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THE INSECTICIDE-RESISTANCE PROBLEM

A Review of Developments in 1956 and 1957

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SYNOPSIS

The author reviews the growth of the insecticide-resistance problem throughout the world during the period between July 1956 and November 1957, and the developments in research on the subject during the same period.

Three new resistant species have been discovered—*Anopheles subpictus*, *Chrysomyia putoria* and *Rhipicephalus sanguineus*—and eight new types of resistance in already resistant species have been observed. Moreover, the geographical area covered by certain resistant insect populations has considerably increased.

The research accomplishments during the period under review include: systems of detecting resistance in the field by standard test methods; confirmation of two distinct types of resistance to chlorinated-hydrocarbon insecticides in mosquitos and bed-bugs as well as in houseflies; evidence that DDT-resistance in the housefly, *Anopheles sundaicus* and *Aëdes aegypti* is due mainly to a single genetic factor associated with the ability to dehydrochlorinate DDT, and that dieldrin-resistance of *Anopheles gambiae* also derives from a single factor present even in untouched populations; a fuller understanding of the physiological mechanism of BHC-resistance in the housefly; and demonstration that selection pressure from organo-phosphorus compounds induces resistance to themselves and to chlorinated-hydrocarbon insecticides.

In July 1956 a WHO Expert Committee on Insecticides noted that the problem of insecticide-resistance was growing more rapidly than the necessary measures to deal with it, and called for an international co-operative programme of research for which WHO was the appropriate body to assume leadership. In November 1957 an Expert Committee on Insect Resistance and Vector Control met and reviewed the accomplishments of this co-operative programme of research against the background of the continuing spread of resistance among insects of public health importance.

The following sets forth the subject-matter for that review. It is divided into two parts: (*a*) the growth of the resistance problem throughout the world, and (*b*) developments in research on the problem of resistance. The material reviewed is restricted to discoveries made and papers published during the period between the meetings of the two committees.

Growth of the Resistance Problem throughout the World

During the period under review the number of resistant species has increased by three (*Anopheles subpictus ; Chrysomyia putoria ; Rhipicephalus sanguineus*), and the number of new types of resistance in already resistant species has increased by eight (2 to DDT; 4 to BHC and dieldrin; 2 to organo-phosphorus compounds). The geographical area covered by resistant populations has considerably increased in 11 out of the 18 species considered in the review.

Musca domestica

The housefly developed resistance to diazinon in the Province of Latina, Italy (Sacca, 1957) four years after this insecticide came into use. The highest resistance levels were found just outside the town of Latina itself, and were 60 times the normal. A similar resistance to diazinon has developed in the Canton of Valais, Switzerland, as reported orally by R. Wiesmann. Diazinon-resistance was first reported from Denmark in 1955 (Keiding, 1957) where it was associated with resistance to parathion and Resitox.

Resistance to malathion has been reported from Orlando, Fla. (Labrecque & Wilson, 1957), where it had first been observed in 1955 (Lindquist, 1957). Resistance levels for contact malathion in Orlando and Tampa were, respectively, 37 and 23 times the normal. Control failures occurred in 1956 with malathion baits at Savannah, Ga., the resistance level on topical application being 2.4 times the normal (Kilpatrick & Schoof, 1958).

Remarkable increases in the housefly population after the application of dieldrin to latrine superstructures have been reported from Georgia (Kilpatrick & Schoof, 1956). Increases with dieldrin were about 200 times over the normal density; BHC and chlordane showed this effect in less degree, while DDT caused no increase. That houseflies became very conspicuous after domestic applications of dieldrin was reported from Liberia, Nepal, Saudi Arabia, Japan, Kenya and Tanganyika.

With the publication of instances of resistance to DDT and BHC in Japan (Byers, Wheeler & Blakeslee, 1956) and to DDT and dieldrin in Kenya (McMahon, 1957), resistance in houseflies has now been reported from all regions except the mainland of China.

Pediculus corporis

The body louse has shown itself to be capable of developing resistance to BHC as well as to DDT. Use of the standard WHO kit for field testing has revealed BHC-resistance in many places in Japan and at two localities in south-eastern Cape Province, Union of South Africa (Wright & Brown, 1957). An expert consultant visited Africa and confirmed the existence

of BHC-resistance at Freetown, Sierra Leone, and St. Louis, Senegal (C. N. Smith—unpublished working document WHO/Insecticides/62). Control failures with BHC occurred in three villages of north-eastern Iran in 1957 (McLintock, Zeini & Djanbakhsh, 1958).

