humaneness, efficacy, or environmental fate. Facing similar concerns, Cowled et al. (2008) identified sodium nitrite (NaNO₂) as a potential active ingredient for development in Australia. Cowled’s work was the precursor to NaNO₂ registry in New Zealand and continuing efforts to develop and register a product in Australia and more recently in the U.S. NaNO₂ has many attributes that lend it to feral pig control. It is readily available due to widespread use in human food, medicine, and manufacturing processes (NTP 2001). Its frequent use as a food preservative in the U.S. (NTP 2001) has resulted in extensive knowledge (National Pork Board 2012) that is useful for evaluating the ramifications of its use as a vertebrate pesticide. These datasets may also fulfill some of the registration requirements of the United States Environmental Protection Agency (EPA), which are laborious and costly to satisfy. Here we present a preliminary summary of 3 pilot trials of microencapsulated NaNO₂ formulations in grain-based baits.

METHODS
Pilot trials were conducted in a captive setting at Kerr Wildlife Management Area (KWMA) near Hunt, Texas. Male and female feral pigs ≥20 kg were captured in Texas and group housed in a 2.02 ha pen for ≥14 days with a variable population of (30-80) feral pigs. Feral pigs are continuously stocked into the holding pen to maintain populations needed for ongoing research at the facility. Feral pigs had free access to shade, water, and a commercial pelleted pig ration (i.e., maintenance diet).
We evaluated 3 different formulations of microencapsulated NaNO₂ in KBAIT to determine whether any combination would reach our goal of 90% mortality and satisfy EPA. KBAIT is an extruded, grain-based bait similar to a cattle cube, approximately 1.3 cm in diameter × 5.1 cm in length. The TX1 formulation contained 10% NaNO₂ in the finished bait, which was prepared by extruding 11.1 g microencapsulated NaNO₂ (90% NaNO₂ with a 10% coating) per 100 g bait. The TX2 formulation contained 10% NaNO₂ in the finished bait, which was prepared by extruding 12.5 g microencapsulated NaNO₂ (80% NaNO₂ with a 20% coating) per 100 g bait. The TX3 formulation contained 20% NaNO₂ in the finished bait, which was prepared by extruding 22.5 g microencapsulated NaNO₂ (90% NaNO₂ with a 10% coating) per 100 g bait. We conducted only 2 replicates with TX3 because low consumption (i.e., rejection), and 0% mortality rates in the first 2 replicates indicated the formulation was not viable.

At onset of trials, 21 feral pigs were randomly allocated from the holding pen to three 0.20-ha gamefenced pens (n = 3 males, 4 females/pen). Toxic formulations were randomized to pens and tested contemporaneously to control for effects of weather and pen. Maintenance diet, placebo, and toxic bait were presented in 2 front-entry troughs/pen approximately 0.6 m apart between 1600-1700 hrs. A Reconyx PC-800 remote camera (Reconyx, Inc, Holmen, WI) in “Hyperfire” mode was fixed on each feeder to photograph feeding events. Maintenance diet was provided at 1.1% of group body mass for each test group on the first day of the trial. On Days 2-4, each test group was provided with the placebo version of KBAIT at a rate of 1.1% of group body mass. A continuation rule was used, which required that 100% of placebo bait be consumed on 2 consecutive days prior to initiation of toxic delivery. Placebo bait was offered for additional days until the stopping rule was met. Fresh toxic KBAIT was provided ad libitum on both Days 5 and 6 with effects being monitored daily at 1000 hrs.

Late effects were monitored on Day 7 and subjects were euthanized. One-way ANOVA tests were used to detect differences in group body mass. Summary statistics (mean and standard deviation) for body mass of pigs that survived and those that were killed by toxicant are provided pending a more comprehensive analysis of effects of body mass on probability of death across all trials and toxicants, including future trials. Mortality and consumption were summarized by formulation using the calculated mean of group results. In photograph analyses, a feeding event was recorded each time an individual placed their snout over the front perimeter of the feeder. Feeding events were assessed for each member of the trials and were summed by group. A consumption index was generated by formulation using the calculated mean of group feeding events.

