Resistant Pest Management

A Biannual Newsletter of the **Pesticide Research Center (PRC)**
in Cooperation with
the **Insecticide Resistance Action Committee (IRAC)**,
the Western Regional Coordinating Committee (WRCC-60)
and the International Organization for Pest Resistance Management (IOPRM)

Vol. 10, No. 1 **Winter 1998**

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Letter from the Editor

Resistance management for transgenic plants?

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Resistance management (RM) entails the amelioration of the evolution of genetic adaptation to pesticide selection. The four basic principles of RM are to diversify mortality mechanisms, to reduce selection pressure, to manage susceptibility, and to monitor and predict resistance development. RM is strategic feature of Integrated Pest Management and supports the ideal of minimizing adverse pesticide effects on society and the environment. RM objectives include decreasing chemical inputs, while increasing the products use life.

Resistance is an evolutionary phenomenon correlated with use intensity. The potential for resistance begins with the first application of a pest population suppression tool, and may increase with each successive use. Insect pests have the potential to become resistant to any number of tactics and tools including genetically engineered transgenic plants. Currently, efforts are underway to understand the resistance potential of this new pest suppression technology.

Since 1989, the RPM newsletter has published 342 articles on resistant pests and Resistance Management (Pat Bills, personal communication 1998). Through this publication scientists are able to communicate with other RM researchers worldwide. The goal of RPM newsletters is to provide for communication, which may lead to RM implementation and refinement of RM tactics.

Bacillus thuringiensis (B.t.), an ubiquitous gram-positive bacteria, has been broadly deployed in conventional spray applications since the 1970's, and is an important key tool for IPM today. B.t. toxins have been used globally to manage numerous pests. Conventional synthetic insecticides such as organophosphates and carbamates were and still are effective in many production systems. However, regulations in the US (Food Quality Protection Act of 1994) and resistance globally may curtail this use in the future. A good example of resistance mitigating the usefulness of organophosphate, carbamate, synthetic pyrethroid and other insecticides has been documented in the Colorado potato beetle (CPB)(Forgash 1985, Ioannids et al. 1991, Heim et al. 1990). Certainly this history and many others demonstrate the need for RM for even new technologies such as transgenic plants.

Conventional pesticides used for pest population suppression in the past 30-40 years are, in some instances, being replaced with biologically based approaches. Transgenic plants capable of expressing B.t. are currently available in many crops. Cotton, corn and potatoes were the first transgenic plant containing B.t. to be commercially released in the U.S. In 1996, 1.5 million acres of transgenic cotton were planted. Scientists and farmers alike remain guardedly enthused with the efficiency of these plants. Within the next two years, transgenic plant use in the US is expected to exceed 15 million acres (Whalon and Norris 1997). These transgenic plants are no longer experimental curiosities, but are part of a new generation of emerging pest suppression tools. Their rapid implementation has created the urgent need for resistance management plans.

With new highly valuable tools such as transgenic plants, proper RM is needed to protect their long-term effectiveness. In order to implement a successful RM plan, proper awareness and education are needed for growers and distributors. In the laboratory, further studies will continue the development of transgenic plants that have built in resistance fighting tactics. However, managing susceptible pest populations in the field to prevent resistant individuals from interbreeding will be the most important RM tactic for some time. Stated differently, transgenic plant RM uses susceptible individuals to mate with resistant individuals to "swamp out" or dilute resistance genes in the next generation.

The Resistance Pest Management newsletter could also serve as a worldwide link in RM communication for scientists, policy makers, and RM workers interested in genetically engineered plants. The goal of this newsletter has been to "foster communication, research, and policy that will result in the amelioration of pesticide resistance problems" (Whalon et al. 1989). In order to retain this goal as pesticide strategies, tactics and tools change, we should expand our scope to include RM of transgenic plants as well as conventional pesticides.

In the U.S., Europe, Australia, New Zealand, etc., efforts have been initiated to further develop RM plans for various transgenic plants (James and Krattiger 1996). National organizations are creating symposia that focus on specific transgenic plant issues. These issues are on a wide range of topics including economics, engineering, implementation, management, and research. Regulatory agencies like the USEPA are promulgating rules that require RM as a condition for registration (USEPA Science Advisory Report 1998).

Information on IPM is exchanged on a worldwide basis through international efforts in organizations such as CABI, FAO, UNDP, CGIAR, CICP, IPM-CRSP, and IPMEurope (Kaeb et al. 1998). Committees and Symposia addressing RM have been developed through these organizations as well. Table 1 summarizes several recent and future conferences that include sections on RM plans for transgenic plants. This situation has provided the RPM newsletter with the opportunity to aid in worldwide communication for the management and development of transgenic RM strate-

gies, tactics and tools. Therefore, we invite readers to submit research reports in this emerging arena.

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Table 1 : Listings of Conferences dealing with Transgenic Plants.

Title of Conference	Location	Date
Commercialization of Transgenic Crops: Risk, Benefit and Trade Considerations (BINAS)[1]	Canberra	March 1997
IBC's International Conference on Transgenic Plants	Washington, DC	February 1998
Agricultural Biotechnology and environment Quality: Gene Escape and Pest Resistance (NABC)[2]	Greenville, South Carolina	May 1998
Council for Agricultural Science and Technology Roundtable on Biotechnology Exports	Washington D.C.	June 1998
World Congress Of Environmental And Resource Economists	Venice, Italy	June 1998
Plant - Derived Therapeutics - Cloning and Transgenics (IBC)[3]	San Francisco, California	September 1998
27th International Symposium "Actual Tasks on Agricultural Engineering (EurAgEng)[4]	Opatija, Croatia	February 1999
Gene Flow and Agriculture - The Relevance of Transgenic Crops (Keele University)	University of Keele, England, UK	April 1999

[1]Biosafety Information Network and Advisory Service.
[2]International Business Communications.
[3]National Agricultural Biotechnology Council
[4]European Society of Agricultural Engineers

News and Reviews

EPA regulation of resistance management for Bt plant-pesticides and conventional pesticides

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The Environmental Protection Agency (EPA) considers the development of pesticide resistance and pesticide resistance management in its regulatory decisions (see reviews Matten et al., 1996 and updated in Matten, 1997). In general, pesticide resistance management is likely to benefit the American public by reducing the total pesticide burden on the environment and by reducing the overall human and environmental exposure to pesticides. Although EPA does not yet have a published policy or standard data requirements in place for pesticide resistance management, the Agency has required the submission of such data on a case-by-case basis. EPA supports the efforts of all stakeholders to promote pesticide resistance management through the development and use of pesticide resistance management plans, appropriate pesticide labeling and education programs. EPA's desire is that pesticide resistance management recommendations/requirements not overly burden the regulated community, jeopardize the registration of reduced risk pesticides, or exclude conventional pesticides or other control practices which can contribute to the further adoption of integrated pest management (IPM). EPA believes that appropriate resistance management can further these goals. EPA is continuing to evaluate and

refine the role pesticide resistance management has in the Agency's regulatory decisions.

Current regulation of Bacillus thuringiensis (Bt) plant-pesticides

With a greater focus on pollution prevention and pesticide risk reduction, the EPA believes that it is important to implement effective resistance management strategies for pesticides such as *Bacillus thuringiensis* (Bt) plant-pesticides. A great deal of Agency attention has focused on the potential development of resistance to the delta-endotoxins of Bt genetically-engineered into plants (Bt plant-pesticides). This is because Bt plant-pesticides produce the pesticidal active ingredient, the Cry delta-endotoxin(s), throughout the growing season. Long-term exposure to a pesticide is one of the factors that increases the potential selection pressure upon both the target pests and any other susceptible insects feeding on the transformed crop. EPA recognizes the value of Bt plant-pesticides as effective and safer pest management tools and has determined it is appropriate to conserve this resource by requiring resistance management plans for certain transformed crops. In addition to Bt delta-endotoxins being used in plant-pesticides, they are also widely used in a variety of Bt microbial spray products on many crops. Therefore, the Agency has requested that all registrants for Bt plant-pesticides voluntarily submit pesticide resistance management strategies because the high benefits of using Bt plant-pesticides could be diminished by the development of resistance to individual Bt plant-pesticides and because of the threat cross-resistance poses to Bt microbial pesticides.

EPA's review of Bt plant-pesticide resistance management strategies

The Agency identified seven elements that should be addressed in a Bt plant-pesticide resistance management plan (Matten and Lewis, 1995). These elements are: (1) knowledge of pest biology and ecology, (2) appropriate dose expression strategy, (3) appropriate refugia (primarily for insecticides), (4) monitoring and reporting of incidents of pesticide resistance development, (5) employment of IPM, (6) communication and educational strategies on use of the product and (7) development of alternative modes of action. These elements were presented to the March 1, 1995 Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) Science Advisory Panel (SAP) Subpanel on Plant-Pesticides. The SAP Subpanel approved of these seven factors (SAP, 1995; see Office of Pesticide Program (OPP) docket, OPP-00401).

All registrants of Bt plant-pesticides voluntarily submitted Bt plant-pesticide insect resistance management strategies to the Agency for Bt delta-endotoxins produced in potato (Bt potato); field corn, sweet corn, and popcorn (Bt corn); and cotton (Bt cotton). When necessary, the Agency made certain recommendations and requirements of registration for the development of data to develop and implement long-term resistance management strategies as part of the registration decisions. The Agency's reviews of the resistance management strategies for registered Bt plant-pesticides are summarized in EPA FACT sheets (EPA 1995a, b, c, 1996 a, b, 1997, 1998 a, b, c).

Following the March 1, 1995, SAP Subpanel meeting, the Agency registered the CryIII delta-endotoxin and the genetic material necessary for its production in potato (Bt potato) in May 1995. No requirements related to resistance management were imposed on the registration of Bt potato based on the Agency's scientific analysis of Monsanto/Naturemark's resistance management strategy and comments received from the SAP Subpanel (SAP, 1995; see Office of Pesticide Program (OPP) docket, OPP-00401). Voluntary interaction between the registrant and EPA was recommended by the SAP and certain areas of research and monitoring were suggested. EPA and Monsanto/Naturemark have worked together on the development and implementation of appropriate long-term resistance management following the registration of Bt potatoes in 1995. Monsanto/Naturemark requires a mandatory refuge through their Grower's Agreement for each of its growers to follow and the overall Bt potato resistance management strategy is being refined as more data become available.

The Agency mandated specific resistance management data requirements and mitigation measures with a resistance management strategy for all of the Bt corn and Bt cotton registrations. Registrations for Bt corn plant-pesticide products expire April 1, 2001 and the registration for Bt cotton plant-pesticide products expire January 1, 2001. These registrations were conditional to allow, in part, for completion of the studies related to resistance management. Collection of various data, e.g., target pest biology and behavior, secondary pest biology and behavior, population dynamics, cross-resistance potential, refuge strategies, dose deployment adequacy, discriminating concentration, monitoring, and reporting made conditions of registration for the Bt corn and Bt cotton registrations. Refuge requirements were mandatory for Bt cotton. Development of draft refuge options by August 1998, a final refuge strategy by January 1999 with implementation by April 1, 2001 were required of Bt corn registrations. As part of the terms and conditions of registration, EPA will reevaluate the effectiveness of each registrant's resistance management plan before the expiration date and decide on whether to convert the registration to a non-expiring registration.

The Agency registered the use of CryIA(b) in sweet corn (Bt sweet corn) and popcorn (Bt popcorn) as amendments to existing registrations in March 1998. Specific monitoring and sales reporting were made requirements of the Bt sweet corn registration. No specific refuge requirements were mandated for Bt sweet corn (Event BT 11) because harvesting occurs before insects mature, approximately 21 days after silking.

Growers are instructed in all labeling and technical material to destroy any CryIA(b) sweet corn silks that remain in the fields following harvest or within a short period of time (a maximum of one month) later in accordance with local production practices. Stalk destruction will reduce the possibility of larvae surviving to the next generation. The Bt sweet corn registration expires April 1, 2001.

