FORUM

Incorporating Ecologically Relevant Measures of Pesticide Effect for Estimating the Compatibility of Pesticides and Biocontrol Agents

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ABSTRACT The compatibility of biological control agents with pesticides is a central concern in integrated pest management programs. The most common assessments of compatibility consist of simple comparisons of acute toxicity among pest species and select biocontrol agents. A more sophisticated approach, developed by the International Organisation of Biological Control (IOBC), is based on a tiered hierarchy made up of threshold values for mortality and sublethal effects that is used to determine the compatibility of pesticides and biological control agents. However, this method is unable to capture longer term population dynamics, which is often critical to the success of biological control and pest suppression. In this article, we used the delay in population growth index, a measure of population recovery, to investigate the potential impacts that the threshold values for levels of lethal and sublethal effects developed by the IOBC had on three biocontrol agents: sevenspotted lady beetle, *Coccinella septempunctata* L.; the aphid parasitoid *Diaeretiella rapae* (M'Intosh), and *Fopius arisanus* (Sonan), a parasitoid of tephritid flies. Based on life histories of these economically important natural enemies, we established a delay of 1-generation time interval as sufficient to disrupt biological control success. We found that delays equivalent to 1-generation time interval were caused by mortality as low as 50% or reductions of offspring as low as 58%, both values in line with thresholds developed by the IOBC. However, combinations of mortality and reduction of offspring lower than these values (from 32 to 43% each) over a simulated 4-mo period caused significant population delays. Furthermore, the species used in these simulations reacted differently to the same levels of effect. The parasitoid *D. rapae* was the most susceptible species, followed by *F. arisanus* and *C. septempunctata*. Our results indicate that it is not possible to generalize about potential long-term impacts of pesticides on biocontrol agents because susceptibility is influenced by differences in life history variables. Additionally, populations of biocontrol agents may undergo significant damage when mortality approaches 50% or when there is mortality of ≈30% and a 30% reduction in offspring caused by a sublethal effect. Our results suggest that more ecologically relevant measures of effect such as delays in population growth may advance our knowledge of pesticide impacts on populations of beneficial species.

KEY WORDS IOBC, pesticides, biocontrol

One of the major goals of integrated pest management (IPM) is to obtain economic pest control with the judicious use of selective pesticides in concert with biological control agents. Often, biological control agents alone cannot provide economic control of a pest species; thus, supplemental use of a pesticide may be necessary to provide adequate control. However, how we determine compatibility of pesticides with biological control agents is a contentious issue (Stark and Banks 2003). Several methods have been proposed, and some are used regularly, but there is no standardized approach in the United States for determining whether a pesticide can be safely used with a biocontrol agent. Entomologists have often used selectivity ratios to determine whether an insecticide is compatible with a biocontrol agent. This approach entails dividing the acute LC_{50} (lethal concentration that kills 50% of a population) or LD_{50} of a biocontrol agent by the LC_{50} or LD_{50} of a pest species. Selectivity ratios >1 indicate that the insecticide is more toxic to the pest species than to the beneficial species (Respicio and Forgash 1984, Purcell et al. 1994). Although quick and inexpensive, this approach unfortunately ignores important factors known to impact population dynamics such as age structure, sublethal effects, and differences in life history traits among focal species (Banks and Stark 1998; Stark and Banks 2001, 2003; Stark et al. 2004a, 2004b).

Another common approach was developed by the International Organisation of Biological Control (IOBC) (Hassan 1992, 1998; Boller et al. 2005). This

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approach, most widely used in Europe, is based on a tiered system. Tier I consists of an initial screening done in the laboratory to develop toxicity data. The IOBC classifies pesticides into the following four categories, depending on the degree of damage that they cause to beneficial species in laboratory studies: 1, harmless (<30%); 2, slightly harmful (30–79%); 3, moderately harmful (80–98%); and 4, harmful (>99%). The evaluated endpoint of interest is either mortality or a reduction in beneficial capacity (parasitism or performance [e.g., oviposition]) compared with controls. If a pesticide falls within category 1, no additional testing beyond tier I is required (Vogt 2000, Boller et al. 2005). If a pesticide falls within category 2, it also may be suitable for IPM, but additional testing is recommended. Pesticides falling within categories 3 and 4 need additional testing with semifield tests (tier II). Categories for outcomes of tier II (semifield) tests are slightly different. For semifield studies, the following categories of effect are used: 1, harmless (<25%); 2, slightly harmful (25–50%); 3, moderately harmful (51–75%); and 4, harmful (>75%). As with tier I tests, endpoints for tier II are either outright mortality or some reduction in beneficial capacity. If the results of semifield tests fall within category 1 or 2, no additional testing is required, and the pesticide is acceptable. If the results fall within category 3, then full field tests (tier III) are required. If a pesticide falls within category 4, it is considered incompatible with IPM. Field testing (tier III) categories of effect are the same as those for tier II (semifield) studies.

