Effect of a Feed-Through Insecticide (Imidacloprid) on the Flea Index of a Norway Rat (*Rattus norvegicus*) Focus in Los Angeles, California

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ABSTRACT: Norway rats and their fleas are associated with transmitting diseases, such as murine typhus and plague, to humans. The use of rodenticide baits may increase human exposure to rodent fleas due to loss of their preferred host. Therefore, flea control is an important component of risk reduction. However, the use of insecticide powder or spray can be difficult in certain situations. In this field study we used a feed-through insecticide, imidacloprid, in a grain-based pellet formulation without a rodenticide to determine the effect on the flea index of a well defined, problematic Norway rat focus in a dense urban area of Los Angeles. The flea index was determined to be 12.9 (116 fleas/9 rats) 5 days prior to applying the product. Rats were allowed to feed on the pellets ad libitum for 48 hours, in the presence of previously existing competing food sources. Flea counts were then taken, and again 7 days later, resulting in flea indices of 2.3 (25/11) and 1.5 (23/15), respectively. Since this field trial was conducted in a very dynamic urban area, attempts to survey a concurrent control group were not successful. However, Norway rat surveys conducted in the previous 4 years from the same urban area for the same months, July and August, produced flea indices of 7.4 and 10.1, respectively. In this field trial, we found that a rodent bait formulation containing imidacloprid reduced the flea index of a Norway rat focus from 12.9 to 2.3 (82%) in 48 hours, and to 1.5 (88%) 7 days later.

KEY WORDS: ectoparasites, fleas, imidacloprid, insecticides, Norway rats, public health, *Rattus norvegicus*, *Xenopsylla cheopis*

Proc. 24th Vertebr. Pest Conf. (R. M. Timm and K. A. Fagerstone, Eds.)
Published at Univ. of Calif., Davis. 2010. Pp. 333-336.

INTRODUCTION

Controlling fleas is an important public health consideration when addressing rodent populations associated with the bacterial zoonoses, plague and murine typhus (APHA 2004). The etiologic agents of plague and murine typhus, *Yersinia pestis* and *Rickettsia typhi*, respectively, are distributed worldwide and are maintained primarily in rodent hosts and their fleas (Gage et al. 1995). Other vertebrate hosts can be involved, especially with *R. typhi*, a typhus group rickettsia (Williams et al. 1992, Azad et al. 1997). Rodent fleas become infected with *Y. pestis* or *R. typhi* by feeding on bacteremic/ rickettsemic rodent hosts, and can subsequently transmit the bacteria to other rodents and incidental hosts (Azad 1990, Gage and Kosoy 2005). Additionally, vertical transmission has been demonstrated with *R. typhi* in a common commensal rodent flea, *Xenopsylla cheopis*, the oriental rat flea (Azad et al.1985). The risk of human infection can be elevated when rodent numbers are reduced by disease or rodenticides, as fleas can seek alternative hosts after losing their preferred host (Gratz 1999, Azad et al. 1997).

Although the focus of this paper is on rodent flea control, we note here that a spotted fever group rickettsia, *R. felis*, has been associated with previous murine typhus investigations involving non-rodent hosts and fleas, especially cat fleas (*Ctenocephalides felis*) in areas where antibody positive rodent reservoirs and rodent fleas are scarce (Williams et al. 1992, Sorvillo et al. 1993, Schriefer et al. 1994). It has become the subject of much study, and is now considered the etiologic agent of a typhus-like illness referred to in the literature as flea-borne spotted fever, cat flea rickettsiosis and others (Karpathy et al. 2009, Civen and Ngo 2008, Pérez-Osorio et al. 2008, Eremeeva et al. 2008, Reif and Macaluso 2009). Furthermore, rodent fleas (*X. cheopis*) infected with both *R. typhi* and *R. felis* have been reported (Eremeeva et al. 2008).

One rodent host/flea relationship, the Norway rat (*Rattus norvegicus*) and the oriental rat flea, is associated with both plague and murine typhus. Gratz (1999) noted that *X. cheopis* is the most important vector of plague and murine typhus. Regarding murine typhus, this relationship is described as the classic urban cycle. It effectively maintains *R. typhi*, as both are susceptible to infection, but it does not cause illness in either the rat or the flea. The rat can also serve as an amplifier, providing fleas with infectious blood meals (Azad 1990). As mentioned earlier, the flea itself can play a maintenance role as well via vertical transmission. Plague, however, affects both the flea and the rat and can cause mortality in both (Burroughs 1947, Sebbane et al. 2005). As such, they are more associated with epizootic transmission (Gage et al. 1995).

Murine typhus is widely distributed in coastal areas and port cities around the world, typically where humans and *Rattus* species rodents and their fleas interact (Azad et al. 1997). It was prevalent in the United States in the early to mid 1900s, especially the southeastern states and southern California, including Los Angeles, but was greatly reduced by a concerted rodent and flea control effort initiated in 1945 (Mohr et al. 1953). Currently, the majority of cases in the United States, typically <50 annually, are in California and Texas, as well as out-

breaks in Hawaii (Manea et al. 2001, CDC 2003, Eremeeva et al. 2008). Except for Hawaii, which has evidence of rodent/rodent flea involvement, many of these are likely associated with non-rodent/flea hosts as previously mentioned (Civen and Ngo 2008, Reif and Macaluso 2009).