The Agency mandated specific refuge requirements on the use of Bt popcorn (Event 176) based on the USDA NC-205 recommendations (Ostlie *et al*., 1997). Specifically, a 25% unsprayed or 40% sprayed non-Bt corn structured refuge in close proximity to Bt corn is required. The refuge must be established within 1500-2000 feet of the Bt corn. Specific monitoring and sales reporting requirements were also made for the Bt popcorn registration. All previous data required for Bt field corn were also required for Bt popcorn. The popcorn registration expires April 1, 2001.

The Agency registered the use of Cry9(c) field corn in May, 1998. This is a one-year registration for 120,000 acres for animal feed, industrial non-food, and seed increase uses expiring on May 30, 1999. EPA mandated specific refuge requirements based on the USDA NC-205 recommendations (Ostlie *et al*., 1997). Specifically, a 25% unsprayed or 40% sprayed non-Bt corn structured refuge in close proximity to Bt corn is required. The refuge must be established within 1500-2000 feet of the Bt corn. Because of the one-year duration of this registration, only sales reporting and grower education are required as part of this registration. Additional resistance management factors must be addressed for a full commercial registration.

All stakeholders are concerned with how EPA regulates resistance management for Bt plant-pesticides. Scientifically-sound long-term resistance management strategies are essential to the survival of Bt plant-pesticides, protection of Bt microbial pesticides, and reduction in the risks from the use of pesticides. EPA is continuing to evaluate and refine how it regulates resistance management of Bt plant-pesticides. EPA has worked and is working with stakeholders (industry, university and USDA extension entomologists, individual growers, user groups, trade organization, public interest groups, and government agencies) to address long-term resistance management for Bt plant-pesticides.

EPA white paper on Bt plant-pesticide resistance management

The Agency published a recent analysis of the current resistance management strategies for Bt potato, Bt field corn, and Bt cotton in a paper entitled "The Environmental Protection Agency's White Paper on Bt Plant-Pesticide Resistance Management" (January 14, 1998) (EPA, 1998d). In this paper, the Agency summarized the findings from the March and May 1997 public hearings on Bt plant-pesticide resistance management (OPP Docket, OPP-00470), the 1996 growing season reports on resistance management activities for Bt potato, Bt field corn, and Bt cotton, the 1997 research efforts for resistance management, published literature, information from public meetings and discussions with academic or extension entomologists on Bt plant-pesticide resistance management (OPP Docket, OPPTS-00231). The EPA White Paper can also be obtained electronically from the EPA Home Page at: Federal Register—Environmental Documents—"Laws and Regulations" (http://www.epa.gov/fedrgstr/). A summary of EPA's White Paper is provided below.

White paper summary

Since Bt plant-pesticides became commercially available in 1996, growers have adopted this technology as part of their IPM practices to control pests in potato, corn, and cotton. Based on industry reports sent to EPA, the greatest adoption of Bt crop technology has been by cotton growers, especially in the southeastern United States in 1996, with about 13% of the cotton acreage, 1.8 million acres, and an estimated 2.2 to 2.4 million acres in 1997 planted in Bt cotton. Corn growers planted about 400,000 acres of Bt corn in 30 states in 1996 and an estimated 4 million acres in 1997. Potato growers planted about 10,000 acres of Bt potato in 1996 and an estimated 25,000 acres in 1997. The differences in the rate of adoption of Bt potato, Bt corn, and Bt cotton are likely due, in part, to the availability of effective alternatives, the cost of the biotechnology crop, extent of regional pest problems, and familiarity and acceptance of the technology by growers. For example, there are several insecticide alternatives for Colorado potato beetle control. The cost and familiarity with the technology and type of hybrids available may have discouraged a wider adoption by corn growers in the first years of commercialization. The adoption rate for Bt cotton was especially high for a new technology because few, if any, effective alternatives existed to control tobacco budworm (*Heliothis virescens* (Fabricius), TBW) in cotton especially where resistance to registered conventional pesticides was extremely high in states such as Mississippi and Alabama.

No evidence exists that resistance to Bt Cry proteins produced in transgenic potato, corn, or cotton has developed in the 1996 or 1997 growing season. Monitoring for susceptibility changes to the different registered Cry proteins, CryI(A)b, CryI(A)c, and CryIIIA, has been conducted for Colorado potato beetle (*Leptinotarsa decemlineata* (Say), CPB), European corn borer (*Orsinia nubilalis* (Hübner), ECB), tobacco budworm (*Heliothis virescens* (Fabr.) TBW), cotton bollworm (*Helicoverpa zea* (Boddie), CBW), and pink bollworm (*Pectinophora gossypiella* (Saunders), PBW). Baseline susceptibility studies show a wide-range of variability, so it is important to look at susceptibility changes in the context of the baseline range for a particular geographic location of the pest (i.e., different portions of a state). No changes in baseline susceptibility have been detected for any of the target in-

sects exposed to the Cry proteins expressed in Bt potato, Bt corn, and Bt cotton. This information indicates that there has been no measured increase in tolerance to date to the Cry proteins expressed in Bt crops.

Laboratory-tolerant colonies of CPB, ECB, TBW, CBW, and PBW have been created through selection against purified Cry proteins or mixtures of Cry proteins using Bt microbial pesticides. However, the CBW laboratory colonies tolerant to high levels of Cry proteins do not currently exist. The ability of insects to develop high levels of tolerance to Bt in the laboratory indicates that these insects possess the genetic potential to develop resistance to Cry delta-endotoxins expressed as Bt plant-pesticides. It is unlikely that laboratory selective procedures provide the identical selective conditions that exist in the field. However, the ability to select for tolerance to Cry proteins in the laboratory in different insect pests indicates that it is prudent to use appropriate resistance management strategies.

In 1996, cotton bollworm populations were the highest seen in ten years in parts of the Cotton belt (i.e., Brazos Valley,Texas, Mid-South and Southeast growing regions). Monsanto reported to the Agency the potential Bt cotton control failures as early as July, 1996, and followed up with a full analysis of these incidents in the Fall of 1996. Monsanto performed studies at all Bt cotton areas affected by high cotton infestations to determine whether cotton susceptibility to the CryI(A)c toxin had changed and whether the Bt cotton was expressing the CryI(A)c and whether the CryI(A)c expression levels and patterns had changed. Monsanto also provided the results of these studies in its 1996 annual report on resistance monitoring activities. Results of these studies indicate that there was no change in cotton bollworm susceptibility and no change in Bt expression in the Bt cotton areas affected by high cotton bollworm infestations. These studies indicated no detectable level of resistance in these populations. Unusually high infestation levels of CBW may have, in part, resulted from the dramatic increase in corn acreage in the South. In addition, CBW has a lower sensitivity to the CryI(A)c delta-endotoxin relative to TBW and PBW. Scouting detected the CBW lower in the plant canopy of Bt cotton than expected and, in some cases, supplemental chemical insecticides were used to control CBW. The fact that supplemental insecticides might be necessary to control unusually high CBW infestations was not unexpected and was considered in the Agency's review of the initial resistance management strategy for Bt cotton. Modifications to the CBW scouting program for Bt cotton were made for the 1997 season to improve detection of the CBW larvae which might escape the Bt delta-endotoxin by feeding on blooms and bloom tags that are lower in the cotton plant.

Most cotton growers complied with the structured refuge requirements. Cotton growers seem to prefer the 20% sprayed refuge option which allows them to treat the refuge with chemical insecticides normally used to control TBW, CBW, and PBW (except for Bt microbial pesticides). This option appears to more reliably provide a higher yield in the refuge acreage than the 4% unsprayed refuge option which often had higher management costs and lower yields. Most cotton researchers who commented at the two public hearings, held in March and May 1997, favored the 20% structured refuge as a better strategy for Bt cotton resistance management. They believed that this refuge option is more likely to provide a greater percentage of susceptible insects throughout the growing season to mate with any rare resistant individuals that might survive in the Bt cotton fields. EPA received comments that the 4% unsprayed refuge was decimated early in the growing season so that there were few, if any, adult moths surviving to mate with any resistant insects that survived in the Bt cotton fields later in the growing season.

EPA believed that during the first five years following the first complete growing season in 1996, there would not be enough Bt corn acreage to provide substantial Bt selection pressure for the development of ECB resistance. Consequently, EPA did not mandate specific refuge requirements for Bt corn, but EPA has required research data on the size, structure, and deployment of a structured refuge. A combination of temporal and structured refuges are being studied. A draft refuge strategy must be submitted to the Agency by August, 1998, and a final refuge strategy is required to be submitted by January, 1999. Implementation of an EPA-approved structured refuge plan or an EPA-approved alternative resistance plan is required no later than April 1, 2001. Monsanto and Dekalb are requiring structured refuges as part of grower agreements. Beginning in the 1998 growing season, Novartis Seeds has adopted the NC-205 consortium's recommendations published in NCR-602 publication entitled "Bt Corn & European Corn Borer - Long Term Success Through Resistance Management" (Ostlie *et al*., 1997). The NC-205 recommended a 20-30% structured non-Bt corn refuge to prevent Bt delta-endotoxin exposure to 20-30% of the larval populations. They also recommended that in continuous corn acreage sprayed with insecticides, the refuge size would be increased to perhaps 40% to compensate for larval mortality. In addition, a smaller refuge size may also be suitable if there are many alternate hosts providing adequate numbers of susceptible ECB. Mycogen has not made any specific refuge recommendations in its Grower Guide, but is supportive of the use of refuges and supportive of the NC-205 recommendations.

Monsanto/Naturemark requires a structured refuge as part of grower agreements for use of Bt potato. EPA has required that Monsanto mandate specific refuge requirements as a condition of registration for Bt cotton. Monsanto has implemented these refuge requirements through a grower agreement. Research is underway to study whether in-field narrow strip refuges or mixed Bt cotton/non-Bt cotton seed mix options are vi-

able for PBW resistance management because of the limited larval movement. Based on Monsanto's reports to the Agency, there has been a high level of compliance with a structured refuge in Bt cotton and Bt potato. EPA is encouraged by reports of a tremendous reduction in the use of conventional insecticides that has resulted from adoption of Bt cotton.

A great deal of research is underway to study the elements that are necessary for long-term resistance management strategies for Bt potato, Bt corn, and Bt cotton. Specific research data were required as part of the Bt corn and Bt cotton conditional registrations and was recommended for the Bt potato registration. These data included: the dosage effectiveness on the target pest(s), monitoring data including baseline susceptibility and validation of the diagnostic dose concentration, pest biology and ecology, influence of the Bt crop on secondary lepidopteran pests, the impact of CryI(A)b/CryI(A)c produced in Bt corn on the selection of CEW/CBW resistance in Bt corn and Bt cotton, impact of Bt on CEW overwintering survival and fecundity, effective refuges, alternate hosts as refuges, and cross-resistance potential. Additionally, alternative pest control strategies and integration into existing IPM programs are being examined for each of the Bt plant-pesticides. All of these data will provide the basis for specific improvements to the existing resistance management strategies. Future information is especially important for understanding the selection of CEW/CBW resistance in overlapping Bt corn and Bt cotton regions of the southern United States. This is because CEW/CBW usually moves from silking corn to cotton, has multiple generations per year, and overwinters in the South. Exposure to Cry delta-endotoxins produced in both Bt corn and Bt cotton in two or more generations per year could rapidly accelerate development of resistance. Research results and predictive models studying this situation are expected to be submitted to the Agency in 1998.

Science advisory panel review of EPA's white paper

The Agency asked the February 9-10, 1998 OPP FIFRA Science Advisory Panel Subpanel on Bt plant-pesticide resistance management to review specific questions posed by EPA based on its "White Paper" (EPA, 1998d) on Bt plant-pesticide resistance management strategies for Bt potato, Bt corn, and Bt cotton. Oral and written statements were received from approximately 20 different groups representing industry, growers or grower groups, trade organizations, academia, and environmental groups. The Subpanel provided the Agency with a final report of the meeting on April 28, 1998 (SAP, 1998). Copies of the written statements and the Subpanel report can be obtained from the OPP Docket Office (OPPTS-00231). The Subpanel's report can also be obtained electronically at the site mentioned above. A brief summary of key points made in the Subpanel report is provided below.