Anopheles gambiae

Dieldrin-resistance of this important malaria vector has been discovered in the western Sokoto region of Northern Nigeria (Elliott & Ramakrishna, 1956). It was also present at Kano, but not at Lagos, Ibadan or Ilaro in the southern forest belt of Nigeria (Elliott & Armstrong, 1957). In 1957, dieldrin-resistance was discovered near Bobo Dioulasso, Haute Volta, nearly all adults surviving exposure to 4% dieldrin papers (J. Hamon —unpublished report to WHO, 8 September 1957). Late in the year it was discovered that 91-100% of the *A. gambiae* in the Bahn area of up-country Liberia were dieldrin-resistant (H. F. Schoof—personal communication to WHO, 16 October 1957).

Anopheles sundaicus

DDT-resistance was discovered in 1956 at Soerabaya in eastern Java (C. Y. Chow & A. Soeroto—unpublished report to WHO, 18 July 1957), to add to the three pockets of resistance already known along the north shore (Chow & Soeparmo, 1956). In 1957, DDT-resistance was found in coastal Burma, near Akyab (by F. Delphin) and Kyaukpyu (by S. Sundararaman).

Anopheles stephensi

A certain degree of resistance to DDT by larvae developing in wells has been reported from Erode, south India (Rajagopalan, Vedamanikkam & Ramoo, 1956). DDT-resistance of both larvae and adults was discovered in 1957 at Basrah and Nassriya, Iraq (G. Gramiccia et al.—unpublished working document Mal/Inform/29). At the same time, DDT-resistance was found to be prevalent at Abadan, Iran, and at points extending from Shushtar through Ram Hormuz and Kazerun down to Bandar Abbas (C. Mofidi et al.—unpublished working document Mal/Inform/29). This DDT-resistance had first been discovered on the opposite side of the Persian Gulf at Dhahran in 1955 (G. Davidson—unpublished report to WHO, December 1957).

Anopheles quadrimaculatus

The discovery of dieldrin-resistance of this species in Bolivar County, Miss., was published in 1956 (Mathis et al.). In 1957 it was discovered to be

present at Mississippi Hills, 200 km to the north-west (K.D. Quarterman—personal communication to WHO, 27 July 1957).

Anopheles subpictus

Pronounced DDT-resistance was discovered in 1956 at Jwalaheri and Nanglisahidan, Uttar Pradesh, India (Sharma & Krishnamurthy, 1957a). This strain was, however, still susceptible to gamma-BHC and dieldrin (Sharma & Krishnamurthy, 1957b). Dieldrin-resistance in *A. subpictus* was reported in 1957 from Tjirebon, Java (G. Davidson—personal communication to WHO, 8 November 1957).

Anopheles sacharovi

Abnormally high tolerances of dieldrin as well as DDT were found in Greek DDT-resistant strains in the Peloponnese in 1956 (Hadjinicolaou, 1956) and in the Thessalonika district in 1957 (G. D. Belios—personal communication to WHO, 20 September 1957). Pronounced dieldrin-resistance of larvae, extending to chlordane but only slightly to DDT, was reported from Arkadia, Epirus and the Ionian Islands (Belios & Fameliaris, 1956).

Aëdes aegypti

DDT-resistance in the yellow-fever mosquito on the island of Trinidad has been reported in the scientific literature (Gilkes, Kellett & Gillette, 1956). Control failures occurred in Ciudad Trujillo, Dominican Republic (Pan American Sanitary Bureau, 1956) and at Cucuta, Colombia, where tests showed the larval tolerance of DDT to be 32 times the normal (Pan American Sanitary Bureau, 1957). DDT-resistance approximately 30 and 300 times the normal, respectively, was discovered at Carupano and Caracas, Venezuela (J. Blazquez—unpublished working document WHO/Insecticides/68).

Culex fatigans

Larval DDT-resistance amounting to 100 times the normal level has been described from Taiwan (Liu, 1958). DDT-tolerance and a more pronounced resistance to chlordane, dieldrin and gamma-BHC has been reported from Okinawa (Gentry & Hubert, 1957). Dieldrin-tolerance following rapidly upon DDT-resistance has been observed at Singapore (Collins, 1956). Dieldrin-resistance without any change in DDT-susceptibility was found in both adults and larvae at Ubai, Malaya, after five residual applications of dieldrin (Wharton, 1958). Strong DDT-resistance has been demonstrated at Puerto Cabello, Venezuela (J. Blazquez—unpublished working document WHO/Insecticides/68) and considerable BHC-resistance at Cayenne, French Guiana (Floch & Fauran, 1958).

Culex tarsalis

This vector of encephalitis was found to have developed resistance to malathion, but not to parathion, in Fresno County, Calif.; tolerance levels for malathion were 30 times the normal for larvae and 100 times the normal for adults (Gjullin & Isaak, 1957). DDT-resistance has developed in larvae at Oakridge, Oreg., the level being approximately 200 times the normal (Eddy, Hopkins & Robbins, 1958).

