Chapter 6

Colony differences in termiticide transfer studies, a role for behavior?

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Abstract. Donor-recipient termiticide transfer laboratory tests were performed by using destructive sampling with two delayed-action non-repellent (DANR) termiticides against each of three colonies of *Reticulitermes flavipes* (Kollar). Two of the three colonies showed no response to indoxacarb, but all three showed a response to chlorantraniliprole. These results indicate that behavioral variation among colonies is not likely responsible for the variability in recipient mortality among colonies noted in transfer studies in the literature. Donor mortality with these compounds and colonies suggests that variable physiological susceptibility of individual colonies to certain compounds may be more important than variations in behavior.

Introduction

Over the past several years, laboratory studies on the movement of termiticides among termites have been reported in the pest control (*1, 2*) and scientific literature (*3 - 10*). These reports have generally found that delayed-action non-repellent (DANR) termiticides are capable of movement among termites, while more traditional repellent compounds do not move among those termites directly exposed (*11*).

Recent studies at the University of California, Riverside (*8, 10*) have indicated that for some compounds, lethal termiticide transfer can only happen via primary transfer. In other words individuals exposed to a toxicant (donors) may pass it to naïve termites (recipients), but those recipients do not become secondary donors themselves, mainly due to the limited amount of toxicant available from the original donor (*8*). There may also be a location component to this situation: termites exposed to treated soil (donors) would have the

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toxicant coating their cuticle, whereas recipients picking up the toxicant via grooming and/or trophallaxis would ingest the materials, thus making them unavailable for movement via grooming. However, transfer via trophallaxis, or proctodeal feeding could still occur.

Toxicant transfer has only been documented in the laboratory, although anecdotal evidence from field studies has been used to support its occurrence in the field (*1, 12*). Without direct evidence of the effects of termiticide transfer on field populations there are serious doubts about the importance of transfer in real-world control situations. This is perhaps best demonstrated in a field study by Osbrink *et al.* (*13*), in which soil application of imidacloprid a DANR termiticide previously shown to transfer among *Coptotermes formosanus* Shiraki individuals in the laboratory (*6*), did not lead to population suppression of *C. formosanus* consistent with what the authors termed a liquid-bait model. For the purposes of this chapter, the idea of "termiticide transfer" is limited to mortality induced by the movement of soil-applied chemicals and not termiticidal baits. Bait products are designed to be consumed by termites and spread throughout colonies via social interaction, whereas the transfer discussed in this chapter consists of movement of chemicals from the soil (not necessarily consumed) to exposed and eventually to naïve termites (almost an "accident" in terms of product design). For information on the movement of a bait toxicant (hexaflumuron), the reader is referred to Sheets *et al.* (*14*). Unfortunately the most useful tool for examining transfer is the use of radiolabelled termiticides, which are unikely to receive approval for use in field experiments. Transfer of termiticides also may not have large effects on foraging populations as mortality in these laboratory studies can be quite low (*6, 9*). To some extent these problems have made termiticide transfer into more of an academic curiosity, a phenomenon to be studied certainly, but not to be relied upon for termite control.

Because there are problems with field observations of transfer, although such data are sorely needed, examinations of transfer are best suited to laboratory work. Some authors have chosen to examine termite colony origin in relation to termiticide transfer (*4, 6, 11, 9*). Differences in recipient mortality among colonies exposed to the same concentrations of single toxicants could have a number of possible explanations (*3, 6*). In previous papers, termite body mass did not predict susceptibility to toxicant transfer (*i.e.*, low body mass was not associated with increased susceptibility, nor vice versa). Dosage may also be an issue with studies where termites are left to walk across treated surfaces, as there is little guarantee of the consistency of the dose received (compared with topical applications). Topical applications, however, unless based upon known concentrations picked up by termites interacting with treated soil, are themselves rather arbitrary. It has been suggested that behavioral differences among colonies may lead to such differences in horizontal transmission mortality (*15, 14, 3, 6*), keeping in mind that recipient mortality depends upon the movement of toxicants from donors to recipients over the course of the study. It is easy to imagine how the rates of behaviors such as grooming or trophallaxis could influence the rate at which toxicants are passed among individuals, with particularly low rates possibly leading to lack of transfer altogether.