The Subpanel agreed with EPA that the widespread use of crops that express Bt insecticides is in the public good by providing additional pest control options to producers and by reducing the use of conventional pesticides. The Subpanel also agreed with EPA that appropriate resistance management is necessary to suppress the emergence of insect resistant to Bt toxins expressed in transgenic crop plants. The Subpanel recognized that resistance management programs should be based on the use of both high dose expression levels and structured refuges designed to provide sufficient numbers of susceptible adult insects with a minimum of economic impact on producers. Resistance management strategies should be sustainable and to the extent possible, strongly consider grower acceptability and logistical feasibility. The Subpanel made the following overall recommendations: a) EPA should require mandatory resistance management strategies for all Bt plant-pesticides, b) a refuge/high dose strategy is needed to delay the development of resistance, c) EPA should require mandatory structured refuges for all Bt plant-pesticides, d) refinements to existing monitoring and remedial action plans are necessary, e) grower acceptance and implementation of resistance management strategies are essential to the success of long-term resistance management, and f) regional working groups for specific implementation of resistance management strategies should be established for each of the major Bt crop producing regions.

The Subpanel defined a high dose as 25 times the amount of Bt delta-endotoxin necessary to kill susceptible individuals. It is possible that a heterozygote may develop with higher than 25-fold resistance. A cultivar could be considered to provide a high dose if verified by at least two of the following five approaches: (1) Serial dilution bioassay with artificial diet containing lyophilized tissues of Bt plants using tissues from non-Bt plants as controls; (2) Bioassays using plant lines with expression levels approximately 25-fold lower than the commercial cultivar determined by quantitative ELISA or some more reliable technique; (3) Survey large numbers of commercial plants in the field to make sure that the cultivar is at the $LD_{99.9}$ or higher to assure that 95% of heterozygotes would be killed (see Andow and Hutchison, 1998); (4) Similar to (3) above, but would use controlled infestation with a laboratory strain of the pest that had an LD_{50} value similar to field strains; and (5) Determine if a later larval instar of the targeted pest could be found with an LD_{50} that was about 25-fold higher than that of the neonate larvae. If so, the stage could be tested on the Bt crop plants to determine if 95% or more of the later stage larvae were killed.

The Subpanel defined structured refuges to "include all suitable non-Bt host plants for a targeted pest that are planted and managed by people. These refuges could be planted to offer refuges at the same time when the Bt crops are available to the pests or at times when the Bt crops are not available." The Subpanel stated that a good resistance management strategy should provide efficacy of the

toxin(s) for more than 10 years. The Subpanel suggested that a production of 500 adults in the refuge that move into the transgenic fields for every adult in the transgenic crop area (assuming a resistance allele frequency of 5×10^{-2}) would be a suitable goal. The placement and size of the structured refuge employed should be based on the current understanding of the pest biology data and the technology.

EPA is reviewing the Subpanel report and other materials submitted as a result of the February 9-10, 1998 SAP Subpanel Meeting. This information will contribute to how EPA continues to evaluate and refine its regulation of resistance management for Bt plant-pesticides. EPA will continue to work with stakeholders from industry, academia, extension entomologists, user groups, trade organizations, public interest groups, and government agencies to address long-term resistance management for Bt plant-pesticides.

North American Free Trade Agreement (NAFTA) project on pesticide resistance management labeling

Canada, the U.S., and Mexico have joined together under NAFTA to develop voluntary pesticide labeling guidelines for pesticide resistance management (NAFTA Project RR970RF). This voluntary initiative was originally proposed by Canada's Pest Management Regulatory Agency (PMRA) in December, 1996, and became part of NAFTA in June, 1997. EPA's Office of Pesticide Program's Pesticide Resistance Management Workgroup (PRMW[1]) reviewed the original December, 1996, draft guidelines and provided comments on the recent December, 1997, draft. The Pesticide Resistance Management Labeling Guidelines are based on labeling for target site mode of action and including standard label statements concerning resistance management for all classes of pesticides. The use of standard statements will simplify label review and facilitate user comprehension of label statements concerning pesticide resistance management. EPA will continue to work with Canada and Mexico on this voluntary initiative.

Section 18 policy revision and resistance management

EPA is seeking to clarify its guidance and regulations for issuing emergency exemptions as reported in *Resistant Pest Management* in 1997 (Matten, 1997). Following the November, 1996, Stakeholder meeting held in Washington D.C., EPA decided to revise its Section 18 policy to allow emergency exemptions for one, two or more requested pesticides (with different modes of action) for resistance management based on strict criteria. These criteria are being developed. EPA is seeking to eliminate unfounded claims of resistance problems and limit emergency exemptions based on pest resistance to the most serious situations that can be legitimately proven. It is also hoped that clarity in EPA's guidance for issuing emergency exemption based on pest resistance management will improve the Agency's ability to manage pest resistance by allowing unregistered uses of pesticides with different modes of action to be used to control emergency situations.

In the future

EPA is continuing to evaluate and refine the role that pest resistance management has in pesticide regulatory decisions. EPA believes that it is good public policy to manage pesticide use to minimize the development of pesticide resistance. Effective pesticide resistance management can reduce the total pesticide burden on the environment and reduce the overall human and ecological exposure to pesticides. EPA joins other stakeholders in addressing long-term resistance management issues and developing scientifically-sound and efficient resistance management strategies that will not be overly burdensome to the regulated community, jeopardize the use of reduced risk pesticides, exclude conventional pesticides that contribute to the overall concept of integrated pest management, or jeopardize their adoption by the grower community.

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Inheritance of pesticide resistance in stored product insects

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Insect pests cause significant losses in quality and quantity of stored products and pose a major sanitation and quality control problem for the food industry. Treatments with chemicals including application of contact insecticides and fumigations are the only economical methods available at present for the protection of stored products. Over the years, however, evidence has been accumulating on the occurrence of resistance to these chemicals among field populations of stored product insect pests. Reviews are available in literature on the incidence of resistance in stored product insects in different parts of the world (Champ 1986, Badwin 1990). Despite quarantine regulations, the resistant insects have been able to spread from one country to another through trade channels and the occurrence of resistant strains has been recorded even in countries where the particular insecticide has not been used at all (Champ 1986). In the developed nations, there is a nil tolerance for insect infestation in food grains and in view of the growing public awareness for quality food commodities, the demand is likely to expand to other countries also. The actual monetary and manpower costs of resistance have not been quantified yet. But, in a bid to overcome resistance problem, application frequencies and dosage rates of pesticides have been increased, resulting in higher contamination of our food and the environment (Roush & Mckenzie 1987). Hence, persons engaged in food commodity storage and preservation worldover , are concerned about the resistance of stored product insects to contact insecticides and fumigants. It is, therefore, necessary to work out strategies to delay or minimise the probability of resistance evolution. This will be possible only by studying the various facets of resistance such as insect physiology, population ecology and the genetics with reference to insecticides. A knowledge about the inheritance of resistance is very important to understand the development, rate of spread and stability of resistance in the population. Champ and Dyte (1976) in their report on the global survey of pesticide resistance examined the genetics of pesticide resistance in stored grain insects. The present review discusses the inheritance of resistance to both contact insecticides and fumigants by stored product insect pests.

Mode of inheritance

Resistance to an individual pesticide may be due to a single gene (monogenic or monofactorial inheritance) or due to more than one gene (polygenic or polyfactorial inheritance). The gene(s) involved may be of recessive nature or partly or fully dominant. The resistance gene may be found on the sex chromosome (sex-linked inheritance) or on the autosomal chromosomes. The type of inheritance of resistance in stored product insects has been generally determined by bioassays of insects from a backcross between F1 (hybrid of resistant and susceptible strains) and potential resistant or susceptible strains. A few workers have discussed the type of inheritance based only on the pattern of *ld-pm* lines obtained during successive selections in the laboratory without any confirmatory backcross tests. Shukla and Srivastava (1984) studied malathion resistance in the tropical warehouse moth, *Ephestia cautella* from India by selections for 8 generations in the laboratory and based on the regression lines, they reported that resistance could be controlled by a single or few genes. In addition to backcross tests, genetic marker genes as well as molecular techniques have also been used to ascertain the type of inheritance (Tabashnik 1991). Reports on inheritance of pesticide resistance in stored product insect pests are summarised in Table 1.

Table 1. Inheritance of pesticide resistance in insect pests of stored products.

Pesticide	Insect	Inheritance			Reference
		Type	Dominance	Linkage	
Insecticides					
Malathion	*O.surinamensis*	Monofactorial	Dominant	Autosomal	PICL,1981
		Two alleles	Partial	*VI*	Beeman & Nanis,198(
	R.dominica	Monofactorial	Partial	*b*	Champ & Dyte,1976
	T.castaneum	Monofactorial	Partial	Autosomal	Kaur,1993; White & Bell,1988
		Monofactorial	Dominant	Autosomal	Noiman & Wool,1982;
		Multifactorial	Dominant	Autosomal	Pasalu & Bhatia,1983
		Monofactorial	Dominant	Autosomal + *Y*	Wool *et al*.,1982
		Monofactorial	Partial	*VI, VIII,* + *X*	Wool *et al*., 1982
		Monofactorial	Partial	*VI*	Champ & Dyte,1976
		Monofactorial	Recessive	Autosomal	Beeman, 1983 *a*
	P.interpunctella	Monofactorial	Partial	Autosomal	Beeman, 1983 *b* Attia, 1981
Fenitrothion	*O.surinamensis*	Multifactorial	Partial	Autosomal	Collins, 1986
Pyrethrins	*S.granarius*	Monofactorial	Partial	Autosomal	Lloyd & Shaw, 1968
Lindane	*S.oryzae*	Monofactorial	Partial	Autosomal	Champ & Dyte, 1976
		Two factors	Partial	Autosomal	Champ & Cribb, 1965
	S.granarius	Monofactorial	Partial	Autosomal	PICL, 1981
		Monofactorial	Partial	Autosomal+ *X*	PICL, 1981
	T.castaneum	Multifactorial	Partial (?)	*II,IV,V,VIII,* + *X*	Champ & Campbell-Brown, 1969
		Monofactorial	Partial	Autosomal	Kumar & Bhatia, 198
p,p'DDT	*S.oryzae*	Monofactorial (+ alleles ?)	Partial	Sex-linked	Champ, 1967
		Monofactorial	Recessive	*IV* + *m*	Erdman, 1970
		Monofactorial	Partial	Autosomal	Bhatia & Panicker, 1976
	P.interpunctella	Monofactorial	Partial	Autosomal	Attia, 1981
Fumigants					
Phosphine	*R.dominica*	Monofactorial (+ allele ?)	Partial	Autosomal	Ansell *et al*., 1990
	S.oryzae	Multifactorial	Partial	Autosomal	Li Yan-sheng & Li Wen-zhi, 1994
	T.castaneum		Partial	Autosomal	Bekon *et al*., 1988
		Multifactorial	Recessive	*II*	Ansell *et,al*., 1990
		Multifactorial	Partial	Autosomal	Bengston et al., in press
Methyl bromide	*S.granarius*	Polyfactorial		Autosomal	Upitis *et al*., 1973

Though resistance statistics in field strains indicate 18 species of Coleoptera and 7 of Lepidoptera, studies on inheritance of resistance have been confined to five Coleoptera species covering five insecticides (two organophosphates, two organochlorine compounds and one pyrethrin) and two fumigants, and one Lepidoptera. The majority of the investigations involve the red flour beetle, *Tribolium castaneum,* since comprehensive literature on the genetics of the species is already available (Champ & Dyte 1976). Usually, a single factor which is incompletely dominant and autosomal has been implicated in the inheritance of resistance. However, full dominance has been observed only in two malathion resistant species i.e, *T. castaneum* from Nigeria (Noiman & Wool 1982, Wool et al. 1982) and the sawtoothed grain beetle, *Oryzaephilus surinamensis,* four strains of different origins (PICL 1981). Recessive alleles have been implicated in p,p DDT resistance in the rice weevil, *Sitophilus oryzae* (Erdman 1970) and in phosphine resistance in *T. castaneum* (Ansell et al.1990). There has been no difference in the type of inheritance of malathion - specific and malathion - non-specific resistance in the insects studied. Among moth pests, inheritance of resistance has been investigated only in the Indian mealmoth, *Plodia interpunctella,* with reference to malathion and DDT.