**RESULTS**

Seventy feral pigs (30 males, 40 females) were challenged in 3 trials. Pig mass ranged from 20.0-85.0 kg. Mean mass was 36.7 kg (F = 0.84, P = 0.58). All maintenance diet was consumed by each test group on Day 1 of each trial (Table 1). All placebo baits were consumed by all test groups. Mean consumption of toxic formulas on Day 1 was 0.46 kg (TX1), 0.285 kg (TX2), and 0.295 kg (TX3). Mean mortality rates ranged from 0% (TX3) to 50% (TX1). Daily results for each trial are presented in Table 1. Mean number of feeding events in the first hour of toxic feeding bouts ranged from 78-216 events/hr. First-hour feeding events were highest in the TX2 treatment (Figure 1). Difference between mean number of feeding events between hours 1 and 2 of toxic consumption ranged from 22-182 events/hr (Figure 1) with the greatest difference occurring in TX2 treatments. Mass of pigs that died (n = 19) in all trials ranged from 20.0-66.0 kg. By trial, mean body mass of surviving pigs was 33.9 kg (SD = 17.8; TX1) and 36.3 kg (SD = 19.0; TX2), versus 36.5 kg (SD = 14.7; TX1) and 43.8 (SD = 21.4; TX2) for those that died.

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**Table 1. Results of KBAIT trials with 3 different formulations of NaNO₂ conducted in outdoor pens, 20 September - 18 October 2013, Kerr Wildlife Management Area, Texas.**

| Trial | Formula | Consumption (kg) | Day 1 Dead | Day 2 Dead |
|-------|---------|------------------|------------|------------|
|       |         | Ration provided at 1.1% of group mass | Ad libitum |            |            |
|       |         | Day 1 Maintenance | Day 2 Placebo | Day 3 Placebo | Day 3 Placebo | Day 1 Toxic | Day 2 Toxic |
| 3.1.23 | TX3     | 2.5              | 2.5         | 2.5         | n/a         | 0.46        | 0.00         | 0           | 0           |
| 3.1.24 | TX1     | 2.6              | 2.6         | 2.6         | n/a         | 0.94        | n/a         | 7           | n/a         |
| 3.1.25 | TX2     | 3.5              | 3.5         | 3.5         | n/a         | 0.47        | 0.02         | 3           | 0           |
| 3.2.23 | TX2     | 2.7              | 2.7         | 2.7         | n/a         | 0.20        | 0.00         | 0           | 0           |
| 3.2.24 | TX3     | 3.1              | 3.1         | 3.1         | n/a         | 0.13        | 0.00         | 0           | 0           |
| 3.2.25 | TX1     | 2.9              | 2.9         | 2.9         | n/a         | 0.40        | 0.00         | 2           | 0           |
| 3.3.24 | TX1     | 2.6              | 2.6         | 2.6         | n/a         | 0.30        | 0.00         | 2           | 1           |
| 3.3.25 | TX1     | 2.5              | 2.5         | 2.5         | n/a         | 0.23        | 0.00         | 1           | 0           |
| 3.4.24 | TX2     | 2.1              | 2.1         | 2.1         | n/a         | 0.36        | 0.03         | 1           | 0           |
| 3.4.25 | TX2     | 3.0              | 3.0         | 3.0         | n/a         | 0.11        | 0.05         | 1           | 0           |
DISCUSSION

Placebo KBAIT was readily accepted by pigs. Toxic TX1 produced the highest mortality rates of formulations. Mortality rate for TX1 (100%) in trial 3.1.24 along with mortality rates ≥14% in all replicates are a preliminary indication that some pigs will consistently accept TX1 at lethal doses. However, preliminary mortality rates along with test group’s failure (in all replicates) to consume all available toxic KBAIT are indicative that taste aversion was occurring, bait was not available to all members in test groups (e.g., resource completion), potency and/or timing of release was insufficient, or a combination of the aforementioned. It is possible that resource competition was occurring prior to group members becoming alarmed as a result of direct observation of intoxication of cohorts (i.e., learned aversion). Regardless, our most lethal formulation was not suitable to cause mortality at our goal of 90%. We presume 90% mortality rates in captive settings will facilitate field mortality rates required for registry of a NaNO₂ product with the EPA.

The conditions of our trials must be understood to provide reliable simulations for field settings where operational use will occur. Although captive settings facilitate control of environmental factors, the captive environment may not be an accurate simulation of field conditions and is certainly not natural. Moreover, little, if any information has been published with regard to the effects of captivity on feeding behavior of feral pigs and how such effects hinder translation of captive results into field settings. Thus, our trial design is based on unpublished experiences in pilot trials at our facility. We attempted to simulate conditions which reflect field conditions in which bait uptake is most probable (i.e., periods with low resource availability). Our subjects were on an ad libitum diet while in captivity prior to the trial. Low resource availability was simulated only during the trial. In Texas, periods of high ambient temperatures and low rainfall outside of anthropomorphic baiting associated with deer hunting seasons and mast drop (i.e., high availability of feed) are optimum for capturing pigs in traps. We project that such periods offer the highest potential for cost-effective feral pig control with toxicants in Texas.