How can the impact of intercolony behavioral differences be tested? One way of testing this hypothesis involves making a simple assumption that these behavioral rates are consistent within colonies, but not necessarily among colonies. It should be kept in mind that it would be unreasonable to assume that some colonies simply do not engage in all social behaviors (assuming Occam's razor has taken out any unnecessary behaviors via evolution), however different colonies might very easily vary in their rates of conducting these behaviors.

Work with lower termites (Termopsidae, Kalotermitidae) has indicated that all non-larval (*i.e.* third instar and above) "workers" or pseudergates seem to take on equivalent roles (engage in the same sets of behaviors) within colonies (*16, 17*). There is a little evidence here for temporal division of labor; Howse (*16*) found that within the pseudergate caste of *Zootermopsis nevadensis* (Hagen), first and second instars exhibited little to no behaviors aside from trophallaxis, and that the amount of time spent in trophallaxis for all stages had a weak inverse correlation with time spent in other activites such as building or digging. Although no inference testing was done, some of the non-trophallaxis behaviors (building and oscillatory movements) increased with pseudergate instar, but others remained steady (digging). In general the sixth instar performed most of the colony "work" activities (*16*). The concepts suggested by Howse (*16*) were verified in papers by Crosland and colleagues (*18, 19*) with *Reticulitermes fukienensis* Light, indicating that all pseudergates perform the same behaviors, but the rates of those behaviors varied with age class. *Reticulitermes flavipes* (the model insect for this paper) often includes larval termites in the foraging populations (*20*), however most laboratory studies with this species do not include larval stages or nymphs (wing-pad bearing pre-alates), unless otherwise noted. Additionally, one might expect the age class distribution of a colony to be somewhat steady at any given point in time, thus as long as only pseudergates are counted (no larvae, nymphs) without bias into groups for an experiment, those groups (for a given colony) should be fairly similar in terms of age class distribution. Thus, if there were a temporal division of labor in *R. flavipes*, it would be unlikely to invalidate the assumption due to experimental methodology. Testing the hypothesis above requires looking for intracolony differences in recipient mortality in transfer studies with toxicants that have been previously shown to transfer among termites, using the same colonies (and thereby keeping the rates constant).

Saran and Rust (*10*) provide some insight into what behaviors might be most important in toxicant transfer. They examined movement of fipronil among *R. hesperus* Banks whose mouthparts had been sealed with glue. Saran and Rust (*10*) conclude that movement of the toxicant did not rely on trophallaxis at all, only body contact. However, with sealed mouthparts, other behaviors, such as grooming, would also be impeded, and this was attributed to the movement of imidacloprid in Tomalski and Vargo's study (*2*). These researchers also found that corpses of termites killed with fipronil were toxic enough to kill recipients, depending on concentration. Their data indicate trophallaxis and proctodeal feeding are not necessary for transfer with *R. hesperus*, although it is still possible that these behaviors (in addition to grooming) might accelerate the movement of termiticides.

From the arguments above, it can be assumed that any given colony will have constant rates of trophallaxis/grooming that might allow for transfer to occur. The hypothesis is that these behaviors have some influence on the rate of recipient mortality due to insecticide transfer and are responsible for the differences among colony mortalities noted in previous studies (alternate hypothesis; H_a). To make this a testable hypothesis, we need something to examine: two pesticides capable of being transferred as toxicants against a single colony in a simple donor-recipient transfer laboratory study (with replications being the testing of new colonies). If the hypothesis stated is correct, then both compounds will produce similar results for each individual colony. Finding non-relative differences (i.e. not just a small difference in percent mortality, but rather that one compound has an effect and the other does not) in each colony's response to individual compounds would not support the alternative hypothesis of behavior influencing recipient mortality and would indicate that some other factor is responsible for the differences noted in previous studies (null hypothesis; H_0).

The following study investigates this idea using two new termiticides produced by E. I. DuPont de Nemours, Inc. The first is an oxadiazine compound known as indoxacarb, and the second is chlorantraniliprole (class anthranilic diamide). The literature provides some information on transfer with indoxacarb (*7*) against another subterranean termite species, *C. formosanus*. Both compounds are capable of being transferred by *Reticulitermes flavipes* (Kollar) using 5% of the test population as donors exposed to 100 ppm of toxicant treated sand, as determined in preliminary studies.