Cimex lectularius

Resistance in bed-bugs is a serious matter because it lessens the acceptability of domestic spraying for vector control. DDT-resistant populations are now of frequent occurrence in Israel (N. Gratz—personal communication to WHO, 16 January 1957) and are present in Teheran, Iran, where they are susceptible to BHC (C. Mofidi & B. Samimi—mimeographed report from Institute of Parasitology and Malariology, Teheran, May 1956). DDT-resistant bed-bugs have been introduced into Cayenne, French Guiana, with immigrant labourers from the island of Santa Lucia (Floch, 1955). BHC-resistance became highly developed in 1956 at Beth Shaan, Israel, and moderately so at Tiberias (Cwilich, Mer & Meron, 1957).

Cimex hemipterus

DDT-resistance in this tropical bed-bug has been reported from Feng-Shan in southern Taiwan (Chen, Tseng & Pletsch, 1956). It has also developed in many parts of Bombay State, particularly in the warm regions below the Western Ghats (Ramachandra Rao & Halgeri, 1956). Control failures with DDT have become notable in Hong Kong and Singapore (J. R. Busvine—Working Paper No. 8, WHO Expert Committee on Insect Resistance and Vector Control). Dieldrin-resistant bed-bugs have appeared in a malaria eradication area at South Pare, Tanganyika; however, they remain susceptible to DDT (A. Smith—unpublished report to East African Institute for Malaria and Insect-Borne Diseases, 7 October 1957).

Blattella germanica

Resistance in the German cockroach aggravates the public health problem of this food-contaminating species, as well as destroying confidence in domestic insecticide applications. The chlordane-resistance which had developed in the Gulf Coast region of the USA was assessed quantitatively and found to be 20 times the normal in three cities of the southern states (Keller et al., 1956) and 10-25 times the normal in at least 20 strains from the south-eastern states (DuChanois, 1956). During 1956, chlordane-resistance became prevalent in the north-eastern states and in the Chicago area (P. J. Spear—personal communication to WHO, 5 November 1957). Many

of the resistant populations in the southern states showed resistance also to pyrethrins of an intensity 3-30 times the normal (Keller, Clark & Lofgren, 1956).

Chrysomyia putoria

The report has appeared (W. Bervoets, P. Bruaux, A. Lebrun & M. A. Ruzette—mimeographed report from Institut Marcel Wanson, 1957) of resistance to organo-phosphorus compounds by this pest blowfly of human faeces at Léopoldville, Belgian Congo. After BHC-resistance in 1949, diazinon-resistance developed in 1954, followed by malathion-resistance in 1955.

Acarina

Since the development of resistance in ticks would be serious in the case of vectors of human disease, instances of it occurring in animal parasites are of considerable concern. A report has appeared of chlordane-resistance in the brown dog-tick, *Rhipicephalus sanguineus*, in New Jersey, USA (Hansens, 1956). DDT-resistance appeared in 1956 in the blue tick, *Boophilus decoloratus*, in the East London district of the Union of South Africa, where BHC-resistance had developed in 1948 and arsenic-resistance in 1938 (Whitehead, 1956). DDT-resistance was also reported in 1956 to have developed in the cattle tick, *B. microplus*, in the Rockhampton district of Queensland, Australia, where BHC-resistance had developed by 1952 and arsenic-resistance by 1937.

Developments in Research on the Problem of Resistance

A great advance has been made in the systematization of knowledge on the genetical origin of insecticide-resistance by Crow (1957) in North America and Milani (1956a, 1957 [1]) in Europe. It is clear that resistant strains have derived from normal populations from selection of the less susceptible genotypes by the action of the insecticide in killing the more susceptible individuals. This Darwinian explanation is accepted by Busvine (1957) in his recent review of the present status of resistance in insects of public health importance.

The normal susceptibility levels have been determined for a great number of mosquito species by the method of Busvine & Nash, thus enabling resistance to be detected as soon as the test shows a loss of susceptibility. The data obtained from various parts of the world have been assembled by Busvine (1956) and by the Malaria Section of WHO (1957).

[1] An English translation is available as Supplement C, Information Circular on the Resistance Problem, WHO

The wide application of a WHO standard test method has resulted in a picture of resistance in body lice based on data from 37 countries (Wright & Brown, 1957). A survey of housefly resistance involving 42 localities has recently been completed in Israel (Ascher, 1957a).