Linkage to sex chromosomes has been

implied in a few cases. For example, Wool et al. (1982) reported that males of *T. castaneum* in a strain from Kano, Nigeria, were more resistant than females and this selective response has been attributed to the involvement of a factor on the Y chromosome in the population. Linkage to X chromosome has been indicated in other instances (Champ & Dyte 1976, PICL 1981).

Most of the stored product insect species have been known to have developed resistance to phosphine fumigant and control failures have already been documented in some countries (Taylor 1989, Rajendran & Narasimhan 1994). Hence insect resistance to phosphine has been considered as a serious threat to the continued use of the fumigant. Nevertheless, genetic studies with reference to phosphine resistance has been made only in four species. Polygenic control of resistance has been observed in most cases (Ansell et al. 1990, Li yan - sheng & Li Wen - zhi 1994, Bengston et al. 1997). Perhaps this is the reason for the multiple mechanism of resistance in phosphine resistant insects (Chaudhary 1997). Evidently, sex linkage in inheritance of fumigant resistance has not been noticed.

Genetic, reproductive, behavioural and operational factors have been implicated in the evolution of resistance (Wood 1981). Among them, genetic factors have been considered as the basic ones. Genetic models have been proposed to elucidate, to delay and/or predict the onset of resistance, mainly for the Diptera comprising insect pests of public health importance. To work out such models the required data such as the rate of change in gene frequencies and the relative fitness of the genotype in the presence or absence of insecticides are available only for a very few stored product insects. Muggleton (1986) generated data on the relative fitness of the resistant homozygote, the heterozygote and the susceptible homozygote after subjecting malathion-resistant *O. surinamensis* to selection for 10 generations at three different doses. His work revealed that resistance is delayed to the longest period of time at the lowest dose. The relative fitness value of heterozygotes compared to the resistant homozygote at the lowest and highest doses of selection were 1.0 and 0.4, respectively. The relative fitness of susceptible and resistant strains of *S. oryzae* in the presence and absence of deltamethrin and pirimiphos-methyl has been studied by Longstaff (1991).

Polygenic inheritance is likely to spread at a slower rate under field conditions unlike resistance conferred by a single gene (Roush & McKenzie 1987). Phosphine resistance, though multifactorial, has developed and spread quickly in most parts of the world probably due to continuous use of this particular chemical, since suitable alternative fumigants have not been available, resulting in intensive selection pressure. It is believed that there is a relationship such as single gene - single detoxifying enzyme and polygenes - multiple target sites/ enzyme systems (Collins 1986). However data correlating the number of genetic factors with the enzyme systems including total esterases, carboxyesterases, mixed function oxidases and glutathione transferases for resistant insects are lacking. Cases wherein a single genetic mechanism conferring resistance to a range of unrelated insecticides have been recorded (Muggleton 1987). Biological attributes linked with resistance have been characterised in some insects. These include changes in body weight, fecundity, developmental rates, feeding and locomotory behaviour and genetic fitness (Kumar & Gupta 1984).

Management of resistance

For the management of resistance, the available options include use of pesticides in rotation or in sequence, application of pesticides preferably belonging to different classes in mixtures and change over to alternate chemicals. Currently we have enough contact pesticides and a few insect growth regulators to adopt such changes. Unfortunately we have limited options with regard to fumigants. At present phosphine and methyl bromide are the only fumigants widely used for pest control in stored products. The latter has been declared as an ozone depleting substance and it is under severe criticism by environmentalists and regulatory bodies and therefore the fumigant is being phased out. With regard to phosphine resistance attempts have been already made by the developed nations to improve the application methods for the effective use of the fumigant. Changes in application techniques e.g. SIROFLO (a patented phosphine application method using cylenderised phosphine) in Australia, have been worked out in developed countries.

To prevent the genetic spread of resistance it has been suggested to introduce susceptible males facilitating infusion of susceptible genotypes into a resistant population. In laboratory tests with malathion resistant *T.castaneum* and Ephestia cautella in Isreal, this concept has given satisfactory results (Wool & Manheim 1980,Wool et al. 1992). In a subsequent study in the confused flour beetle, *Tribolium confusum*, resistant to malathion, Wool and Noiman (1983) proposed a combination of insecticide application and the release of susceptible males as an integrated approach in ameliorating insecticide resistance. But, in practice, release of any insect pests into a food commodity storage premise is not acceptable as the objective is to maintain stored food commodities without any infestation.

Another way of delaying resistance would be to use the highest dose of the insecticide, while at the same time permitting an acceptable proportion of insects to remain untreated (Muggleton 1986). To allow some insects to survive without treatment, it requires refuges. It is expected that insects from the refuges will allow gene flow into the resistant population to dilute the homozygous resistant strain. However, experiments on *O. surinamensis* conducted in UK in bins under simulated field conditions with pirimiphos-methyl yielded negative results, as the insects from refuges not only moved to treated surfaces, but also developed resistance (Mason et al. 1997).

SUMMARY

Detailed genetic studies on the inheritance of pesticide resistance are limited to only six species of stored product insects. Obviously, the type of inheritance and the number of genes involved vary between species and between strains in the same species. Sex linkage has been observed rarely. Genetic models and relevant data to work out strategies with reference to resistance in stored product insects are not available as much as those for insect pests of public health importance like mosquitoes and houseflies. More studies on this aspect are urgently required involving predominant species, especially moth pests and on phosphine fumigant.

ACKNOWLEDGEMENTS

The authors thank the Australian Centre for International Agricultural Research for financial support of the Project PN 9415 and the Director, CFTRI for encouragement.

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Insecticides: status of resistance by bollworm and tobacco budworm in the United States

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There are no populations of the bollworm which are resistant to registered insecticides used in commercially grown fields of corn, cotton or vegetable crops. There can be some portion of a population with elevated levels of response. This does not mean that the populations are resistant because the elevated levels for that portion are not sustained.

Corn is the major and favorite host of this insect but most commercially grown fields are either not treated or inadequately treated. Inadequately treated corn does little to cause resistance in portions of populations. Methomyl is applied to corn and it is still toxic to this insect. Methyl parathion is applied to cotton and it is still toxic to this insect. The pyrethroids are toxic to this insect on any crop. This insect also has many wild plant hosts which are never treated.

Resistance to insecticides is present in portions of populations of the tobacco budworm. The portion that is resistant to any insecticide in each commercial field of cotton or vegetable crop will not be 100%. Some populations may not have any resistant individuals at one time in one field or fields across an area. Large populations [>1 egg or larva/cotton plant or >5 egg or larva/vegetable plant] may not be resistant. There may be too many survivors for adequate control on cotton or any vegetable crop even if 90% are killed. They may all be susceptible, but the most likely scenario is that some portion is resistant to one insecticide. Some type of bioassay for the insecticide[s]s in question can be used to indicate the portion of the population which is resistant.

The pyrethroids are the most widely used insecticides for control of this insect on cotton and vegetables. Organophosphorus i.e. methyl parathion and profenofos, and carbamate i.e. thiodicarb, insecticides are also widely used. The insecticides control >50% of the populations when applied correctly. Ninety percent control is probably the best control which can be obtained with an application of any insecticide.

Resistance by this insect to transgenic cotton has not been found in the field. This protein is toxic to larvae of the tobacco budworm. Today, refugia, plants without the protein are planted in or near the transgenic cotton, is the method used to prevent resistance. Transgenic cotton plants are a setup for resistance because the insecticide is present in >98% if tge plants 100% of the time. No transgenic vegetable crop has been developed which is attacked by this pest. Foliar sprays of formulations of *Bacillus thuringiensus* have been shown to be effective against this insect in fields of both vegetables and cotton if correctly used.

Variation in response to an insecticide is universal for tobacco budworm populations because any individual insect in any cotton or vegetable field can be resistant, susceptible or some degree between the two designations. If a population of the bollworm and tobacco budworm is collected from any crop and location is subdivided into smaller populations and each subdivision bioassayed this variation can be determined.

What stage of either species should be used to bioassay for resistance to any insecticide? If larvae should be bioassayed what stadia should be selected? Does resistance or susceptibility of adult populations from field populations indicate the same for larvae the next generation? Does resistance of larvae indicate resistance by adults in the same generation? These questions are proposed so bioassays will be used to designate resistance or susceptibility of these populations in each field at any time.

The size of populations of all stages of both species at any time at any location across the United Stated is unknown. The size is dynamic and will change frequently. This also makes the size of the portion of each population of the tobacco budworm unknown.

Dispersal of immature stages is minimal. Adults of both species can and do disperse. Wind patterns have the potential to cause large numbers of adults to move from one location to another. These patterns are capable of moving large numbers in a short period of time. Adults of these species live 12 to 18 d and females lay their complement of eggs from 2 to 10 d of their lifespan. Females usually mate 1 to 2 times during their lifetime with 1 or 2 males. Response levels may change depending on whether the female mated with a susceptible male and then with a resistant male. Time is of importance for perpetuation of these species and the mating and oviposition process reduces time for dispersal.

Firko and Wolfenbarger [1991] stated that it was difficulty to increase tolerance [=resistance] because of the genetic basis of this insect. Wolfenbarger [1990] showed a large depression in the response level of permethrin by a field collected strain of tobacco budworm after eight generations of selection. The selected population from one single pair was almost immune one generation, but totally susceptible the next generation. Not enough is known about the inheritance of resistance in tobacco budworm populations. Future experiments should be conducted to define inheritance of resistance factors and how they move in proportion of populations in the field. The problems of determining the number of genes responsible for the resistance factors and how they move in proportion of populations in the field. The problems of determining the number of genes responsible for the resistance levels in progeny from crosses of strains were discussed by Firko ;1991] . Not enough is known about inheritance of biological activities [i.e. fitness] which may directly or indirectly affect resistance or susceptibility. I feel that some kind of "gene switching", pleitropism or complex inheritance factors are present in proportions of populations of tobacco budworm that are responsible for reversion of response

[resistance to susceptibility]. If this is occurring it must be determined. Will all or part of a resistant portion of populations become susceptible following constant selection? Co-dominance or incomplete dominance is prevalent among responses of crosses of strains which comprise that portion of a population which is resistant and the portion which is susceptible. Sex linkage with the male is also a factor.

Resistance should only be defined by results of a bioassay from portions in the populations of both insect species which survive in the field following spray applications. If there are 100,000 fields of cotton and vegetables there are 100,000 scenarios for response.

The laboratory should be used to collect the data, such as the level of resistance or susceptibility from one generation to the next, the biochemical mechanisms involved and the mode of inheritance of these mechanisms which cause the movement or level of resistance in the populations of both of these species, Insects will be from individuals or populations from field collections.

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New action plan for managing herbicide-resistant grass-weed

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The UK Weed Resistance Action Group (WRAG) has recently produced an 8 page leaflet: "Revised Guidelines for Preventing and Managing Herbicide-Resistant Grass-Weeds". This was produced with funding from the Home-Grown Cereals Authority and updates the previous 1993 edition which dealt only with black-grass (*Alopecurus myosuroides).* The new edition incorporates results from the most recent research and extends coverage to include resistant wild-oats (*Avena spp.)* and Italian rye-grass (*Lolium multiflorum).*

Herbicide-resistant black-grass occurs on over 750 farms in England, which represents about 4% of cereal farms with this weed. However less than 10% of farms have had samples tested for resistance so the true extent of the problem is almost certainly greater. Herbicide-resistant wild-oats and Italian rye-grass are more limited problems at present, having been found on fewer than 50 farms so far in the UK. However the farms affected are widely distributed, so the potential for an increase in resistance problems with these weeds is very real.

The Revised Guidelines provide information on resistance risk factors, recognition of resistance in the field, and present key management advice to prevent, or at least delay, the development of resistance. Advice is also given on the best ways to contain the problem and minimise its impact if resistance has already developed. The Guidelines include detailed information on cultural control methods, a table of all the herbicides available in the UK for controlling these weeds grouped according to mode of action and a summary of recent research results. Emphasis is placed on adopting long term strategies integrating cultural and chemical control methods.

The Guidelines are aimed primarily at farmers and agronomists, and technical personnel who require all the background information. A complementary poster ("Keeping Herbicide-Resistant Grass-Weeds at Bay Throughout the Year") has also been produced with sponsorship from the British Crop Protection Council (BCPC) and support of Crops Magazine, and this presents the key management elements in a more concise format.