Methods and Materials

Termites. Termites were collected by removing infested logs (cut into manageable sections) from active termite colony sites in the John W. Starr forest (maintained by Mississippi State University), the Noxubee National Wildlife Refuge (maintained by the U.S. Fish and Wildlife Service), and the campus of the USDA Forest Service facility in Starkville MS (all termite colonies were collected within 15 miles of Starkville). Log sections were placed into 30 gal. (114 L) metal trash cans, and returned to the laboratory, where cans were kept under ambient conditions (~24 C) until use. Termites were identified from morphological soldier characters by using the key of Hostettler *et al.* (*21*).

The studies were simple donor-recipient mortality studies run for two weeks. However, to get a more detailed view of mortality over this time period, the tests were run by using destructive sampling, with 6 replicates from each treatment (controls and 100 ppm pesticide) broken down and surviving termites counted on every other day during the test period (2, 4, 6, 8, 10, 12, and 14 days after treatment). Due to the number of replicates needed for this sampling method, only two treatments per compound were included (a distilled water-only control, and a 100 ppm pesticide treatment). For each colony, tests for each compound were run in separate incubators (25 ± 1 °C; ~75% R.H.) with separate control groups for each compound (*i.e.*, indoxacarb replicates + indoxacarb controls in incubator 1, chlorantraniliprole + control replicates in

incubator 2). For each colony, 168 experimental units (jars) were necessary. Data sets were collected for both compounds using each colony.

With the exception of the destructive sampling, the methods for each study were a modification of those previously published (*9*). Donors were stained by feeding them filter papers (Whatman #2, Whatman International Ltd., Maidstone, United Kingdom) stained with Sudan Red 7B (0.5% wt./wt.; Sigma-Aldrich co., St. Louis, MO; *22*) for one week prior to the start of the test. Staining took place in Petri dishes (9 cm dia.) provided with two stained filter papers, moistened with 1 ml of distilled water each, containing 200-250 termites (mixed caste) and incubated at 25 ± 1 °C, ~75% R.H in an unlit incubator. Arenas were standard 8 cm diameter × 10 cm tall screw top plastic Quorpak jars, filled with 150 g of silica sand (Fisherbrand; Fisher Scientific, Pittsburgh, PA), and moistened with 27 ml of distilled water. On the test initiation day, recipient termites were counted fresh from the cans into groups of 95 workers only, and one group was placed into each jar. Donor termites were counted into six groups of 100 workers and placed in petri dishes containing 25 g treated sand (three dishes per treatment; either water only or 100 ppm wt./wt. of pesticide/sand), which was provided with 6 ml of distilled water 3 hrs prior to adding termites (to allow for evaporation). Donor groups spent 1 hr on the treated sand (consistent with previous papers on this subject: *6, 11, 9*) before being moved to clean petri dishes containing only a single dry filter paper for 30 min (this allowed any sand attached to the donors to dislodge). Finally, donors were placed into jars according to treatment, at a rate of 5 donors per jar. On breakdown days (described above) jars were emptied onto plastic trays and surviving donor (as stained individuals) and recipient workers were counted and recorded.

Statistically, each compound + control grouping (per colony) was considered separately, with percentage recipient mortality transformed by the arcsine of the square root and subjected to a general linear model procedure (GLM, *23*). Concentration of pesticide, day of test, and their interaction were investigated for influence on recipient mortality. Of these, the most important measure is that of concentration, which indicates whether transfer of the pesticide led to mortality of the recipients. Certainly, transfer which does not lead to recipient mortality cannot be measured using these methods, but sub-lethal movement of pesticides is not the metric being examined here.

Results

Figures 1 and 2 illustrate mean ± SEM percentage mortality for donors (Figure 1) and recipients (Figure 2) by colony and compound for these studies. Details of each colony's response to both compounds are given separately below.

