Pesticide Effects on Plant Physiology: Integration into a Pest Management Program


ABSTRACT  Consideration of subtle, nonvisual pesticide effects on crop physiology and yield has generally been ignored by entomologists responsible for developing pesticide recommendations. The literature since 1979 on nontarget pesticide effects was reviewed with respect to plant physiology. Suggestions concerning integration of these effects into present pest management programs are made.

Use of synthetic organic pesticides in modern agriculture has allowed the use of crop varieties that have a higher yield per hectare and higher susceptibility to damage caused by arthropods, weeds, and plant diseases (Pimentel et al. 1978, McEwen 1978). During the so-called “era of optimism” (1946–1962) (Metcalf 1980), the detrimental aspects of pesticide use were either ignored or given little attention by many in the entomological community. However, the increasing frequency of insecticide resistance, pest resurgence (of both primary and secondary pests), and a more general awareness of environmental problems led to the “era of doubt” (1962–1976) (Metcalf 1980). This era was characterized by a more skeptical attitude towards insecticides and their use in agro-ecosystems. The “era of integrated pest management” (1976–present) (Metcalf 1980) has brought insecticide use into a more reasonable perspective, where the insecticide is one of a variety of possible control measures and positive and negative aspects of applying a particular pesticide are considered for possible side effects before application.

Numerous studies have documented the ecological implications associated with pesticide application to insect pest populations. These have concentrated on 1) destruction of natural enemy complexes that can lead to secondary pest outbreaks as well as an increased dependence on insecticide use for control of pest populations (McMurtry et al. 1970, Doutt and Smith 1971); 2) genetic selection that leads to pesticide resistance (Georgiou 1972, Brown 1971); and 3) sublethal effects on pest populations that increase their reproductive capacity or decrease reproduction in predator populations (Huffaker et al. 1970, Maggi and Leigh 1983, Jones and Parrella 1984). These problems have resulted in the establishment of pesticide use recommendations based not only on pesticide efficacy against the pest, but also with regards to natural enemy conservation (Huffaker et al. 1970, Hoyt and Caltagirone 1971, Croft and Hoyt 1978, Huffaker and Smith 1980, McMurtry 1982, Jones and Parrella 1983).

Despite increased awareness of pesticide-induced problems, some entomologists have remained generally unaware or have ignored subtle, nonvisual pesticide-induced changes in plant physiology and concomitant changes in plant yields. A large number of studies concerned with visible phytotoxicity of compounds (par-
particularly on ornamental plants) have been published in the Entomological Society of America’s publication, *Insecticide and Acaricide Tests*. However, only six studies concerned with the more subtle effects of pesticides on crop physiology appeared in *Environmental Entomology* and *Journal of Economic Entomology* from January 1980 to December 1984.

The purpose of this paper is to present work published since the review of Ferree (1979) (with the inclusion of two comprehensive papers published before 1979) and to indicate how pesticide-induced changes in plant physiology can be incorporated into the economic threshold concept of Stern et al. (1959) and van den Bosch and Stern (1962).

**Pesticide Effects on Plant Physiology**

Many insecticides/acaricides may be described as general biocides, with the effects on nonarthropod organisms dependent on the concentration of the compound in question. Before development for marketing, insecticidal compounds are routinely screened for biological activity against many organisms including weeds, fungi, and nematodes. These compounds may show herbicidal action at one concentration and insecticidal action at another concentration. Some compounds may be excellent insecticides, but poor herbicides and are, thus, marketed only as insecticides.

The mode of action of most pesticides that affect plant physiology was broadly characterized by Boger (1979) as “acting quite specifically on certain components of the photosynthetic redox chain” and more specifically by Murthy (1983) as “either inhibitors of photosynthetic electron flow at the site of the primary quencher (Q) of photosystem II and before the reduction of plastoquinone or inhibitors of electron flow after the functional site of plastoquinone, inhibiting the reoxidation of the plastoquinol pool.”

While orientation of most entomologists to pesticide-induced reduction in crop yields does not require knowledge of the exact site of action, information on the physiological aspects can be obtained from Anonymous (1979) and the review of Murthy (1983) and the references contained therein.

Various types of selective activity caused by photosynthesis-inhibiting herbicides were characterized by van Oorschot (1979). He indicated that cuticle structure and composition, size, and distribution of stomata, and additives to the spray solution may affect herbicide penetration into the leaf. This means that the formulation or adjuvants may be responsible in some cases for the noted effect on plant physiology. Differences in transpiration rate may also be important because they may affect absorption and translocation of the herbicide throughout the plant.