Although the Guidelines relate to agronomic systems in the UK, the general principles, if not the specific herbicide advice, is relevant to the management of resistant weeds generally.

The UK Weed Resistance Action Group (WRAG) was formed in 1989 and comprises an informal group of representatives from independent organisations and British Agrochemical Association (BAA) member companies involved in herbicide resistance research. It is independent from, but maintains excellent liaison with, the international Herbicide Resistance Action Committee (HRAC), which has representation only from the agrochemical industry.

Copies of the Guidelines and the poster are available free of charge from the Home Grown Cereals Authority, Caledonia House, 223 Pentonville Road, King's Cross, London N1 9NG, UK or from the British Agrochemicals Association Ltd., 4 Lincoln Court, Lincoln Road, Peterborough PE1 2RP, UK.

Further information about the UK Weed Resistance Action Group (WRAG) can be obtained from the secretary.

Pyrethroid resistance in horn flies in Argentina

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Pyrethroid resistance in Argentine horn flies was first detected in 1996 in Corrientes Province (Sheppard and Torres 1996). At that time horn flies in other areas sampled were highly susceptible, exhibiting fenvalerate $LC_{50's}$ lower than a susceptible colony of horn flies. Since then reports of poor control in other areas have raised concerns that this problem is spreading. In late 1997 and early 1998 Pyrethroid resistance in horn flies was evaluated in Corrientes, Entre Rios and Buenos Aires Provinces, where apparently resistant horn flies are causing concern among cattle producers. $LC_{50's}$ were determined using the petri dish bioassay of Sheppard and Hinkle (1987). Resistance ratios were calculated by dividing the Argentine horn fly LC_{50} by the 1997 average LC_{50} of the Tifton susceptible horn fly colony (0.40 ug/cm^2).

The resistance ratio in Corrientes province was 40.4, somewhat higher than that found in 1996 (Sheppard & Torres, 1998). In Entre Rios Province, the Province located immediately South of Corrientes Province, the resistance ratio was 31.8, which is much higher than the 0.21 or 0.39 found in 1995 (Sheppard & Torres, 1998). This is the first report of pyrethroid resistance in this province. A test conducted in Buenos Aires Province did not produce a regression, but indicated a resistance ratio comparable to those in Corrientes or Entre Rios Province. We did not anticipate this response and bioassay doses were too low to produce any significant kill.

These data show that Pyrethroid resistance in horn flies is well established in the three provinces that were sampled, with resistance in two provinces where horn flies were extra susceptible 2-3 years ago. These populations should be monitored and strategies for control should be implemented. Available information indicates that Organophosphates are effective.

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Resistance around the globe

Control of isoproturon resistant biotypes of *Phalaris minor* by chlorotoluron and clodinafop-propargyl

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The resistance of *Phalaris minor* (littleseed canarygrass) to isoproturon in India, documented in 1991-92 (Malik & Singh, 1995) has increased in 0.8 million ha of wheat; heavy weed intensity (»2000 plants of *P. minor*/m2) causes considerable loss in crop yield especially in the rice-wheat rotation areas of Haryana and Punjab states. Investigations have shown that resistance to isoproturon was not due to alteration at the target site as in vitro oxygen evolution (photosynthesis) and chlorophyll fluorescence were equally inhibited by isoproturon in the resistant (R) and susceptible (S) biotypes of *P. minor* (Singh et al., 1997a). The recovery of chlorophyll fluorescence in the R biotype, however, was complete within 24 h of removing the treated leaves from herbicide solution; wheat was slower to recover than the R biotype and the S biotype of *P. minor* did not recover. Uptake and translocation of [14C] isoproturon were similar in the R and S biotypes of *P. minor*, whereas the degradation was more rapid in the R than S biotype (Singh et al., 1996a). The R biotype was found to mimic wheat in the apparent mechanism of isoproturon degradation (Singh et al., 1996 b).

The present experiment was undertaken to compare the effect of chlorotoluron, clodinafop-propargyl and isoproturon on the R and S biotypes of *P. minor* under controlled environment conditions. Chlorotoluron is structurally similar to isoproturon and affects the same target site (photosystem II), while clodinafop-propargyl inhibits acetyl Co carboxylase (ACCase).

MATERIALS & METHODS

Plants of *P. minor* biotypes H-2 (S), seed collected from Research farm of CCS Haryana Agricultural University, Hisar, Haryana, India; H-3 from farmers' field in Hisar district under rice-wheat rotations and KR-1 from Kurukshetra district (both R), were raised in the glasshouse (14 h photoperiod with mercury vapour lamps at 198.6 mE m-2 s-1, 33 ± 4/17 ± 3°C maximum/minimum temperature) in polystyrene pots in a field soil of sandy loam texture. At the 2-3 leaf stage plants were transferred to

Hewitt nutrient solution in 50 ml bottles in a growth room (16 h photoperiod with fluorescent lamps at 83 mE m-2 s-1 photon flux density, 31 ± 1/24 ± 3°C day/night temperature and 84 ± 0/44 ± 6 % maximum/minimum RH). After 3 days the nutrient solution was replaced with isoproturon (Sabre, 55.3 % SC.; AgrEvo), chlorotoluron (Dicurane, 70 % SC., Ciba-Geigy) both at concentration of 0, 0.31, 0.62, 1.25, 2.5, 5.0 and 10.0 mM and clodinafop-propargyl (Topic 24 % EC; Ciba-Geigy) at 0, 0.062, 0.125, 0.25, 0.50, 1.0 and 2.0 mM. There were four replicate plants for each treatment and species. Plants were replenished with herbicide/nutrient solution as required.

Fresh and dry weights (DW) of shoots were recorded at harvest (3 weeks after treatment) and data were analysed by analysis of variance (ANOVA). DW accumulated by the plants at the start of experiment (treatment with herbicides) was subtracted from the final DW at harvest and percent DW of control is presented in Figures on log scales. At harvest the average DW of the H-2, H-3 and KR-1 biotypes was 142, 145 and 132 mg/plant, respectively.

The experiment using the three test species was repeated under similar growth conditions in pots and plants sprayed at various doses with isoproturon, chlorotoluron and clodinafop-propargyl, data from only one experiment is presented.

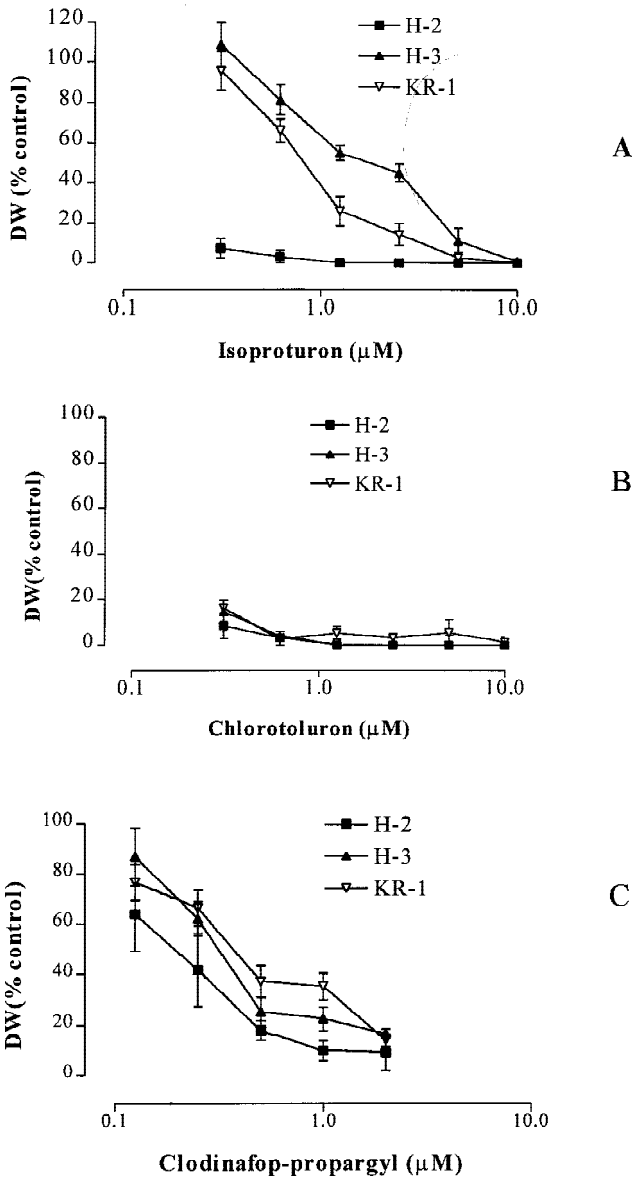

Figure 1. The effect of isoproturon (A), chlorotoluron (B) and clodinafop-propargyl (C) on DW of three test biotypes of *P. minor.* (bars represents mean standard error)

RESULTS & DISCUSSION

Effect on DW

At the lowest concentration (0.31mM) isoproturon reduced the DW of the S biotype by >80% over the control treatment; similar effects were observed with isoproturon at 2.5 and 5 mM in the KR-1 and H-3 biotypes, respectively (Fig. 1A), whereas chlorotoluron (0.31 mM) reduced the DW of all three biotypes by more than 80 % (Fig. 1B). Clodinafop-propargyl was less effective than chlorotoluron and the effect varied according to the biotypes (Fig. 1C). In contrast to isoproturon, the reduction in DW with clodinafop-propargyl was greater in the H-3 than KR-1 biotype. Chlorotoluron resulted in complete kill of both R and S biotypes, whereas phytotoxicity resulting from clodinafop-propargyl developed slowly and the effect was less than chlorotoluron against the R biotypes. Clodinafop-propargyl (1.0 mM) reduced the DW of the H-2, H-3 and KR-1 biotypes by 90, 77 and 65 % of the control, respectively. The effect of the highest concentration (2.0 mM) of clodinafop-propargyl was equivalent to the lowest concentration of chlorotoluron (0.31 mM) against the R biotypes of *P. minor* (Fig. 1B & 1C).

The H-3 (R) biotype required a higher concentration of isoproturon than KR-1 (R) thus exhibiting a higher level of resistance, conforming with earlier observations (Singh et al., 1995). The chlorotoluron resistant biotypes of A. myosuroides and L. rigidum are also cross-resistant to isoproturon (Kemp et al., 1990; Burnet et al., 1991); the level of resistance, however, was lower with

isoproturon than chlorotoluron. Resistance to phenylurea herbicides in these grass weeds is considered to be due to increased activity of cytochrome P-450 enzymes. Both chlorotoluron and isoproturon are degraded by P-450 monooxygenase enzymes in wheat; their differential response in the R biotypes of *P. minor* suggests that there could be different P-450 isozymes degrading these herbicides. Experiments conducted during 1998, under similar conditions but with lower concentrations of these herbicides, revealed no significant differences in the activity of chlorotoluron against R and S biotypes. The inhibitory effect of chlorotoluron on chlorophyll fluorescence was also observed in the R (KR-1 and H-3) and S (H-2) biotypes of *P. minor* (Singh, 1998 unpublished data) which conforms with chlorotoluron activity in the whole plant study. Similar trends in effect of isoproturon, chlorotoluron and clodinafop-propargyl were observed in the activity study with the R and S biotypes of *P. minor* in the pot study (data not presented).

In Israel, fenoxaprop-P resistant biotype of *P. minor* was found to be target site cross-resistant to clodinafop-propargyl (Tal et al., 1996); no resistance was observed, however, with isoproturon and methabenzthiazuron (another phenylurea herbicide) at the used dose rates. The resistant biotypes of *P. minor* from India have shown differential responses to clodinafop-propargyl under controlled environmental conditions; a lower level of cross-resistance in the fields has been observed within one year of application. Preliminary field trials with clodinafop-propargyl, fenoxaprop, sulfosulfuron+adjuvant and tralkoxydim at two locations suggest control of R biotypes of *P. minor* in the affected areas in India, though variations were observed in the activity of these herbicides under conventional and stale seedbed conditions (Malik and Yadav, 1997). Cross- resistance to clodinafop-propargyl was also observed recently in diclofop resistant Eleusine indica in Malaysia (Tiw et al., 1997) and to chlorotoluron resistant A. myosuroides in the UK (Ryan and Mills, 1997; Reed et al., 1997). *P. minor* has shown cross-resistance to diclofop-methyl and pot studies indicate some cross-resistance to clodinafop-propargyl.