Colony 1. Donor mortality shows a trend with chlorantraniliprole increasing donor mortality until roughly day 6, then leveling off (Figure 1). Indoxacarb treated donors begin showing mortality at day 10 then leveling off below 40% (Figure 1). Mortality of donors from colony 1 was significantly influenced by concentration of both insecticides (chlorantraniliprole: dF = 1, 83;

F = 289.69; $P < 0.0001$; indoxacarb: dF = 1, 83; F = 12.12; $P = 0.0009$). Concentration of chlorantraniliprole significantly influenced recipient mortality of colony 1 workers compared to controls during this study (dF = 1, 83; F = 58.29; $P < 0.0001$), but concentration of indoxacarb did not significantly influence recipient mortality (dF = 1, 83; F = 3.18; P = 0.0792) in comparision to untreated controls. Recipient mortality increased until day 6 for chlorantraniliprole treated replicates, and leveled off after that point (Figure 2), but did not increase with indoxacarb over the 14 day test (Figure 2) for colony 1 termites. Recipient mortality with both compounds was not significantly influenced by day of test (chlorantraniliprole: dF = 6, 83; F = 1.85; P = 0.1020; indoxacarb: dF = 6, 83; F = 2.15; P = 0.0584). The interaction of concentration by day significantly influenced recipient mortality only for indoxacarb for colony 1 workers (dF = 6, 83; F = 2.31; $P = 0.0429$; chlorantraniliprole: dF = 6, 83; F = 1.60; $P = 0.1594$).

Colony 2. During breakdown of replicates for this colony, some individuals in five replicates were noted to have a bright red coloration commonly associated with *Serratia* sp. infection (note that Sudan Red staining results in a much deeper red color). These termites only showed up in five replicates of the chlorantraniliprole treatment (two on day six, one on day eight, and two on day 12). These replicates were left out of the analysis, as well as Figures 1 and 2.

Donor mortality for colony 2 is quite similar to the response of colony 1, with chlorantraniliprole donor mortality leveling off by day 6 (reaching 100% by day 10; Figure 1). For indoxacarb, donor mortality is slightly increased above that of control recipients, eventually overlapping on day 14 (Figure 1). As with colony 1, colony 2 donor mortality was significantly influenced by concentration for both compounds (chlorantraniliprole: dF = 1, 78; F = 382.85; $P < 0.0001$; indoxacarb: dF = 1, 78; F = 10.68; $P = 0.0017$). Concentration of chlorantraniliprole significantly influenced recipient mortality of colony 2 workers (dF = 1, 78; F = 133.93; $P < 0.0001$). Indoxacarb did not significantly influence recipient mortality of colony 2 workers (dF = 1, 78; F = 2.08; $P = 0.1539$), also similar to the results obtained with colony 1. Chlorantraniliprole treatment recipients never seemed to reach a plateau for colony 2 termites, although the slope of the data changes at around day 10 (Figure 2). For indoxacarb, recipient mortality essentially mimics control recipient mortality for the entire duration (Figure 2). For colony 2 workers, day of test significantly influenced recipient mortality for both compounds tested (chlorantraniliprole: dF = 6, 78; F = 6.63; $P < 0.0001$; indoxacarb: dF = 6, 78; F = 16.94; $P < 0.0001$). For colony 2 workers the interaction of day and concentration significantly influenced recipient mortality only for chlorantraniliprole (dF = 6, 78; F = 3.06; $P = 0.0106$; indoxacarb: dF = 6, 78; F = 0.28; $P = 0.9432$).

Colony 3. Donor mortality (Figure 1) follows the same path for both indoxacarb and chlorantraniliprole for colony 3 termites, in that both reach a maximum (100% mean donor mortality) on day 6, which holds for the remainder of the study. As with both other colonies, colony 3 donor mortality was significantly influenced by concentration for both compounds (chlorantraniliprole: dF = 1, 83; F = 332.42; $P< 0.0001$; indoxacarb: dF = 1, 83; F = 511.40; $P < 0.0001$). As with colonies 1 and 2, chlorantraniliprole