Although the acceptable levels of mortality and/or reduction in beneficial capacity (up to 79%) seem high in the laboratory test scenario, the assumption is that laboratory exposure is a worst-case situation and that exposure in the field, and thus mortality and sublethal effects, will be lower.

Although the tiered approach advocated by the IOBC is based on a large body of literature (see Haskell and McEwen 1998 for several chapters on this subject), there are limitations to this approach for pesticide side effects assessment (Stark et al. 1995). The primary limitation is that only one or at most two toxic effects are considered. In reality, exposure to pesticides may result in a wide range of effects on an organism, including the simultaneous manifestation of multiple sublethal effects. For example, in addition to death, exposure to a toxicant can result in shortened life span, reduced number of offspring, changes in the time to first reproduction, longer generation times, weight loss, and mutations in offspring (Stark and Banks 2003). Chemical exposure also may induce subtle behavioral changes that result in a loss of sexual competitiveness, the loss of ability of predators to capture prey, or both. Together, these effects can lead to low predator population growth rates due to reduced mating success in small populations, a scenario (commonly referred to as an Allee effect) that may lead to further population decline and collapse (Stephens and Sutherland 1999, Stephens et al. 1999).

Recent field research and models have demonstrated that higher levels of natural enemies as well as the ability of natural enemies to search for prey are critical for biological control success (Kean et al. 2003, Snyder et al. 2006). Thus, exposure to pesticides causing both lethal and multiple sublethal effects may greatly undermine biological control.

Finally, most measurements of pesticide effect are taken on individuals, not populations, and multiple effects are not considered. If, for example, there are survivors after exposure to a pesticide, then theoretically the population will recover. This assumes that organisms are exposed to chemicals that do not have long persistence in the environment, a characteristic of most of our modern insecticides.

Despite extensive research on the effects of chemicals on the survival of nontarget organisms, there has not been enough focus on whether toxicants directly disrupt biological control at the population level. The compatibility of selective pesticides and biological controls is still often determined with short-term studies of individuals (Johnson and Tabashnik 1999). In particular, an important question that has not been adequately addressed is, To what degree can biological control agents withstand lethal or sublethal effects and still effectively suppress pest populations? To answer this question, it is necessary to carefully define acceptable levels of pest suppression. When pesticides are applied, both pest and natural enemy populations are often reduced. Reduction of pest populations by pesticides often results in a corresponding reduction in natural enemy populations, because they have a reduced or nonexistent food source. Thus, the combination of a reduced food source and toxic effects of pesticides can be devastating to biological control agents.

The IOBC tiered approach indicates that levels of mortality as high as 50% in a biocontrol agent are acceptable. But as discussed above, such measurements of acute mortality, such as the LC_{50}, can be woefully inadequate for determining the actual impact that pesticides might have on biological control.

It is our contention that, additional, more ecologically relevant measures of pesticide effect are needed to obtain more accurate estimates of pesticide impact on beneficial species (Van Straalen and Kammenga 1998, Forbes and Calow 1999, Stark and Banks 2003). Our work and that of others has shown that population-level measures of pesticide effect obtained from demographic studies provide better estimates than acute mortality measures (Forbes and Calow 1999, Stark and Banks 2001). Modeling based on demographic parameters provides a clearer picture of actual pesticide impacts (Stark et al. 2004b).

In previous studies, we have developed protocols for incorporating population processes directly into assessments of pesticide impacts (Wennergren and Stark 2000, Stark et al. 2004b). Our approach uses a measure of population recovery, referred to as the delay in population growth index, to capture nuances of population dynamics that are critical to biological control success. Because a delay in natural enemy recovery from a toxicological insult that lasts 1-generation time interval or more can drastically disrupt

Table 1. Life table parameters of the species evaluated

	Net reproductive rate (R_0)	Intrinsic rate increase (r_m)	Generation time (d)
C. septempunctata	72.767	0.085	50.233
F. arisanus	27.400	0.121	27.300
D. rapae	25.450	0.218	14.834

Life table data are from Stark et al. (2004b).

biological control, we think that population delay is a biologically meaningful way to measure pesticide impacts. We hypothesize that this approach should generate a more realistic assessment of pesticide effects than the use of predetermined "one size fits all" levels of mortality and sublethal effects such as those advocated by the IOBC. We present here a simple exercise comparing these two approaches, exploring the effects of pesticides on several biological control agents.