Using our experiences and knowledge from research on domestic pigs (our best surrogates for feral pigs in captivity) may help to explain our results and improve design of captive simulations. Subordinate feral pigs may be displaced from food resources by more dominant animals (Graves 1984) and could contribute to biased mortality rates in a group setting. Unfortunately, we do not fully understand the mechanisms (e.g., body mass, previous experience, sex, aggression, etc.) that determine dominance in feral pigs. These factors must be investigated to improve captive simulations.

Turner et al. (2000) found that pig live weight did not encourage sufficient competition to reduce drinking by smaller pigs in captive settings. Georgsson and Svendsen (2002) found that a single feeder promoted fewer visits and greater consumption/visit than 2 feeders presented to group-housed pigs in captivity. In an ad libitum setting (such as our toxic deployments) and barring alarm by a group member’s observation of intoxication (which is not likely, given a 1-hr induction time), a single ad libitum feeder should ensure that all pigs receive sufficient dosing of bait, especially in cases where subjects were accustomed to a restricted ration prior to ad libitum toxicant. Our paired feeder layout, remaining toxic baits in all trials, and no notable differences in mass between dead and surviving subjects suggest that resource competition was not the cause of imprecision and variability of mortality in treatment groups.

Timing and quantity of individual consumption might explain whether competition occurred prior to onset of intoxication and could have affected dosing (e.g., individuals consuming a disproportionate amount of bait, individuals occupying feeders for a disproportionate amount of time). Yet, we have been unable to develop a protocol and facilities that provide reliable and efficiently obtained information upon which to make inference. These problems could be mitigated with individual pen trials or use of a radio-frequency identification-based feed intake monitoring system of group housed subjects. At present, we are still considering single housed pigs for future work. However, we are concerned that depression in sequestered pigs might result in abnormal feeding behavior (Martin and Foster 2013, unpubl.) and therefore may not well simulate uptake and mortality in field trials. This is the basis for our current group-testing regimes.

Unfortunately, group housing of feral pigs makes it difficult to quantify the timing and consumption by individuals. Firstly, it is difficult to identify (in remote camera images) similar-looking individuals in trials which occur under cover of darkness. Though pelage characteristics may certainly be used to identify individuals, this technique is not sufficient (time or reliability) when most subjects are uniformly black or dark-colored.

Figure 1. Consumption of KBAIT with 3 different formulations of NaNO₂ (# events) by feral pigs during trials in outdoor pens, 20 September - 18 October 2013, Kerr Wildlife Management Area, Texas.
Such pelage characteristics appear very similar in infrared images. Marking techniques have not significantly improved our ability to reliably distinguish individuals; identification of subjects with paint and/or livestock tags has not resolved the issue. Feral pigs obscured paint markings and tag identifications with frequent rubbings on trees and mudbaths prior to trial conclusions.

In pilot trials, we observed what appears to be disproportionate partitioning of equal resources (i.e., 7 pigs with 7 equal portions spaced equidistantly may still compete for any given proportion until it has been fully exploited) much like a feeding frenzy in fish. Although aggression amongst group members was observed, the subordinates did not appear to utilize other readily available identical resources. Onset of NaNO₂ intoxication may occur within 30-50 minutes (Cowlled 2008), quickly affecting feeding behavior and increasing the chance of learned aversion in individuals by observation of other group members. In our experience, feeding bouts of randomized feral pigs are not always synchronous. For instance, we have observed a single individual that was more conditioned to our feeding activities and did not appear to associate with other members of a randomized test group. This individual was the first to arrive when feed was presented. In such a case, it is possible that intoxication may present in the individual prior to other members of the group and increase potential for learned aversion, due to group awareness of the individual’s symptoms. We hypothesize that breaking up established bonds (through the randomization process) may not be representative of free-ranging feeding behavior. We are considering the implications of randomization in future trials.

Our preliminary results indicate that microencapsulation of NaNO₂ can positively impact acceptance and mortality rates in feral pigs. However, the protection of active ingredients is only part of the equation. We must also achieve release and absorption sufficient to produce acceptable mortality rates and mitigate other unforeseen results (e.g., non-target uptake). Though captive trials alter natural feeding behaviors, our results suggest that our inadequate mortality rates are the result of taste aversion rather than a function of our trial design. We will attempt to increase mortality rates by masking the taste of NaNO₂.

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