To quantify a pesticide’s effect on plant physiology and because of the difficulties in chemical determination of herbicide (or other pesticide) metabolism, van Oorschot (1979) stated “... in the case of inhibitors of photosynthesis, inactivation in the leaves can be readily established with equipment suitable for continuous measurement of photosynthesis. Usually a distinct inhibition of photosynthesis indicates a toxic amount of herbicide has entered the leaf. When uptake is stopped, subsequent recovery from inhibitors is a measure of herbicidal inactivation (from plant metabolism) in the leaves provided the contribution of new leaf growth is negligible during a test period of sufficiently short duration ...” These methods were most commonly used in the studies reviewed in following sections, where either some form of infrared gas analyzer or quantification of the uptake of radio-labeled isotopes was used to quantify photosynthesis rates.

**Orchard Crops**

**Apples.** The most comprehensive study of pesticides on a single crop was performed by Ayers and Barden (1975) on 1-year-old apple seedlings, *Malus pumila* Mill., in a greenhouse environment. Only 12 of the 33 pesticides studied (which included insecticides, acaricides, and fungicides) induced changes in photosynthetic rate. Four increased photosynthetic rate and the other eight caused a reduction. It is interesting to note that of the 12 pesticides that changed the photosynthetic rate (maximum change indicated in brackets) (carbaryl 50WP [+16%], chlorprophamate 2EC [-56%], maneb 80WP [+7%], dithian 80W [-12%], karathane 25W [-10%], formetanate hydrochloride 92SP [+17%], parathion 8EC [-11%], superior oil [70s] [-73%], phosalone 3EC [-47%], cyhexatin 50W [+16%], oxathiinoxacin 25W [-8%], and sulfur 95W [-6%]), all are insecticides or acaricide/fungicide combinations except maneb. Photosynthetic rate determinations were made at 0, 1, 6, and 11 days posttreatment, thereby quantifying the rate of recovery from pesticide-induced changes. With most insecticides, the peak reductions were found 6 days posttreatment, with a trend towards recovery of normal photosynthetic activity occurring 11 days posttreatment.

Sharma et al. (1978) also tested several pesticides on 1-year-old apple seedlings, *M. domestica* Borkh. They found that of the 37 pesticides tested, 5 increased photosynthetic rate and 20 decreased photosynthetic rate by >5% 28 h following application. The insecticide/acaricide diazinon 50WP, dicyclo 42EC, and superior oil (70 s) caused a respective 26.6, 23.6, and 66.3% reduction in photosynthetic rate while leptophos 30.6EC and propargite 30WP caused 13.3 and 15% reduction, respectively.

**Pecan.** The importance of pesticide formulations was demonstrated by Wood and Payne (1984) working on ‘Currit’ pecan seedlings *Carya illinoensis* (Wang.) K. Koch. They investigated the effect of monocrotophos 5EC, methomyl 1.8EC and 90SP, phosalone 3EC and 25WP, carbaryl 50WP, azinphosmethyl 2EC, fenvalerate 2.4EC, petroleum oil (70 s), dimethoate 25WP and 2.67EC, and a combination of fenvalerate and petroleum oil on the photosynthetic rate in three separate experiments. In all three experiments, the photosynthetic rate observed 1 day after treatment of treated plants was significantly lower (the reduction ranged from 15 to 30% depending on the treatment and experiment) than the untreated controls. In the first experiment at 9 days postapplication, the photosynthetic rate of the plants treated with the wettable powder formulations of methomyl and phosalone recovered to control levels, while the respective rates of emulsifiable concentrate formulations were still ca. 20% below that of the untreated control. In the second experiment at 9 days postapplication, the carbaryl (WP) and fenvalerate (EC) treatments were 20% below the control rates and petroleum oil was 40% below that of the controls, but the plants treated with azinphosmethyl (EC) had recovered. The third experiment demonstrated that plants treated with the WP formulation of dimethoate recovered by 7 days postspray, while those treated with the dimethoate (EC), fenvalerate (EC), and the fenvalerate-oil combination required another 7 days to recover to the control levels.