Materials and Methods

We evaluated three species in this study: sevenspotted lady beetle, *Coccinella septempuncata* L., a predator that feeds on aphids; the aphid parasitoid *Diaeretiella rapae* (M'Intosh) that attacks a range of aphid species; and *Fopius arisanus* (Sonan), a parasitoid of tephritid flies. These species were chosen for study because they exhibit differences in key life history parameters (Table 1).

We used the Delay in Population Growth Index (Wennergren and Stark 2000, Stark et al. 2004b) to determine potential damage to a population. The Delay Index is a measure of population recovery that compares control populations to those exposed to a stressor that results in either mortality, reductions in the number of offspring or a combination of these effects. When using the Delay Index, a population number is chosen as the endpoint of interest, and the time it takes for a stressed population to reach that number is compared with the time it takes a control (unstressed) population to reach that number. Therefore, this method measures the number of days a population's recovery is delayed. The Delay Index is based on an age-based matrix model that incorporates survivorship and fecundity probabilities developed from demography studies or from direct measurement of population data (Wennergren and Stark 2000). Population growth is simulated by multiplying a transition matrix with vital rates (survivorship and fecundity) by an initial population vector, n(t), that contains the number of individuals in each age class (Stark and Banks 2003). Each of the three species was modeled using four age classes, corresponding to eggs, larvae, pupae, and adults. For our study, we stipulated that populations began with 10 eggs (the initial starting vector), to simulate field conditions after early colonization by each species. This model, commonly used to compare population growth rates among different species or management scenarios (Caswell 2001), assumes exponential population growth and does not consider density dependence, carrying capacity, immigration, or emigration. Furthermore, the model assumes no host or egg limitation for the parasitoid species. The time step for the model was 1 d.

In a previous study, we evaluated the same three species evaluated in this study as well as other species with a similar modeling exercise (Stark et al. 2004b). However, the difference between the study by Stark et al. (2004b) and the current study is that in Stark et al. (2004b), the goal was to show that the use of surrogate species to predict effects of stressors on endangered species could be problematic if the surrogate and endangered species did have very similar life history strategies. In the current study we focused on the levels of effect developed by the IOBC and their potential to protect biological controls from pesticides. Here, we modified the Delay Index such that the endpoint of interest was a delay of 1-generation time interval. Thus, we ran our model with control populations and then imposed various levels of either mortality, reductions in the number of offspring, and a combination of both effects and looked for levels of stress that caused a delay (in days) of one- or more generation time intervals. We conservatively stipulate that if a population of biological control agents is delayed for 1-generation time interval, it will be difficult for this species to be an effective control agent. This is particularly true for species that are univoltine or have long life cycles. For example, in the current model, a delay of 1-generation time interval for the coccinellid (*C. septempunctata*) would mean its aphid prey [*Acyrthosiphon pisum* (Harris)] would be able to complete 7 generations on peas (*Pisum* spp.) before the ladybird beetle recovered (Stark et al. 2004b). This is based on a 14-d versus a 50-d generation time interval for *A. pisum* and *C. septempunctata*, respectively. Allowing 100 d for *C. septempunctata* to recover would give *A. pisum* enough time to complete 7 generations. However, we assume here that *A. pisum* populations are not affected by the pesticide.

The model was run until at least 100,000 individuals had been reached in all of the treatments listed in Table 2. This number of individuals was reached within 120 d (120 time steps) for all treatments. Populations were considered extinct if population size was <1% of the control after 1-generation time interval for each species. Models were run such that each species was subjected to the levels of stress listed in Table 2. We chose these levels of mortality and reductions in offspring to reflect the high end of the IOBC laboratory categories 1–3 (30, 79, and 98%). Additionally, we evaluated 50% mortality and 50% reduction of offspring to reflect the high end of effect for the semifield and field study categories developed by the IOBC (Hassan 1998). We also ran iterations of each model to determine the actual amount of mortality, reduction in offspring, and a combination of both effects that resulted in a delay of 1-generation time interval (in days).