concentration significantly influenced recipient mortality of colony 3 workers (dF = 1, 83; F = 85.43; $P < 0.0001$). Colony 3 responded differently to indoxacarb than colonies 1 and 2, in that indoxacarb concentration significantly influenced recipient mortality of colony 3 workers (dF = 1, 83; F 164.39; $P < 0.0001$). Figure 2 indicates that in indoxacarb treatments, recipient mortality increases until roughly day 6, when it plateaus for several days, increasing again on the final day (day 14). Recipient mortality in chlorantraniliprole treatments spikes fairly early (day 4) with colony 3 termites, and then falls to a plateau for the remainder of the study (Figure 2). For colony 3 workers, day of test with both compounds significantly influenced recipient mortality (chlorantraniliprole: dF = 6, 83; F = 2.80; $P = 0.0167$; indoxacarb: dF = 6, 83; F = 9.45; $P < 0.0001$). As with colony 1 workers, colony 3 worker recipient mortality was significantly influenced only by indoxacarb in the day by concentration interaction (dF = 6, 83; F = 2.49; $P = 0.0306$; chlorantraniliprole: dF = 6, 83; F = 1.24; $P = 0.2948$).

Discussion

Figure 2 illustrates recipient mortality during these studies, with indoxacarb data in the left column and chlorantraniliprole data in the right column. By viewing each colony's response to these compounds individually, it is obvious that colony 1 and colony 2 did not respond in a similar manner to both compounds. The GLM analysis of these data confirm that both colonies' recipient mortality was significantly influenced by exposure to donors treated with chlorantraniliprole only. Colony 3 recipient termite mortality was significantly influenced by both indoxacarb and chlorantraniliprole treated donors. There are two possibilities suggested by these results: a) the initial assumption regarding the consistency of behavioral rates within any given colony is not correct for *R. flavipes* workers, or b) intercolonial variability in recipient mortality in transfer studies previously reported is not due to variations in behavioral rates among colonies.

While the behavioral rate consistency assumption seems plausible, there have been no attempts to determine the rate differences (if any) among the workers within these colonies. Evidence from other lower termites suggests that this is not an unreasonable assumption. Studies with *Z. nevadensis, R. fukienensis,* and *Kalotermes flavicollis* (Fabricius) indicate that workers engage in similar behavioral capacities within fairly broad age groups (*16, 18, 19, 17*). In other words, termites beyond the 2nd instar are engaged in similar activites as other workers up to the pre-alate nymph stage (*18, 19, 17*). It should be noted that this assumption would certainly be invalid for some social Hymenoptera (*24*). The possibility of temporal division of labor as suggested earlier remains, although the age class distribution of groups counted from these colonies should have been similar within each colony as discussed in the introduction. Variations in the performance of behaviors have been noted between colonies for behaviors such as tunnel building (*25*) and agonism (*26 - 28*). However, variations in rates of behaviors within single castes of individual colonies would likely be absorbed in the error term in most studies. Perhaps this area deserves more careful study.