The other herbicides which have provided good control of R biotypes of *P. minor* under pot studies are terbutryne, propachlor, tralkoxydim, fenoxaprop-P-ethyl, trifluralin and pendimethalin (Kirkwood et al., 1997). Metazachlor and atrazine also provided good control of the R biotypes but these herbicides were also phytotoxic to wheat at higher doses and can only be used at lower doses in combination with other herbicides. Some of these herbicides need to be used in mixtures to increase the spectrum of weed control. Rotation of these herbicides and integration of physiological and agronomic factors can help in effective management of the R biotypes of *P. minor* under field conditions and delaying the onset of resistance and (Singh et al., 1997b).

Based on these observations, field evaluation of chlorotoluron in the resistance affected areas of Haryana state has been undertaken during the present wheat growing season (1997-98) (Malik, R. K., personal communication). If found effective in controlling the R biotypes, chlorotoluron offers a unique choice to Indian farmers because of its easy availability and low cost compared to other herbicides; if not used in rotation, however, it may soon become useless. Availability of more than one herbicide for controlling *P. minor* offers a choice to farmers for rotation of crops and herbicides. Evaluation of new wheat varieties for their sensitivity to chlorotoluron and clodinafop-propargyl is also required. Clodinafop-propargyl may provide control of the R biotypes of *P. minor* in India for some time as was observed with diclofop-methyl when it was recommended in 1994 for managing isoproturon resistance. After 2-3 applications of diclofop-methyl control of the R biotypes declined considerably at farmers' fields and increasing its dose by even 2 times failed to provide an acceptable control. The differential response of clodinafop-propargyl seen in the laboratory indicates that the evolutionary process under selection process has begun.

ACKNOWLEDGEMENTS

The authors wish to thank Prof. Jonathan Gressel, Dept. of Plant Genetics, Weizmann Institute of Science, Rehovot, Israel for his continuing interest and advice in relation to these studies in the mechanism of resistance and management of resistant *P. minor*. Thanks are also due to Dr. Stephen Moss, IACR Rothamsted, UK for providing chlorotoluron and clodinafop-propargyl and for many helpful discussions.

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Prevalence of the cypermethrin resistance gene in field-collected populations of the German cockroach

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The development of resistance to any insecticide within a population of insects depends on whether the genetic determinant for the resistance mechanism is present within the gene pool of the population. If the gene is absent, resistance will not develop unless a mutation occurs within that population during the course of exposure to the insecticide. Contrarily, if the gene for resistance to that insecticide is present within the population, then resistance can develop. Whether it does depends on several factors. Among them are the size of the population, the frequency of the gene in the population, the dominance relationship of the gene, and the intensity with which the population is exposed to the insecticide.

Gene frequency can vary from 0 (i.e., absent) to 1.0 (i.e., fixed in the population). It is usually assumed that the frequency of an unselected gene for resistance is initially extremely low. Values as low as 10^{-3} to 10^{-6} have been quoted (Roush and McKenzie 1987). Hence, the larger the population the more likely that population will contain individuals carrying the resistance gene. The speed with which that gene increases in frequency depends heavily on the level of exposure to the insecticide and the dominance relationship of the resistance gene. If the population is only occasionally exposed to the insecticide, then the frequency of the gene may change little if at all. However, if the population is routinely exposed, individuals carrying the resistance gene should have an advantage. Whether they do and how much of an advantage they have depends on the dominance relationship of the resistance gene (Cochran 1994a).

In the case of genes that are dominant, as occurs in German cockroaches, *Blattella germanica* (L.), resistant to malathion or pyrethrins (Cochran 1973, 1994b, respectively), individuals that are heterozygous for the gene show high-level resistance. Under these circumstances, it is expected that gene frequency will increase rapidly because any mating of a heterozygous individual will produce more heterozygotes and soon homozygous-resistant individuals will also appear. The result is the rapid occurrence of highly-resistant populations, as occurred with malathion (Bennett and Spink 1968) and pyrethrins (Cochran 1994a).

Resistance gene is recessive, it is often the case that it is incompletely recessive (Cochran 1994a, Ebbett and Cochran 1997). When this is true, heterozygotes have a slight advantage allowing at least some of them to survive exposure to levels of the insecticide that kill homozygous-susceptible individuals. In this case, it is expected that resistance will develop more slowly because the heterozygote advantage is slight and it would take longer for sufficient numbers of heterozygotes to accumulate in the population to the point that they mate and produce some homozygous-resistant individuals. It is only when this happens that high-level resistance becomes apparent.

It has been noted that resistance to cypermethrin in the German cockroach is a commonly-occurring event in many field-collected populations (Atkinson et al. 1991, Cochran 1995a, 1996a, Scharf et al. 1995, Zhai and Robinson 1992), even after only limited exposure to cypermethrin. Resistance to this insecticide is inherited as an autosomal, monofactorial, incompletely-recessive trait in this insect (Ebbett and Cochran 1997). Because of these facts, a question of interest is whether the gene for resistance to cypermethrin is widespread in many German cockroach populations and at a frequency that allowed the rapid appearance of resistance to this insecticide.

I will present evidence here that the gene for resistance to cypermethrin is common in field-collected populations of the German cockroach. The implication is that it probably occurs at unusually high frequencies in unselected populations allowing the rapid evolution of detectable levels of resistance to cypermethrin.

MATERIALS & METHODS

The insects used in this study were from infested premises located in various parts of the continental USA and Puerto Rico, as indicated in Tables 1 and

2. Starter colonies were received and placed in culture in the laboratory. Typically, they were reared for I-2 generations to ensure sufficient numbers for insecticide testing. The year in which the cypermethrin tests were conducted on each strain is also shown in the tables. Rearing was conducted as previously described (Cochran 1979).

Cypermethrin-resistance testing followed the method of Cochran (1989). Briefly, the inside surfaces of 0.5-liter glass jars were coated with 1.5 nl/cm^2 (AI) of technical-grade cypermethrin (Zeneca, Wilmington, DE). A known number (7 or 10) of large nymphs were placed in each jar and their response was recorded over time. Three replicates of each strain were tested. In a few cases six replicates were used. The replicates were pooled and subjected to probit analysis (Cochran 1989). LT_{50} values, obtained in this manner, were used for comparison with corresponding values for the VPI-susceptible strain. A resistance ratio (RR) was calculated for each strain as follows: RR = LT_{50} of the test strain ÷ LT_{50} of the VPI strain. Testing was done at 21-23° C.

Gene frequency estimates were calculated as described by Cochran (1994c), based on the Hardy-Weinburg equilibrium expression (Falconer 1981). When resistance is recessive and is inherited monofactorially, as appears to be the case here (Ebbett and Cochran 1997), and one genotype can be clearly distinguished through toxicological testing, as shown for cypermethrin (Cochran 1994c, Ebbett and Cochran 1997), then gene frequency equals the square root of the fraction representing the percent of the insects surviving the test (e.g., 90% survival = 0.90: GF = the square root of 0.90 = 0.95).

RESULTS & DISCUSSION

In more than 100 cypermethrin tests with the VPI-susceptible strain, there were no survivors at 24 h. While much smaller numbers of known heterozygotes were available for testing, they also did not survive a similar challenge. Based on this information, it was assumed that any survivors of a 24 h exposure to cypermethrin at the concentration used here were homozygous resistant. It was also assumed that the inheritance mechanism, described above, is common in the populations tested. This assumption is supported by the results of tests using the cytochrome P_{450} mixed-function oxidase inhibitor, piperonyl butoxide, on many strains that were highly-resistant to cypermethrin (Cochran 1994d, 1997). In most cases, this inhibitor rendered resistant cockroaches completely susceptible.

The number of individuals tested per strain varied from 21 to 60. These are small numbers upon which to base gene-frequency estimates, but the tests were not specifically designed for that purpose. The fact that there were survivors in 57 of the 80 strains tested shows that the gene for cypermethrin resistance is common in this species.

Table 1. Field-collected strains of the German cockroach in which the gene frequency for the cypermethrin-resistance gene was too low to measure.

Strain	Origin	Year tested	Collection site
Boces Bakery	Nassau Co., NY	1993	Bakery
Grouverneur	New York, NY	1992	Closet
Frishman #6	New York, NY	1993	Restaurant
Creek Club	Locust Valley, NY	1993	Kitchen
Bellevue G	New York, NY	1992	Closet
NY Style Pizza	Fayetteville, NC	1992	Restaurant
Cutts	Fayetteville, NC	1992	House
Melvin	Warsaw, NC	1991	House
McIntere	Raleigh, NC	1994	House
King G-14	Raleigh, NC	1994	Apartment
King F-204	Raleigh, NC	1994	Apartment
King H-14	Raleigh, NC	1994	Apartment
King A-14	Raleigh, NC	1994	Apartment
Ping	Raleigh, NC	1994	House
Lowsaw	Clinton, NC	1994	House
Crawford	Clinton, NC	1994	House
Gary	Gary, IN	1992	Apartment
Muncie	Muncie, IN	1992	Apartment
Cooper	Jacksonville, FL	1989	Apartment
Bakery	Miami, FL	1993	Bakery
Hawthorne	Hawthorne, FL	1990	House
Navy #6	Norfolk, VA	1989	Ship
H-360	Hanover, MD	1989	Mess Hall

The 23 strains listed in Table 1 were those in which there were no survivors at 24 h, and the resistance ratios were typically between 1.0 and 2.0. If there had been 1 survivor in a sample size of 21 insects, the estimated gene frequency would have been 0.20. Thus, all that can be said of these strains is that they showed no resistance to cypermethrin, and the gene frequency for the cypermethrin-resistance gene was <0.20. The latter could vary from 0 to slightly less than 0.20, but a much larger sample size would be required to demonstrate a lower value. For example, 1 survivor in a sample size of 500 would produce a gene-frequency estimate of 0.04, which is still relatively high.

The data shown in Table 2 are for the 57 strains in which a gene-frequency estimate could be made, in spite of the small sample sizes. In these strains the estimated gene frequency varied from 0.20 to 0.99. When the estimate was

Table 2. Gene frequency estimates for the cypermethrin-resistance gene in 57 field-collected strains of the German cockroach