Table 2. Levels of stress used in the population models

% mortality	% reduction offspring
0	0
30	0
50	0
79	0
98	0
0	30
0	50
0	79
0	98
30	30
50	50
79	79
98	98

Levels of mortality and reductions in offspring reflect the high end of the IOBC laboratory categories 1–3 (30, 79, and 98%).

Results

C. septempunctata was able to withstand high levels of mortality and reductions in offspring before the population was delayed by ≥1-generation time interval (Table 3). Either mortality or reductions of offspring of ≥50% resulted in population delays <1-generation time interval for *C. septempunctata* (Table 3), whereas combinations of 50% mortality and 50% reduction in offspring caused a delay in population growth >1-generation time interval (Table 3). Mortality and reductions of offspring ≥98% or a combination of both effects ≥79% resulted in population extinction for this species (Table 3).

The parasitoid *F. arisanus* had a similar response to the levels of mortality and reductions in offspring as did *C. septempunctata* (Table 4). However, *F. arisanus* was less robust than *C. septempunctata*, with a higher delay as a proportion of generation time after being subjected to 50% mortality (Table 4). The parasitoid *D. rapae* was overall more susceptible than the other two species. Mortality of 50% resulted in a delay of exactly 1-generation time interval (Table 5). Furthermore, delays as a proportion of generation time were higher for all levels of effect for *D. rapae* compared with the other two species.

Simulations run to determine the threshold levels of mortality, reductions in offspring, and a combination of both effects needed to cause delay of 1-generation time interval further illustrated differences in robustness. (Table 6). The parasitoid *D. rapae* was clearly the most susceptible species, followed by *F. arisanus* and *C. septempunctata.* Overall, the levels of mortality causing a 1-generation time interval delay ranged from 50 to 64%, levels of reductions in offspring ranged from

Table 3. Delay of *C. septempunctata* populations after exposure to various levels of stress and the delay as a proportion of generation time (50 d for *C. septempunctata*)

Effect	Delay (d)	Delay/generation time
30% OS[a]	10	0.20
30% M[b]	14	0.28
50% OS	23	0.46
30%–30%[c]	28	0.56
50% M	31	0.62[d]
50%–50%	67	1.34
79% OS	69	1.38
79% M	92	1.84
98% M	Extinct[e]	
98% OS	Extinct	
79%–79%	Extinct	
98%–98%	Extinct	

[a] OS, percentage of reduction in offspring caused by exposure to a pesticide.
[b] M, percentage of mortality caused by exposure to a pesticide.
[c] Combination of percentage of mortality and percentage of reduction in offspring.
[d] Stress levels below this line all caused delays equal to or >1-generation time interval.
[e] Extinction is defined as reduction in population number that is <1% of the control population after 1-generation time interval (days).

Table 4. Delay of *F. arisanus* populations after exposure to various levels of stress and the delay as a proportion of generation time (27 d for *F. arisanus*)

Effect	Delay (d)	Delay/generation time
30% OS[a]	5	0.19
30% M[b]	7	0.26
50% M	23	0.85
50% OS	17	0.63
30%–30%[c]	22	0.81[d]
50%–50%	48	1.78
79% OS	49	1.81
79% M	60	2.22
98% M	Extinct[e]	
98% OS	Extinct	
79%–79%	Extinct	
98%–98%	Extinct	

[a] OS, percentage of reduction in offspring caused by exposure to a pesticide.
[b] M, percentage of mortality caused by exposure to a pesticide.
[c] Combination of percentage of mortality and percentage of reduction in offspring.
[d] Stress levels below this line all caused delays equal to or >1-generation time interval.
[e] Extinction is defined as reduction in population number that is <1% of the control population after 1-generation time interval (days).

Table 5. Delay of *D. rapae* populations after exposure to various levels of stress and the delay as a proportion of generation time (15 d for *D. rapae*)

Effect	Delay (d)	Delay/generation time
30% OS[a]	8	0.53
30% M[b]	9	0.60
50% OS	12	0.80
30%–30%[c]	14	0.93[d]
50% M	15	1.00
50%–50%	38	2.53
79% OS	40	2.67
79% M	52	3.47
98% M	Extinct[e]	
98% OS	Extinct	
79%–79%	Extinct	
98%–98%	Extinct	

[a] OS, percentage of reduction in offspring caused by exposure to a pesticide.
[b] M, percentage of mortality caused by exposure to a pesticide.
[c] Combination of percentage of mortality and percentage of reduction in offspring.
[d] Stress levels below this line all caused delays equal to or >1-generation time interval.
[e] Extinction is defined as reduction in population number that is <1% of the control population after 1-generation time interval (days).