Wood et al. (1984) investigated the effect of a single application of fungicides on ‘Currit’ pecan seedlings. The fungicides propiconazole 3.6EC, etaconazol 1.125EC, benzophenyl 50WP, triphenyltin hydroxide 30WP and 4L, fenamiphos 1EC, chlorothalonil 50FL, and dodine 65WP were evaluated 1 and 9 days following application. One day following applica-
tion, all treatments except propiconazole and etaconazole reduced leaf photosynthesis rates significantly, with the average reduction ranging from 25 to 35%. The benomyl-treated plants recovered to control levels by 9 days, but the triphenyltin (both formulations), fenarimol, chlorothalonil, and dodine treatments were still 24-40% below the untreated control levels at that time. Based on the recovery time observed by Wood and Payne (1984) working with insects and the results of their study on fungicides, Wood et al. (1984) concluded that pecan physiology was more sensitive to fungicide application than to insecticide application.

Wood and Payne (1984) and Wood et al. (1984) also pointed out the possible effect of pesticides on tree crops the year following pesticide application. This was hypothesized to occur because the reduction in net photosynthesis can affect the amount of stored carbohydrates. This type of delay in expression of spider mite feeding damage has been shown to occur on walnuts (Barnes and Moffitt 1978) and almond (Barnes and Andrews 1978, Wellet al. 1984).

Citrus. Jones et al. (1983) compared the effects of propargite 30WP, dicofol 1.6E, and NR-440 oil on the photosynthetic rate of 15-year-old ‘Lisbon’ lemons, Citrus limon (L.) Burmann, and fenbutatin-oxide 50WP, dicofol 1.6E, oxathioquinox 25W, and NR-440 on the photosynthetic rate of 17-year-old ‘Valencia’ oranges, C. sinensis (L.) Osbeck. They found that the lemons were rather insensitive to pesticide-induced changes in photosynthetic rate, but the propargite treatment showed an increase of 26.8 and 26.6% in mesophyll conductance (which is a measure of internal CO2 transport) and photosynthetic rate, respectively, 12 days postapplication.

‘Valencia’ oranges were much more sensitive to acaricide application. Three days following pesticide application, the fenbutatin-oxide treatment showed a 12 and 9% increase in stomatal conductance (which is an indicator of stomatal opening) and photosynthetic rate, respectively, and was significantly higher than the other compounds tested. Leaves treated with oxathioquinox and dicofol were slightly lower in stomatal conductance (16.7 and 22.5%, respectively) and photosynthetic rate (12.3 and 21.8%, respectively). The trees in the NR-440 oil treatment showed a 31.8% drop in photosynthetic rate and were significantly lower than the water control.

The 11-day postspay samples showed that orange leaves treated with fenbutatin-oxide and dicofol exhibited a significantly higher photosynthetic rate compared with the other treatments. Oxythioquinox and NR-440 caused a 21% reduction in photosynthetic rate compared with the untreated trees. However, based on the differential rate of recovery, NR-440 may be a better choice of pesticide for control of a given mite species given equal activity against the pest species (Jones et al. 1983).

Ornamental Crops

Chrysanthemum. Baun and Peterson (1981) investigated the effects of aldicarb, benomyl, captan, dichlorvos, dicofol, dienochlor, mancozeb, and piperazine (formulations not available) on potted, pinched ‘Bright Golden Anne’ chrysanthemums, Chrysanthemum morifolium Ramat, at various growth stages. Interaction of plant phenology and number of applications influenced height, fresh weight, and dry weight of plant tops but only time of application influenced number of breaks (= vegetative shoots). Developmental rate and number of flowers were also influenced by pesticides. In addition, single or multiple applications of the above pesticides changed the photosynthetic rates compared with the untreated control.

Jones et al. (1984), working with potted ‘Tip’ chrysanthemums, found that after five applications, plants treated with cyromazine 75W or permethrin 3.2E showed a significant increase in photosynthetic rate compared with plants treated with aldicarb 10G or a combination of permethrin and micro-encapsulated methyl parathion 2M. However, at shipping time they did not observe differences in mean flower head weight, mean flower head diameter, mean plant height, mean dry weight of the above ground foliage + flowers, mean number of open flowers, or mean number of flowers + buds between treatments. They attributed the disparity of results between photosynthetic measurements and yield measurements to application of the growth regulator B-9 (sucinom acid 2,2 dimethyl hydrazide).