Although human plague was historically associated with commensal rats, plague cases today are mostly associated with sylvan rodents, but there are some isolated exceptions outside the United States (Perry and Fetherston 1997). In the United States, human plague has not been associated with urban rats since 1924-25, when the last epidemic of pneumonic plague epidemic (32 cases) occurred in Los Angeles, along with 7 bubonic plague cases (Dickie 1926). Since then, plague has become endemic in sylvan rodents in the western states, especially the southwest (Gratz 1999). However, in a report of plague ecologic complexities in Los Angeles, Nelson et al. (1986) noted the potential of plague being introduced from the sylvan cycle into the inner city where Norway rats and oriental rat fleas exist.

It was established by the mid 1900s that reducing the flea burden can result in reduced transmission of *Y. pestis* and *R. typhi* between rodents and to humans. For example, in an evaluation of domestic rat flea suppression operations using DDT to control murine typhus in southern United States, Hill et al. (1951) reported marked decreases in confirmed murine typhus cases, flea infestation rates of rats, and antibody positive rats. Pollitzer (1953) reported reduced human and rat plague with the use of DDT in a number of areas of the world. In a later report of plague control in the United States, Poland and Barnes (1970) noted that flea control measures with insecticides are the primary method of preventing or controlling plague epizootics associated with human exposure, largely by burrow dusting and insecticide bait stations.

Recent field studies regarding flea control in rodents associated with plague in southern California have been conducted. Mian et al. (2004) found good initial control of the fleas of California ground squirrels (*Spermophilus beecheyi*) in San Bernardino County, using deltamethrin dust. In a different approach, the use of feed-through insecticides have also been evaluated in the field. Davis et al. (2008) conducted a study at a plague endemic campground in Ventura County. A chitin synthesis inhibitor was delivered orally using feed cubes over a 6-year period. Reduced flea loads were noted in some native rodent species. In another study of California ground squirrels, Borchert et al. (2009) used grain bait containing the neonicotinoid insecticide, imidacloprid, over a much shorter time frame of 15 - 29 days. The flea burden was essentially eliminated within 15 days.

In this field study, we attempted to extend the knowledge of this flea control strategy into the southern California urban setting. We examined changes in the flea index of a Norway rat focus in a dense urban area, using the same active ingredient used by Borchert et al., imidacloprid, but over a shorter time frame of 2 - 9 days.

SITE DESCRIPTION

The study site was located in a dense urban area of downtown Los Angeles, California, consisting of a long, rectangular planter surrounded by a cement perimeter, heavily infested with Norway rats (estimated at a minimum of 75 rats). A single primary food source consisting of a refuse storage area was located 13 m away. The planter was adjacent to an asphalt parking lot, which was surrounded by mixed-used commercial buildings.

The planter, approximately 25 m long and 1 m wide, had previously contained trees and was covered by heavy gauge 8-mm diamond mesh wire, secured around the concrete perimeter that had effectively rat-proofed the planter. However, trunk and root growth, along with wear and tear and lack of maintenance, left openings in the wire mesh in some areas that allowed rodent access. The trees were later cut at the base, leaving a non-irrigated planter with rat burrows and runs covered by thick wire mesh firmly secured to the perimeter in most sections, but some areas had rodent access.

MATERIALS AND METHODS

The insecticide bait formulation consisted of grain/flour/binder blended pellets, approximately 5 mm in diameter and 12 mm long, containing 0.020% imidacloprid. Active ingredient concentration was verified using High Performance Liquid Chromatography. The formulation was provided by Genesis Labs and Scimetrics, Ltd. Corp., Wellington, Colorado. Bait was applied, to allow feeding ad libitum for at least 2 days, below the wire mesh at well-secured sections that provided very good tamper-resistant protection from access by non-targets, but visible so as to assess acceptance. The pre-existing food source, a refuse storage area, was not altered.

Flea index surveys were conducted by trapping rodents using Tomahawk live traps (14 × 14 × 41 cm) (Tomahawk Live Trap Co., Tomahawk, WI) baited with commercial dog biscuits dipped in peanut butter. Traps were set in the early evening on July 31, 2008, for a pre-treatment flea index. Following bait application on August 5 and 6, early evening post-treatment flea index surveys were conducted on August 7 and 14.

Fleas were collected by brushing and combing the pelage of euthanized rodents, using carbon dioxide gas as described by Ramirez et al. (1991). Briefly, each trapped rodent was placed inside a large plastic bag, which was then filled with carbon dioxide gas. After the rodent was fully anesthetized, it was removed from the trap and the trap removed from the bag. The bag was refilled with carbon dioxide gas and left for 30 minutes to euthanize the rodent and inactivate the fleas. The carcass was then brushed and combed while positioned within the opening of the bag, allowing fleas to fall into the bag. The collected fleas were preserved in alcohol, and then counted and identified using standard taxonomic keys.