Table 6. Minimum effect (percentage) causing a delay of 1-generation time interval for each species evaluated

Species	% mortality	% reduction offspring	Combination (% mortality and % offspring reduction)
C. septempunctata	64	72	43–43
F. arisanus	58	64	37–37
D. rapae	50	58	32–32

58 to 72%, and the combination of both effects ranged from 32 to 43% (Table 6).

Discussion

The results presented in Table 6 show that *D. rapae* was the most susceptible species, followed by *F. arisanus*, whereas *C. septempunctata* was the least susceptible species based on a delay of 1-generation time interval. Our results indicate that each species reacted differently to the same levels of stress, a phenomenon that we demonstrated previously in a different context (Stark et al. 2004b). Therefore, differences in life history variables make it impossible to generalize about potential long-term impacts of pesticides on biocontrol agents across multiple species. With respect to biological control, our results show that levels of mortality as low as 50% can delay a population of *D. rapae* for 1-generation time interval; therefore, levels of mortality of 50% or higher should be cause for concern for some biocontrol agents, which is in line with the recommendations of the IOBC. Our results suggest that levels of diminished biological control can occur when mortality or reductions in offspring approach 50%. However, our data have only been developed for a limited number of species; therefore, more work needs to be done to elucidate the relationship between levels of mortality and long-term population viability. More important is that multiple effects (lethal and sublethal) below 50% were found to have a negative impact on the three species we evaluated in this study.

The results of our simple experiment here highlight the need to improve articulation between basic toxicity test results and long-term population outcomes. Tillman and Mulrooney (2000) found that two laboratory toxicity tests (topical and residual) for several biocontrol agents and insecticides did not accurately predict results in the field. Armenta et al. (2003) evaluated the effects of several insecticides on insect natural enemies inhabiting maize, *Zea mays* L., and compared their results to the IOBC (Koppert 2002) and SELECTV (Theiling and Croft 1988) data bases. Results of their study indicated that carbamate, pyrethroid, and organophosphate insecticides all resulted in reduced abundance of insect natural enemies, but this reduction was short lived with recovery occurring within 8–15 d. The IOBC and SELECTV databases predicted 75–90% mortality on natural enemies but they could not predict the rapid recovery observed in the field, which was probably due to immigration.

More recently, Thomson and Hoffmann (2006) conducted a 4-mo field study where they measured communities of natural enemies in commercial vineyards in Australia and correlated canopy- and ground-dwelling arthropod data to IOBC laboratory toxicity categories for the pesticides applied to these systems. Their results indicated that IOBC categories based on laboratory toxicity studies were correlated with chemical effects on field populations of natural enemies in vineyards even where complexes of pesticides were used and multiple applications were applied (Thomson and Hoffmann 2006). Of the 26 pesticides applied in their study, the majority (17) fell within the IOBC category 1 (harmless), whereas four pesticides were rated category 2 (slightly harmful), two were ranked as category 3 (moderately harmful), and 3 were ranked as category 4 (harmful). Their overall conclusion was that IOBC toxicity rankings seem to be useful in making choices about the toxicity of various pesticides to natural enemies. Furthermore, Thomson and Hoffmann (2006) found that all groups of invertebrates were not equally susceptible to the pesticides applied in their study. This finding may corroborate our results where we found that species susceptibility may be influenced by life history strategies. However, differential susceptibility to chemicals based on differences in physiology also could account for these findings.

A more subtle result stemming from our simulation model is that combinations of mortality and sublethal effects lower than 50% each were detrimental to all three species evaluated in this study. This highlights the need for further exploration of combinations of lethal and sublethal effects on nontarget organisms in IPM studies. Because we used a modeling approach with life table data for the species of interest, the results presented here represent a simplified version of what might happen in the field. A field situation would likely be much more complicated because of natural mortality factors and species interactions that are not considered here. If the pest (prey or host) is reduced by an amount equal to a natural enemy, we would expect our model results to be an overly optimistic assessment of the fate of biological control agents when subjected to pesticide levels deemed "acceptable" by the IOBC standards.

Together, our results indicate that more ecologically relevant measures of effect such as delays in population growth that are based on demography may advance our knowledge of pesticide impacts on populations of beneficial species.

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