Vegetables

Lettuce. The most intensive field investigations of pesticide effects on a single crop were performed on lettuce, Lactuca sativa L., in California by Toscano et al. (1982a,b) and Johnson et al. (1983). Toscano et al. (1982b) did not observe significant differences in pesticide effects on plant physiology when pesticides were applied at noon (stomata open) and midnight (stomata closed). They therefore, concluded that penetration through the leaf cuticle was the mode of pesticide uptake. In addition, they found that the photosynthetic rate of plants treated with methoxychlor (50WP) did not exhibit significant differences in either stomatal conductance or photosynthetic rate compared with untreated plants. However, plants treated with methomyl (50WP) exhibited a 5% reduction in stomatal conductance and an 8% reduction in photosynthetic rate 1 day following treatment; after 1 week, a 20% reduction in photosynthetic rate was recorded. Methyl parathion (4EC) induced a 17% reduction in stomatal conductance and a 10% reduction in photosynthetic rate in the 1-day samples, but this increased to a respective 27 and 18% reduction 1 week later. Permethrin (2EC) exhibited a 14% initial reduction in photosynthetic rate, which increased to 18% 1 week later.

In additional studies, yield measurements were taken from plots where the various treatments mentioned above were applied during the period from germination to thinning (ca. weeks 1–3) and from formation to harvest (ca. weeks 8–11) in contrast to plots treated throughout the entire season. Treatments were applied either once or twice a week in each case. In general, lettuce treated weekly throughout the whole season produced lower yields than lettuce not treated during the growth period from thinning to harvest (ca. weeks 4–8). The methyl parathion treatments caused significant reductions in mean head weight (24% reduction for the weekly spray and 30% reduction for the plots treated twice.
head density, and percent of bolted plants were linearly correlated with the number of methyl parathion treatments. Pesticide-induced reductions in total yield were attributed to reduced head weight and increased bolting of the plant. Only an increase in the percentage of bolted plants was associated with the use of methomyl. A change in application rate produced a curvilinear relationship in photosynthetic rate, mesophyll conductance, and stomatal conductance. Based on these results, Johnson et al. (1983) recommended a “pesticide threshold” of no more than three applications of methyl parathion to avoid a yield reduction of >20%.

**Strawberry.** The effects of acaricides on growth, yield, and physiology of *Fragaria chiloensis* (L.) was investigated by LaPre et al. (1982). Plants were sprayed with cyhexatin (50WP, every 2 weeks; total of six sprays), fenbutatin-oxide (50WP, monthly; total of three sprays at 1- and 2-fold rates), formetanate hydrochloride (92SP, monthly; three sprays), and propargite (30WP, every 2 weeks; six sprays or monthly; two sprays). Early in the season, the propargite and formetanate hydrochloride treatments caused reductions in stomatal conductance, mesophyll conductance, and photosynthetic rate. Reductions in photosynthetic rates were 7 and 21% for the respective 1- and 2-fold rates of formetanate hydrochloride and 20 and 24% for the respective biweekly and weekly propargite treatments. Plots treated with fenbutatin-oxide and cyhexatin showed no physiological differences from the control plots.

Late in the season, it was found that only formetanate hydrochloride caused reductions in photosynthetic rates because of its lasting effect on mesophyll conductance. Total fruit yield and number of fruit were significantly higher in the fenbutatin-oxide treatments compared with the high formetanate hydrochloride and both propargite treatments.

**Tomato.** In a 2-year study on the effectiveness of systemic insecticides for controlling Colorado potato beetle on tomato ('Petro Meeh'), Romanow et al. (1984) found that the insecticides disulfofon, phorate, and thiofaneox delayed and reduced seedling emergence at all rates used. At the higher rates, carbofuran and aldicarb delayed seedling emergence but did not reduce the crop stand. The carbofuran treatment resulted in a significant increase in the number of flowering plants 41 days following planting.

The second year of the study, ‘UC-82B’ was planted. Again, plug mixes (seedlings are planted with a “plug” of soil) of disulfoton, phorate, and thiofaneox reduced seedling emergence compared with the untreated control. However, this same effect was not observed in banded treatments of the same pesticides at the same dosages. A single plug application of aldicarb and two banded-and-incorporated applications of aldicarb significantly accelerated seedling emergence. Aldicarb was also found to increase percentage of flowering plants 23 and 48 days after planting regardless of application method. Carbofuran had a similar effect in the lower plug-mix dosages in the April planting and at all dosages in the May planting.

**Field Crops**

**Cotton.** In experiments attempting to establish DTL’s for bollworms on cotton in South Africa, van Hamburg and Kifir (1982) found that weekly sprays of cypermethrin caused significant reductions in yields compared with plots where a DTL of one larva per plant was maintained. They concluded that the pesticide effect was important enough to preclude treatment on a prophylactic basis. Their graph of yield versus larva density is similar to that obtained by Toscano et al. (1982b) on lettuce (Fig. 1).