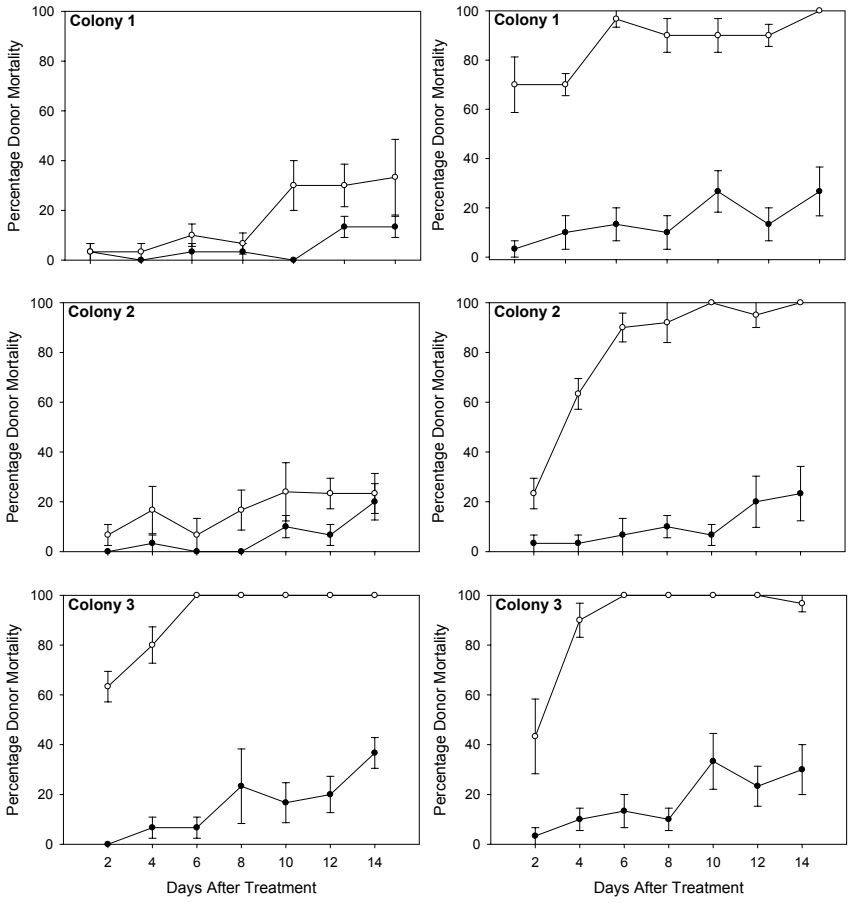

Figure 1. Donor mortality over time for each colony. Indoxacarb results are on the left and chlorantraniliprole results on the right in each column. Each data point is a mean ± SEM of 6 experimental units. For all graphs: ● are 0 ppm donors, and ○ are 100 ppm donors.

Failing to reject the consistency assumption as inaccurate, only the second possibility remains that intercolonial recipient mortality variability is not due to a vaguely defined behavioral variability among colonies. Other authors have already rejected the idea that this variability is correlated with body mass variability (*4, 6*). This presents a different problem: if the data given here suggest that behavioral variability among colonies is not the source of recipient mortality variation in transfer studies, then what is responsible? An explanation may be present in the data from the current study. Donor mortality also varies among colonies in these studies, but is consistent with the variation seen in the recipient mortality. In other words, the donors (who have been directly exposed

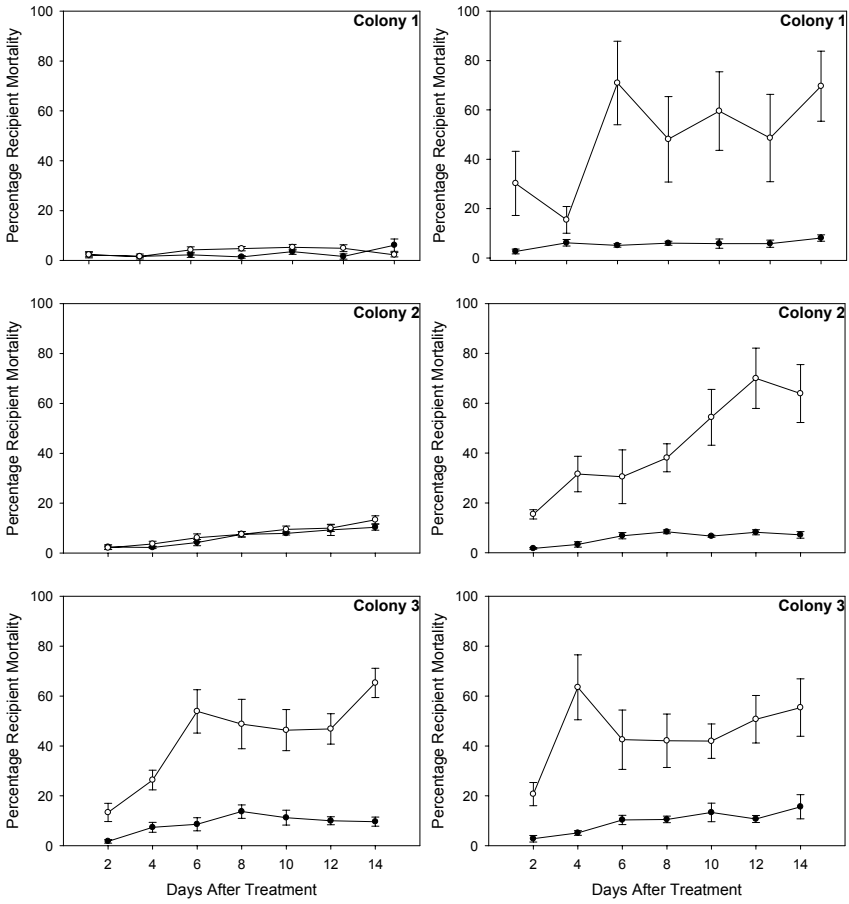

Figure 2. Recipient mortality over time for each colony. Indoxacarb results are on the left and chlorantraniliprole results on the right in each column. Each data point is a mean ± SEM of 6 experimental units. For all graphs: ● are 0 ppm donors, and ○ are 100 ppm donors.