Strain	Origin	Year tested	GF[a]	RR[b]
Hara	Raleigh, NC	1994	0.20	1.2
King J-16	Raleigh, NC	1994	0.21	1.1
	Norfolk, VA	1989	0.22	2.6
Navy #5	Norfolk, VA	1989	0.22	2.4
Sellers	Kerr, NC	1994	0.22	1.1
York	Brenson, NC	1994	0.22	0.9
Williams	Harrells, NC	1992	0.22	1.1
Flushing	Flushing, NY	1992	0.22	2.3
HRDC	Washington, D.C.	1992	0.22	1.9
Bellevue D	New York, NY	1992	0.22	1.0
Pridgen	Salemburg, NC	1992	0.22	2.5
Rockville	Rockville, NY	1993	0.31	2.7
Godwin	Garland, NC	1992	0.31	3.1
Season S	Alexandria, VA	1989	0.31	4.0
Jacksonville	Jacksonville, FL	1989	0.31	3.0
Morris	Coats, NC	1994	0.31	0.9
Fryers	Nassau Co., NY	1993	0.37	1.4
Muttontown	Muttontown, NY	1993	0.38	2.9
Pruda	Elizabethtown, NC	1992	0.38	2.3
Anderson	Roseboro, NC	1992	0.38	1.2
Burbank	Burbank, CA	1992	0.38	1.7
Ft. Knox	Ft. Knox, KY	1989	0.38	2.2
Forest Green	Gainesville, FL	1991	0.38	3.3
Army-K-851	Ft. Knox, KY	1989	0.38	3.6
King L-11	Raleigh, NC	1994	0.42	2.7
Navy #1	Norfolk, VA	1989	0.44	2.7
T-164	Gainesville, FL	1990	0.53	6.6
From Bret	Long Island, NY	1993	0.58	23.0
King M-13	Raleigh, NC	1994	0.61	7.9
Ft. Myers	Ft Meyers, VA	1991	0.69	8.8
Newkirk	Kerr, NC	1994	0.79	>100
Gov't Bldg	New York, NY	1993	0.85	>85
Long Island	Long Island, NY	1991	0.9	>50
Las Palms	Miami, FL	1991	0.9	>50
King Q-305	Raleigh, NC	1994	0.94	>90
Bret F	New York, NY	1992	0.95	>90
Grouverneur K	New York, NY	1992	0.95	>90
Pizza Internat.	Miami, FL	1993	0.95	>65
Salisbury G	E. Meadows, NY	1993	0.95	>75
Forest	Forrest, MS	1994	0.95	>90
Syosset	Syosset, NY	1993	0.97	>65
Nassau Word	Nassau Co., NY	1993	0.98	>100
Puerto Rico	San Juan, PR	1993	0.98	>100
Runnaways	Miami, FL	1993	0.98	>100
Smithtown	Smithtown, NY	1993	0.98	>65
Toughkengnon	Toughkengnon, PA	1992	0.98	>75
New Opelika	Opelika, AL	1991	0.98	>50
Jones	Duplin Co., NC	1991	0.98	>50
Barksdale	Sampson Co., NC	1994	0.98	>90
Jackson	Jackson, MS	1994	0.99	>90
Villa Italia	Miami, FL	1993	0.99	>90
Far Rockaway	Far Rockaway, NY	1994	1.00	>85
Bret NY	New York, NY	1992	1.00	>60
Tien Hung	Miami, FL	1992	1.00	>60
Boston Sub	Miami, FL	1992	1.00	>60
Rogers	Jackson, MS	1993	1.00	>70
Bank	New York, NY	1993	1.00	>75

a. GF, gene frequency. See text for method of calculation
b. RR, resistance ratio. See text for method of calculation

between 0.20 and 0.44, the resistance ratios remained quite low with only a few exceptions. The latter can probably be explained on the basis the chance inclusion of a higher than expected number of resistant individuals in the test sample in relation to their frequency in the whole population, and/or to chance variations in the test results (Cochran 1996b). However, when gene-frequency estimates exceeded 0.50, resistance ratios rose sharply and when they exceeded 0.80 it was no longer possible to calculate resistance ratios because 50% mortality was never achieved. For these strains a resistance ratio of > some number is given depending on the LT_{50} for the susceptible strain in the corresponding test.

It is recognized that more than one resistance mechanism can occur in a given population (Cochran 1994a). There is evidence indicating the existence of a kdr-type of resistance mechanism in the German cockroach (Scott & Matsumura 1983, Cochran 1995b). However, based on the results presented here it is clear that at least one gene for cypermethrin resistance is common in many populations of German cockroaches. This is true both from the standpoints of number of strains tested and from their geographic origins. That gene apparently mediates the piperonyl butoxide sensitive resistance mechanism. Thus, it is not surprising that resistance to cypermethrin has arisen quickly in many naturally-occurring populations of this insect (Cochran 1995a, 1997).

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Sensitivity of cucurbit powdery mildew fungus to myclobutanil in research and commercial pumpkin fields

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INTRODUCTION

Myclobutanil (formulated as Nova 40W) was used commercially for controlling cucurbit powdery mildew on Long Island, NY, for the first time in 1998 through Section 18 (FIFRA Specific Emergency Exemption) registration. This systemic fungicide was used in combination with a contact protectant fungicide by most growers for fungicide resistance management. Control generally was excellent on upper surfaces of leaves but only moderate on lower (under) surfaces of leaves where myclobutanil probably was acting alone due to the difficulty of obtaining good spray coverage. Resistance is a concern with myclobutanil because it is a sterol demethylation inhibiting fungicide and thus it is in the same chemical class as triadimefon, which became ineffective due to resistance. Although triadimefon-resistant strains of the pathogen were shown previously to be less sensitive to myclobutanil than triadimefon-sensitive strains (McGrath et al. 1996), myclobutanil provided full-season control of powdery mildew when triadimefon (formulated with chlorothalonil as Omni) exhibited reduced efficacy due to resistance in the 1993 LI pumpkin powdery mildew fungicide evaluation experiment (McGrath and Staniszewska 1994). Powdery mildew severity on 2 Sep 93 on upper/lower leaf surfaces was 0%/0.3% for pumpkin sprayed twice with Nova at 2 oz/A (14-day interval) plus four times with Bravo 720 at 3 pt/A (7-day), 0%/14% for pumpkin sprayed twice with Omni (triadimefon plus chlorothalonil) at 4.25 pt/A (14-day) plus twice with Bravo 720 (14-day), and 40%/56% for nontreated pumpkin.

MATERIALS & METHODS

Isolates were collected from nontreated and myclobutanil-treated research plots at the LIHRL and from five commercial pumpkin fields in Suffolk County to determine whether reduced sensitivity of the pathogen to myclobutanil could account for powdery mildew development on lower leaf surfaces. Research plots had been sprayed weekly a total of five times with Nova at 2.5 oz/A plus Bravo Ultrex at 1.8 lb/A. The first application was made after mildew reached the IPM action threshold (McGrath 1996). Commercial fields had been sprayed one to three times. Fungicide sensitivity was determined using a leaf disk bioassay performed as described previously (McGrath et al. 1996).

RESULTS

Percentage of isolates able to tolerate 20 ppm myclobutanil was 53% in nontreated plots on 26 Aug and 56% in myclobutanil plus chlorothalonil-treated plots on 1 Oct. This was the concentration tolerated by most triadimefon-resistant strains tested in previous work. Similarly, 16 of 28 isolates (57%) collected from myclobutanil-treated commercial fields also tolerated 20 ppm. The percentage varied from 0% to 100% among these fields, however, this could be due

to the small sample sizes. Two of the 46 isolates tested (1 each from the myclobutanil-treated research field and a commercial field) were able to tolerate 80 ppm myclobutanil.

DISCUSSION

Considering that myclobutanil plus chlorothalonil was one of the most effective treatments examined in the pumpkin powdery mildew fungicide evaluation experiment and considering that there were not substantial differences in sensitivity to myclobutanil between the isolates tested from the research field and commercial fields (when combined), it appears to be more likely that the moderate control achieved was due to number and timing of applications and high disease pressure rather than fungicide resistance. The Section 18 request date may not have provided growers enough time to obtain and apply Nova before powdery mildew was at too high a level to be controlled effectively. Powdery mildew was more severe in 1998 than in 1997 in nontreated control plots in the LI pumpkin powdery mildew fungicide evaluation experiments indicating that disease pressure was higher in 1998 (severity on upper/lower leaf surfaces was 15%/53% on 25 Sep 97 and 57%/85% on 18 Sep 98). The same variety (Harvest moon) was used in both experiments. However, fewer isolates from plots treated with myclobutanil plus chlorothalonil were able to tolerate 20 ppm myclobutanil in 1993 than in 1998 (10% versus 56%, respectively) and disease control was better in 1993.

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Sympossia Summaries

Resistance Management in Reality: A Multidisciplinary Look at Progress, Limitations, and Lessons from the Field. 1998 Annual Meeting of the Entomological Society of America. November 9-11, 1998. Las Vegas, NV.

MANAGING INSECTICIDE RESISTANCE IN THE COLORADO POTATO BEETLE

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Colorado potato beetle, Leptinotarsa decemlineata (Say) (CPB), is the most serious insect pest of potatoes throughout the northern U.S. and Canada and in Europe and the former Soviet Union. Defoliation can be 100% and yield losses can be near total. Colorado potato beetle has developed resistance to all available synthetic insecticides, including organophosphates, organochlorines, carbamates, and pyrethroids, in many parts of the world (Bishop and Grafius 1996, Forgash 1981, Lagunes-Tejeda 1991). Directly because of severe insecticide resistance, CPB cost the Michigan potato industry $8.2 to $14.4 million per year (up to 20% of total crop value) in control costs and yield losses from 1991 through 1994 (Grafius 1997). Control costs and losses were greatly reduced throughout the northeastern U. S. with the registration and widespread use of imidacloprid (Bayer Corp., Kansas City MO) in 1995. However, low levels of resistance began to appear in Michigan in 1996 (Grafius and Bishop 1997) and Long Island CPB were 20-fold more tolerant to imidacloprid than most populations in 1995 (Olsen et al. 1996); over 100 fold resistance was present in CPB from Long Island New York by 1997 (Zhao et al. 1998).

CPB has a wide range of hosts in the Solanaceae family, including potatoes, tomatoes, eggplant, nightshade spp., buffalo bur, etc. Plants in this family commonly contain toxic alkaloids. Some of these alkaloids are cholinesterase inhibitors like organophosphate and carbamate insecticides (Wierenga and Hollingworth 1992) and CPB's ability to resist or detoxify these plant compounds likely facilitates its ability to adapt to insecticides (Wierenga and Hollingworth 1993). Selection for resistance in CPB is intensive because it has relatively few wild hosts in potato-growing areas, both adults and larvae feed on the crop, and alternative controls like biological control and resistant varieties have not been available (until the introduction of Bacillus thuringiensis potatoes in 1995).

CPB adults overwinter in the soil in the potato field or in adjacent field borders; the common practice of growing potatoes following potatoes, without crop rotation, or rotation of crops only across a road or fence line provides an ideal environment for CPB (Casagrande 1987). CPB adults can fly moderately long distances, but generally don't disperse out of the crop if food is available (Caprio and Grafius 1990). Thus, each potato field may contain its own population of CPB, selected for resistance to the insecticides commonly used by that grower (Grafius 1995). Periodically, unusually warm weather in the spring, before the potato crop is up, or in the

fall, after harvest, may stimulate long distance dispersal (Caprio and Grafius 1990). Dispersal of dominant resistant genotypes rapidly spreads resistance.

The following are generalities about insecticide resistance in CPB, that relate to effectiveness of resistance management techniques. [There are, of course exceptions to these generalities.] Insecticide resistance in CPB is often conferred by single dominant or semi-dominant genes (Ioannidis et al. 1992). CPB can express multiple mechanisms of resistance including esterase- and oxidase-based detoxification, target site insensitivity, knock-down resistance, penetration, and sequestration (Bishop and Grafius 1996, Clark and Argentine 1994). Sequestration of plant toxins is likely why CPB are seldom preyed upon by birds and express typical vertebrate-warning colors of orange and black (larvae) or yellow and black (adults). Recent results of Olsen and Dively (Univ. Maryland, unpublished data) indicate that excretion of toxins may also be a resistance mechanism. Individual beetles may carry several resistance genes, with little or no fitness cost (Argentine et al. 1989) and resistance tends to be stable in the absence of selection. Initial resistance gene frequency for resistance to carbofuran was 2:10,000 (Ioannidis et al. 1992) - relatively high for an insect that can have populations of over 100,000 per hectare.

Resistance management tactics. Techniques for use of insecticides to manage insecticide resistance include: (1) high doses, (2) alternation of insecticides, (3) tank mixes, (4) maximum rate/maximum amount use, (5) use of the synergist piperonyl butoxide, and (6) refugia. Each of these has specific assumptions that must be met for the tactic to be effective.

(1) The high dose strategy is the use of doses high enough to kill resistant heterozygotes. This has to begin very early in the resistance process when almost all individuals are susceptible homozygotes or heterozygotes. It assumes that heterozygotes are susceptible to a high dose of insecticide (resistance is recessively inherited). A second assumption is that initial resistance gene frequency is low. This strategy also assumes that a high dose can be applied in the field uniformly in time and space - lower doses are not present which could kill homozygotes but not heterozygotes (thus creating a group of interbreeding heterozygotes that will produce a significant number of resistant homozygotes in the next generation).

Unfortunately, we know that resistance to many insecticides in CPB is inherited as a dominant or semidominant factor. Heterozygotes may be 20 to 50-fold resistant to the insecticide compared to the susceptible homozygote. Also, with widespread cross resistance between previous and new insecticides, initial resistance gene frequency may not be low. Finally, high doses always decay to low doses in the field and areas of the crop canopy shielded from the insecticide spray may receive low doses, even though the application was at a high dose.