**Incorporation into Pest Management Programs**

The economic threshold concept is described by Stern et al. (1959) and van den Bosch and Stern (1962). Briefly, pest suppression activities are initiated on the basis of the relationship between pest population density and sufficient reductions in plant quality to result in economic damage (economic injury level). The point at which suppression activities should be initiated to prevent the increasing population from reaching the economic injury level (EIL) is called the economic threshold (ET) (Fig. 2). Although some entomologists tend to use the idea of “pest-days” (Hussey and Parr 1963, Allen 1976, Hoyt et al. 1979, Sances et al. 1979, Ruppel 1983, Welte et al. 1984) to quantify plant damage and reduction in photosynthetic rates instead of population density, the basic concepts of Stern et al. (1959) are still valuable. Only slight modifications are necessary to incorporate pest days and changes in the definition of EIL and ET.

Classically, EIL is dependent on the stage of crop development, season, projected value of the crop, and cost of the
management system (for monitoring pest populations, pesticide, and application). ET is then normally considered to be a fixed distance below the EIL with the difference between them related to the lag time between recognition of the problem and when control actions reduce population levels (Stern et al. 1959).

When considering the effect of pesticides on plant physiology and yield, the relationship between ET and EIL becomes more complex. EIL is redefined slightly and becomes the total amount of physiological damage that a plant can withstand and not cause economic loss. It is a function of both pest and pesticide damage, crop phenology, cost of the management system, and projected economic value of the crop. The ET, therefore, becomes a function of pest days, pesticide effects on plant physiology, and the lag time between recognition of the problem and when control actions reduce population levels. Because pesticides may have either a positive or negative effect on plant physiology, the redefined ET can be either $>$(if stimulation occurs), $=$ (if no effect), or $<$ (if reduction occurs) the classical ET.

Because the effects of insecticides that change photosynthetic rates seem to be characterized by an initial change in photosynthetic rate and a later recovery period related to the weathering and detoxification of the residue (van Oorschot 1979), the pesticide effect on plant physiology cannot be considered to be a linear relationship but instead appears to be curvilinear (Fig. 3). To quantify the effect of a pesticide on plant physiology, the recovery rate of the plant to the particular pesticide (or combination of pesticides) in question must be known.

Once an estimate of pesticide effects has been obtained, the following simple model can be used to estimate the combined effect of pest and pesticide on yield:

$$ \begin{align*} 
Y_n &= Y_m - P_t - I_t 
\end{align*} $$

(1)

where $Y_n$ = net yield, $Y_m$ = maximum yield under the given cultural practices (for this example yield has been used, but either percentage of reduction in yield or reduction in yield could be used), $P_t$ = total pest damage, and $I_t$ = total pesticide damage. Mathematically this can be expressed as a multiple linear regression equation:

$$ \begin{align*} 
Y_n &= P_a(P_n + I_p(N)) 
\end{align*} $$

(2)

where $P_a$ = accumulated pest days, $P_n$ = pest damage coefficient, $I_p$ = the pesticide impact coefficient, $N$ = the number of pesticide applications, $P_a \times P_n = P_t$, and $I_t = I_p \times N$. $P_a$ is generally $<0$ (some studies have shown that a low level of damage can stimulate plant growth [see Sanches et al. 1981]); and $I_p$ can take on values $>1$ or $<0$ depending on the pesticide effect on plant physiology (Fig. 4). The treatment decision can be guided by consideration of the projected value of the commodity, and whether the pest effect will reduce final dollar return more than the costs associated with the pesticide and its effect on plant physiology.

Examination of equations 1 and 2 reveals four cases of interest to entomologists (in the following hypothetical examples, $Y_m = 1,000$ kg/ha, ET = the damage that would reduce the yield to 800 kg/ha, EIL = the damage that would reduce yield to 700 kg/ha, $P_t = 0.5$ kg per ha per pest-day, and $I_p = -50$ kg per ha per application; therefore, yield decreases 5% of $Y_m$ with each pesticide application or every 100 pest days). The first case occurs when a highly "aesthetic" value crop such as cut flowers is considered. Since virtually no pest damage can be tolerated, pesticide applications are prophylactic and (assuming 100% efficacy of the pesticide) pest days do not accumulate. Therefore, equation 2 simplifies to:

$$ \begin{align*} 
Y_n &= Y_m - I_p(N) 
\end{align*} $$

(3)

or

$$ \begin{align*} 
Y_n &= 1,000 - 50(N) 
\end{align*} $$

(4)

Fig. 4. Hypothetical relationship between yield and number of pesticide applications if pesticide impact coefficient is $>1$, $=1$, or $<1$.

and yield changes are due entirely to the number of pesticide applications (e.g., a "pesticide threshold"). Consequently, the maximum number of applications to reach the ET is four and economic damage occurs at six applications (Fig. 5, Case 1). Using equation 3, the data of Johnson et al. (1983) indicate that the $I_p$ for methyl parathion and methomyl on lettuce is $-1.2$ and $-0.13$, respectively.