to the termiticides) in colonies 1 and 2 are not effected as quickly as colony 3 donors, nor to the extent of colony 3 individuals (100% donor mortality at day 14 for colony 3 versus. < 40% donor mortality for colonies 1 and 2 at day 14). Admittedly, donor mortality for all three colonies was significantly influenced by both compounds over the entire course of the study. However, the data (Figure 1) indicates a very weak influence over controls for colony 2, and only slightly stronger for colony 1. In all, it appears that colonies 1 and 2 were less susceptible to indoxacarb, both when directly exposed (donors) and when exposed through transfer (recipients). The concept of variations in susceptibility

among termite colonies has been investigated by Osbrink *et al.* (*29*) for *R. flavipes* and *C. formosanus*.

One of the qualifications when dealing with mortality as a measurement is the problem of dealing with unhealthy colonies, a question of colony vigor. It has been suggested that perhaps termite colony vigor is not binary (*i.e.*, healthy or not healthy), but is instead a spectrum ranging from very healthy colonies to very unhealthy colonies (*30*). It is possible that any variability observed in mortality among stressed individuals could possibly be the result of a slight stress acting in concert with low vigor to induce mortality. The opposite should also be true, where slight stresses may not induce mortality in very healthy colony members. Vigor-related influences in studies such as this are difficult (if not impossible) to distinguish unless the colony is in such poor health that high mortality occurs in the controls, otherwise mortality appears to result completely from the influence of treatment. However, the possibility of vigor differences bears mentioning whenever "colony effects" are noticed in termite studies. Recent studies have examined both means of determining vigor in laboratory termite colonies (*31*), as well as surveyed possible variables for this purpose (*32*). In the current study, control recipient mortality (Figure 2) did not indicate reduced vigor in the colonies.

Interestingly, while two colonies did not respond to indoxacarb in this study, no colonies were unresponsive to chlorantraniliprole. While both of these compounds are SC formulations, they belong to different classes and have different modes of action. Additionally, indoxacarb is a pro-insecticide, and is less toxic than its *N*-decarboxymethoxyllated metabolite (*33, 34*). Activity for indoxacarb is greatest when ingested by Lepidopteran larvae; and LD_{50}'s for oral *vs*. topical applications vary almost three-fold for certain larval Lepidoptera, although this does not hold for Coleoptera (*34*). If Saran and Rust (*10*) are correct about trophallaxis, it would be expected that only the parent compound of indoxacarb, rather than the toxic metabolite, is moved by recipients grooming donors. Indoxacarb's metabolite is active against insect Na^+ channels, disrupting action potentials (*34, 35*). In contrast the family that chlorantraniliprole belongs to, the anthranilic diamides, act against the ryanodine receptor (RyR) channels which control Ca^+ entry during muscle contraction events (*35*). Cordova *et al*. (*35*) examined 12 anthranilic diamides (of four classes) and compared them with indoxacarb. LD_{50}'s (oral) ranged from 0.4 to >500 ppm, compared with 0.6 ppm for indoxacarb in *Heliothis virescens* (Fabricius) larvae (*35*).

It is difficult to see an obvious reason for the apparent variability in susceptibility among colonies with indoxacarb that would not apply to chlorantraniliprole. It may be that the time necessary to convert indoxacarb from parent to metabolite differs among colonies, although recipient mortality for colonies 1 and 2 give no indication of increasing (beyond that of controls) even by day 14. Perhaps more detailed examinations of relative toxicity (oral and topical) of both compounds against subterranean termites is needed.

This chapter has investigated the source of the intercolonial variability in recipient mortality observed in toxicant transfer studies against subterranean termites in the laboratory. In summary, intercolonial differences in behavior are unlikely to be responsible for this variation in mortality. Instead it would appear

that colonies vary in their physiological susceptibility to the compounds (as seen in directly exposed individuals), and that the absence of mortality is not necessarily correlated with either the presence or absence of transfer. Transfer may occur in colonies that are not susceptible to a particular toxicant, but it is not manifested by recipient mortality.

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