Because of the dynamic nature of the sampling areas within downtown Los Angeles, a concurrent control group was not able to be obtained. However, historical flea index data by month from 40 unrelated surveys conducted from June 2003 through December 2007 were used as a control. This group consisted of 378 Norway rats and 1,743 fleas. The majority of trap sites of this

control group were located within 1.6 km of the study site, mainly in urban alleys associated with refuse containers. Rodent trapping materials and flea collection methodology were identical to that of the study site.

Flea index for the study site was determined by the mean number of fleas per rodent per survey. For the control group, flea index by month was determined by the mean number of fleas per rodent collected during a given month. Trap success, percent infestation, flea index, and range values were calculated for pre-treatment and post-treatment surveys.

Table 1. Flea index, percent infested and trap success of Norway rats at pre-treatment and first and second post-treatment surveys, Los Angeles, CA, 2008.

Survey	Flea Index (Range)	Percent Infested	Trap Success
Pre-treatment	12.9 (6-23)	100%	90% (9/10)
1st Post-treatment	2.3 (0-5)	82%	92% (11/12)
2nd Post-treatment	1.5 (0-4)	87%	125% (15/12)

Table 2. Flea index by month of Norway rats in control group (2003 - 2007) and study group (pre-treatment July, post-treatment August 2008), Los Angeles, CA.

Month	Control Group Flea Index by Month	Study Group Flea Index by Month
Jan	0.5	
Feb	0.8	
Mar	1.7	
Apr	1.3	
May	3.1	
Jun	6.8	
Jul	7.4	12.9
Aug	10.1	1.8
Sep	7.7	
Oct	7.2	
Nov	1.8	
Dec	1.5	

RESULTS

Bait acceptance, assessed by visual estimates, was good. Initially, 1.7 kg of bait was applied on the evening of August 5. The following day, after observing approximately 2/3 of the bait consumed, a second and final application of 1.1 kg was made. Approximately 50% of the bait remained on the evening of the first post-treatment survey and approximately 20% was visible after the second post-flea index survey, mainly broken smaller pieces and dust.

The pre-treatment survey resulted in 9 Norway rats from 10 traps, 100% infestation, and a flea index of 12.9 (116/9, range 6 - 23). The first post-treatment survey yielded 11 Norway rats from 12 traps, 82% infestation, and a flea index of 2.3 (25/11, range 0 - 5). Twelve traps were set for the second post-treatment survey, resulting in 15 Norway rats (3 double captures), 87% infestation, and a flea index of 1.5 (23/15, range 0 - 4) (Table 1). Percent reduction of the pre-treatment flea index was 82% for the first post-treatment survey and 88% for the second post-treatment survey.

The control group analysis of flea index by month revealed seasonal fluctuation. From January to April, the flea index was <2.0. It progressively rose to 7.4 in July, peaked at 10.1 in August, and then declined to 1.5 in December. Examining the study site in terms of flea index by month, the flea index in July (pre-treatment) was 12.9, and combining the 2 post-treatment surveys in August, declined to 1.8 (Table 2). All fleas from the study group and the control group were identified as *X. cheopis*.

DISCUSSION

The value of this flea control strategy is that it provides an additional method to control rodent fleas, especially where dust or residual sprays are not appropriate or feasible. This could be used as a pre-bait material, followed by a rodenticide bait. This would reduce the number of rodent fleas seeking an alternative host as their rodent host dies, thereby reducing the potential for transmission of a flea-borne pathogen if one were present. Moreover, this study suggests that the flea index can be markedly reduced with rodent bait containing imidacloprid within a time frame prior to the lethal effects of anticoagulant rodenticides, which were developed to overcome bait shyness by having a delayed effect (Poché 1986, Hadler and Buckle 1992). Therefore, a formulation containing imidacloprid and an anticoagulant rodenticide may also be a valuable material. By combining the steps of rodent and flea suppression, both in materials and methods, it would produce a more comprehensive treatment without additional materials, procedures, or personal protective equipment.

This field study demonstrated a marked reduction in the flea index of a Norway rat focus in 48 hours, as compared to historical flea index values for the same monthly period of July/August, using a rodent bait containing imidacloprid. Previous field studies in southern California on feed-through insecticides for the control of rodent fleas have been conducted with native rodents in a rural-type setting. These data extend our knowledge of this type of control strategy into the southern California urban environment with Norway rats, an Old World rodent species associated with flea-borne pathogens of public health importance. To our knowledge, this is the first field study in the United States assessing this type of flea control method in an urban setting.

ACKNOWLEDGEMENTS

We express our sincere thanks to Robert Flores and Pedro Gutierrez of the Los Angeles County Department of Public Health, Vector Management Program, for assisting in specimen collection.

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