(2) Alternation of insecticides is the use of insecticides from different chemical groups, alternating each generation or each year. Alternation of insecticides assumes that there is no cross resistance between the two insecticides and that resistance is unstable and decreases in the absence of selection. Alternation of insecticides could be especially effective where there was negative cross resistance between insecticides; i.e., resistance to one product increases sensitivity to the other product. Alternation must be over a time interval of several generations; if individual insects are treated with two different products during the same generation, the situation is actually a case of mixing insecticides.

Again, insecticide resistance in CPB generally does not meet the assumptions required for alternation of insecticides to be successful. Resistance is often stable for long periods of time and significant cross resistance occurs between insecticides in different groups. Ioannidis et al. (1991) suggested possible negative cross resistance between organophosphates and pyrethroids, but generally CPB can exhibit resistance to multiple insecticides with little or no apparent fitness cost.

(3) Insecticide mixes are used with the expectation that no single individual carries resistance to both insecticides. Again, this assumption is false for CPB; individuals are capable of carrying resistance to three or more different classes of insecticides (Ioannidis et al. 1991) and there is little or no fitness cost.

(4) Maximum rate/maximum amount is a tactic that uses frequent, high doses of tank-mixed insecticides. Like the mix tactic, this tactic assumes that no one individual can carry resistance to two or several insecticides and that resistance increases gradually - if it is disrupted in an early stage it will not increase. As we now know, individual CPB can carry resistance to several insecticides and very high levels of resistance can be imparted by a single gene (e.g., carbofuran resistance, Ioannidis et al. 1992) and no possible field application rate could achieve control of resistant individuals. Furthermore, the most intense selection pressure selects for resistance the most rapidly.

(5) The synergist, piperonyl butoxide, can be used to block microsomal oxidase enzymes and has been used effectively in the field to control CPB. Microsomal oxidase enzymes are commonly used for resistance to pyrethroids, organophosphates and carbamates (Bishop and Grafius 1996). However, other mechanisms may also be used against these insecticides. Our experiences in Michigan indicate that within 2 - 3 years CPB adapt to this selection pressure and resistance mechanisms other than oxidase enzymes become common.

(6) Refugia are important for management of resistance to insecticides for many insect pests. Assumptions for effectiveness of a refuge are that (a) resistance must be recessive or semi-recessive, (b) large numbers of susceptible insects must be produced in the refuge, and (c) high levels of gene flow must exist between refugia and crop sites. As indicated above, resistance in CPB is most often dominant in inheritance, refu-

gia are generally small compared to the potato crop, with small numbers of CPB and gene flow between crop and wild hosts is limited. An exception to this situation may be in parts of the western U.S. where potatoes are commonly rotated with small grain over long distances and volunteer potatoes in the rotation crop make up an untreated host for CPB.

Use of the above strategies may slow the development of resistance, especially by optimizing refugia and alternating insecticides from different insecticide classes. However, in general, management of resistance to insecticides in CPB may be impossible if insecticides are the primary mortality factor. To maintain the effectiveness of an insecticide, we must reduce reliance on it for control by introducing non-chemical controls. Integrated pest management programs for CPB must be adopted, especially including crop rotation, resistant varieties. Until transgenic or traditionally bred CPB resistant potato varieties are commonly available, crop rotation will continue to be the single most important factor for managing CPB.

Whalon et al. (1993) have already demonstrated that CPB can adapt to Bacillus thuringiensis. Chaconine, a host plant resistance factor present in Solanum chacoense, could be introduced into potatoes, but it is a cholinesterase inhibitor and insecticide-resistant CPB are more tolerant to it than are insecticide-susceptible CPB (Wierenga and Hollingworth 1992). Once resistant potato varieties are widely planted, then we begin the task of managing the adaptation of this remarkable insect to the new varieties.

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MANAGING INSECT RESISTANCE TO BT CROPS: ARE THE RULES FOR RESISTANCE MANAGEMENT ANY DIFFERENT?

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The introduction of crops genetically altered to express insect toxins produced by the bacterium, Bacillus thuringiensis (Bt), has provided a significant technological advancement in insect control. For many crops such as Bt corn, the technology provides a high level of efficacy at relatively low cost to the grower. Grower response to Bt technology has for the most part been positive. Although Bt corn represents one of the larger U.S. markets, production with many other U.S. crops has also been significant. In 1998, an estimated 15 million acres of Bt corn was planted in the U.S. (approx. 20% market share), with about 30 million estimated for 2000. Also in 1998, Bt cotton, potato and sweet corn were planted to ca. 2 million, 35,000 and 20,000 acres, respectively. Balanced against several advantages of Bt crops, the primary concern of many scientists is the possibility that targeted pests could develop resistance to Bt if market penetration is high. Because of the potential for market penetration, and the desire by many to conserve Bt as long as possible, much is at stake and the debate has been dynamic among stakeholders.

A quick answer to the question posed at the ESA symposium is a resounding yes! The rules have clearly changed and there are many reasons. The process of change has also resulted in new ways for industry, research and extension personnel and government agencies to in-

teract. The purpose of this presentation was to review why the rules have changed, what impact a new risk assessment process for RM has already had, and finally, suggestions for continued dialog among all stakeholders in the development and implementation of RM plans. The development of an RM plan for Bt corn is used as one example of how the rules have changed for Bt crops.

Why have the rules changed? Given the many new advantages of this technology (e.g., full-season insect control, reduced scouting and monitoring costs, reduction in conventional foliar insecticide use), much of the motivation for a new process for RM was the desire to be more proactive. Moreover, there are many unique aspects of Bt, including: naturally occurring soil borne pathogen, selectivity to targeted hosts, compatibility with most natural enemies, and an excellent safety record. Bt can also be viewed as a natural resource to be conserved long-term. With these many advantages contrasted against the backdrop of history of 500+ insect/mite species developing resistance to conventional insecticides, it is clear that a more proactive RM approach is desirable. Specifically, unlike the U.S. registration paradigm of the past 40 years, RM plans should now be explicitly developed and required on the product label prior to registration.

An immediate, positive outcome of the need to develop more proactive RM plans, has been the development of new ways in which industry scientists, business managers and registration personnel now interact with university scientists and cooperative extension personnel. There is now an unprecedented need for all parties to interact, to generate and share new data on a regular basis to meet new demands by US-EPA for RM plans. In response to the EPA requirement for RM plans, seed companies have been very active in supporting and soliciting proposals for new research that will be useful for developing science-based RM plans. Seed companies have also been active in disseminating RM concepts to growers.

With Bt corn as one example, there has been considerable dialog among stakeholders for the past 4 ½ years. Much of the dialog occurs because we are essentially attempting to apply the science currently available, under uncertainty, to manage a resource long-term. As with any management decision under uncertainty, there are 2 critical components of the decision making process that should be kept separate. Later, they can be combined for final policy recommendations. Decision analysis methods allows one to partition the 2 components of risk assessment, i.e., a) uncertainty/risk (via probability theory), and b) multiple/conflicting values and objectives (addressed via utility theory). The U.S. process with Bt corn has benefited from distinguishing each component during most discussions. However, the process can be very difficult as different interpretations or perceptions of either component may exist. Perceptions clearly vary depending on scientist and stakeholder experience or affiliation. A recent quote by Sir Robert May reflects this tension from a scientist's perspective, …"scientific knowledge is built up, not by maximizing certainty or striving awkwardly for consensus, but by minimizing uncertainty." That is, scientists are more comfortable with the first component of risk assessment, and accepting a certain level of uncertainty (e.g., experimental error) in the conduct of experiments, or with models of complex systems. However, scientists are generally not in a position to understand or assign value to multiple or conflicting objectives of diverse stakeholders, such as small vs. large growers. Although this quote may reflect the more usual role for scientists, an added willingness to dialog with other stakeholders to reach consensus for RM plans is needed, albeit awkward, for both scientists, industry representatives, growers, and all stakeholders involved with a particular Bt crop.

Given all current data available for European corn borer biology and ecology, as well as input from several RM models, and a particular risk of high rates of local inbreeding within corn borer populations, the North Central Regional Research Committee on Stalk-boring Lepidoptera (NC-205; Ostlie et al. 1997), with input from industry, developed a proactive RM plan for Bt corn. This plan was the result of 2+ years of discussion and development of new research. For Bt-corn the RM plan included the recommendation that 20-30% of the corn borer population not be exposed to Bt corn (or Bt foliar products), to maintain an adequate ratio of susceptibles to possible resistant individuals. In areas where there are few reliable alternative hosts for corn borer, the practical implication is that at least 20-30% of the corn acreage, on a given farm, should be non-Bt corn (40% non-Bt corn if insecticides are anticipated). The results of this plan, as well as recent results for Bt corn and Bt potato, were discussed at a US-EPA subpanel review all RM plans in February 1998. After consideration of new research results obtained during the 1998 field season, the NC-205 committee reaffirmed these recommendations. The discussion and new research continues, and will likely continue for Bt corn after full registrations are granted. As recommendations continue to evolve, it is also clear that comprehensive, collaborative (industry and academia) resistance monitoring efforts be established for all Bt crops.

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FUNGICIDE RESISTANCE MANAGEMENT: PROGRESS, LIMITATIONS, AND LESSONS

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Progress has been made since the 1970s when the first cases of fungicide resistance occurred and resulted in problems with control. Most importantly, resistance is now recognized as a problem that needs to be dealt with before it occurs. Thus a proactive rather than reactive approach is now taken. Risk assessment has become a routine part of fungicide development. Management is part of standard product stewardship. Warnings and management guidelines are on labels when at-risk fungicides are registered.

Management strategies have been developed and tested. These include using an integrated disease management program with as many practices as possible to reduce the need for fungicides. At-risk single-site fungicides should only be used when needed most, which is usually early in an epidemic, with multisite fungicides applied at other times, thereby minimizing pathogen exposure. At-risk fungicides should be used in alternations or mixtures with other fungicides and not used alone repeatedly or used curatively. Most companion fungicides have been multi-site fungicides which have a low risk of inducing resistance. At-risk fungicides with different mode of action have the advantage as companions of similar coverage and postinfection action due to systemicity. Mixtures have generally been shown to be more effective than alternations. Coformulated mixtures ensure compliance, however, solo products offer freedom of choice. Low (reduced) fungicide rates slow resistance development with major-gene resistance by lowering selection pressure. They also keep mixture programs economical and avoid increases in pesticide use. With polygenic resistance, high rates eliminate individuals with low resistance. In practice, doses reaching target organisms vary greatly over space and time. The general recommendation is to keep to manufacturer's recommended dose rates and intervals. Fungicide performance and pathogen sensitivity to the fungicide should be monitored. The useful life of some fungicides has been extended by stopping use when resistance develops, monitoring the pathogen population, and reintroducing the fungicide on a restricted spray schedule when resistance levels fall. Typically a fungicide is not as effective after resistance develops, therefore it is prudent to implement a resistance management program when a fungicide is registered.

Several lessons have been learned. Resistance risk can be difficult to predict. Persistence of resistant strains determines future usefulness of a fungicide. However, reduced fitness is not correlated with resistance. Selection for resistance and selection for fitness are two different processes. Effectiveness of a strategy can vary with pathogen and over time. Many factors can affect resistance development. Resistance management is more effective when implemented before resistance is detected. Applying fungicides that are ineffective due to resistance is not only a waste of resources, it has been shown to increase severity of some diseases.

Many limitations exist. Resistance is difficult to predict. Necessary tools may be lacking. Systemic companion fungicides that do not have resistance problems may not be available. Several multisite companion fungicides have had their registrations cancelled or are under review through FQPA. Registering new fungicides is a lengthy process. Resistance management program for a new fungicide cannot be evaluated. Growers are often more concerned about economics and efficacy than resistance management. Implementation may be difficult when the resistance management program is more expensive or less effective than using the at-risk fungicide alone fullseason. With highly mobile pathogens, successful management may require regional implementation. Resistance management programs rarely have been enforceable. Managing resistance with fullrate mixtures is at odds with public desire to reduce pesticide use. IPM tactics that delay applications until after disease detection and extend spray intervals until disease-favorable conditions have occurred are in conflict with the accepted resistance management tactics of preventive treatments and maintenance recommended intervals.

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