The second case is similar to the first, except that the classic ET has already been reached and immigration of pests from neighboring fields keeps pest population pressure high. Again, no more pest damage can be tolerated and equation 2 becomes:

$$ \begin{align*} 
Y_n &= Y_m - (Y_m - ET) - I_p(N) 
\end{align*} $$

(5)

or

$$ \begin{align*} 
Y_n &= 800 - 50(N) 
\end{align*} $$

(6)

and yield changes are again primarily dependent upon the number of pesticide applications. The maximum number of

Fig. 5. Hypothetical relationship between yield (in kg/ha) and the number of pesticide applications. Case 1, 'aesthetic' value crop where no insect damage can be tolerated. Case 2, the economic injury level is reached before spraying begins and pest pressure remains high from surrounding areas.
Fig. 6. Hypothetical relationship between yield (in kg/ha) and pest-days when a single application of pesticide will control the pest for the entire season.

Applications possible without exceeding the EIL is two, demonstrating an instance where adherence to the classic ET will result in economic loss if pesticide effects are not considered (Fig. 5, Case 2).

The third situation occurs frequently with indirect pests that can be easily controlled for the entire season with only one pesticide application. In this case, equation 2 becomes:

\[ Y_n = Y_m - 0.5(P_n) - I_p(1) \]  

(7)

or

\[ Y_n = 950 - 0.5(P_n) \]  

(8)

and only 300 pest-days are allowed before the ET is reached and 500 before the EIL is reached (Fig. 6).

The last case occurs when the pest population develops resistance to the compound used for control. Once the economic threshold is reached, equation 2 becomes:

\[ Y_n = Y_m - (Y_m - ET) - P_d(P_n) - I_p(N) \]  

(9)

or

\[ Y_n = 800 - 0.5(P_n) - 50(N) \]  

(10)

and plant yield is decreased because of the increased number of sprays necessary to achieve control and the continued accumulation of pest-days (Fig. 7). This situation is also important in considering two compounds that have different efficacy. If the least efficacious material has 1/2 the effect on yield of the most efficacious material, the least efficacious material would be considered for use if two or less sprays would be used to achieve control.

**Discussion**

In actual practice, pesticide and pest damage are probably not linearly related to yield reductions when the entire range of possible values are considered. In addition, use of binary mixtures, different application rates and methods, frequency of application, crop phenology, and combinations of pests (with their corresponding different types of damage) all undoubtedly affect yield possibly a nonlinear or nonadditive manner. However, for the purpose of demonstrating how pesticide effects can be included into an economic decision model, the linear fit is sufficiently accurate. For simplicity’s sake, we have chosen to use the broadly recognized model of Stern et al. (1959), but the same inclusion of pesticide effects on plant yield could be done with any of the current economic decision models discussed by Mumford and Norton (1984). When used in conjunction with knowledge concerning the effect of pesticides on natural enemies, this concept provides a framework that can be used to aid the construction of pest management systems. The basis of this framework is a broader perspective on a control tactic that affects all three trophic levels involved, instead of the one or two levels currently recognized.

Ferreira (1979) concluded that insecticides should be routinely screened for their effect on plant physiology before use recommendations of that particular pesticide are made. Jones et al. (1983) agreed, and in addition, stated that an understanding of the effects of pesticide-induced reductions on growth and yield of plants is crucial to the development of pest management systems. Toscano et al. (1982a, b) and Johnson et al. (1983) favored the establishment of pesticide thresholds for insecticides shown to reduce crop yields. We reiterate these suggestions and, in addition, feel that data concerning pesticide-induced changes in yield of indicator crops should be required for pesticide registration. In the laboratory, future research in the areas of quantitative structure activity relationships, and optimizing pesticide formulation may yield pesticides that have a maximal effect on the pest species and a minimal effect on plant physiology. Field experiments that utilize portable photo-synthesis-measuring instruments may also be used to help elucidate the effects of pesticides on plant physiology and aid in providing more informed pesticide recommendations.

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