Systematic investigations on the ability of houseflies to develop resistance to the organo-phosphorus insecticides from selection pressure in the laboratory have been made at Riverside, Calif. (R. B. March, R. L. Metcalf & L. L. Lewallen—unpublished working document WHO/Insecticides/59), at Orlando, Fla. and Corvallis, Oreg. (Lindquist, 1957), at s'Graveland, Netherlands (Meltzer, 1956) and at Basle, Switzerland (J. R. Geigy S. A., Pest Control Department—document Diaz./Res.fl./e). As they developed resistance to the selecting agent, these strains also showed a cross-resistance, generally of less degree, to other organo-phosphorus compounds, and a very high cross-resistance to chlorinated-hydrocarbon insecticides.

Some understanding of the physiological mechanism of BHC-resistance in the housefly has been achieved during the period under review. The more BHC-resistant the strain, the less it absorbs the BHC isomers through the cuticle and the more it metabolizes what it absorbs (Oppenoorth, 1956); this has been demonstrated to occur with gamma-BHC (Bradbury & Standen, 1956). The first product of metabolism is evidently pentachlorocyclohexene, although no DDT-dehydrochlorinase enzyme is present; subsequently this compound is metabolized further (Sternburg & Kearns, 1956). As many as 11 different water-soluble metabolites have been detected in resistant flies some time after their exposure to gamma-BHC (Bradbury, 1957).

Further proof has been obtained of the role of the detoxifying enzyme DDT-dehydrochlorinase as the main factor imparting DDT-resistance to houseflies. Individuals with a longer larval life develop more DDT-dehydrochlorinase and are more DDT-resistant (Moorefield & Kearns, 1957). The inheritance of DDT-resistance in the F_2 and back-crosses between resistant and susceptible strains was exactly paralleled by the inheritance of DDT-dehydrochlorinase (Lovell & Kearns, 1956). The DDT-dehydrochlorinase activity in the central nervous system is evidently high enough to protect it against DDT in the haemolymph and so produce a resistant nerve (Miyake, Kearns & Lipke, 1957). Steps have been taken to purify the enzyme, the activity having been concentrated at least 120 times (Moorefield, 1956).

Resistant housefly strains at Basle, however, showed abnormal DDT-metabolic activity whether they had been produced by DDT, BHC, dieldrin or diazinon selection pressure, and this increase was no more than twice the activity in the normal strains (Reiff, 1956). Resistant flies of any strain contained more total lipoid and tarsal lipoid than normal, which being of lower melting-point is capable of dissolving more DDT and thus of more effectively protecting the sensitive tissues (Wiesmann & Reiff, 1956). Lipoid

droplets and vacuoles become visible in the tormogen and tarsal hypodermal cells of resistant flies; on the other hand, normal flies secrete twice as much pulvillar grease as resistant flies, thus increasing the tarsal uptake of DDT (Wiesmann, 1957).

A housefly strain genetically homozygous for DDT-resistance has been obtained after many years of selection from the normal NAIDM strain, and the results of crossing it with a homogeneous susceptible strain has demonstrated the DDT-resistance to be due to a single dominant gene (Lichtwardt, 1956). Crosses of a DDT-resistant strain from Latina with a normal strain also showed that the resistance was due to a single gene allele, having partial penetrance for kill but no penetrance—and therefore being recessive—for knockdown (Milani, 1956b). This gene, termed *kdr*, is chromosomally linked with the gene *bwb* for brown body and the gene *dv* for divergent wings (Milani & Travaglino, 1957).

The dieldrin-resistance of *Anopheles gambiae* in Northern Nigeria extends to aldrin, chlordane and gamma-BHC, but not to DDT (Davidson, 1956a). In crosses with a normal strain, the F_1 hybrid is intermediate in resistance; when this is back-crossed with either parental strain, two phenotypes appear in 50 : 50 ratio. These results indicate dieldrin-resistance to be due to a genetic factor showing no dominance; the homozygotes can survive 4% dieldrin and the heterozygotes 0.4% dieldrin, according to the Busvine & Nash test (Davidson, 1956b). Field tests with these diagnostic concentrations in areas as yet untouched by dieldrin have shown that heterozygotes already pre-exist in 0.04-6.0% of the population in Northern Nigeria (J. A. Armstrong, C. D. Ramsdale & V. Ramakrishna—unpublished working document WHO/Mal/182 (WHO/Insecticides/52)), and in 5-12% of the population in Haute Volta (J. Hamon—personal communication to WHO, 26 October 1957).

The DDT-resistance of *A. sundaicus* from eastern Java extends to methoxychlor and DDD, but not to dieldrin or gamma-BHC. In crosses between resistant and susceptible strains, the F_1 hybrid is susceptible, and 25% of the F_2 are resistant: back-crosses of the F_1 with the resistant strain produce 50% resistant offspring, but with the susceptible they produce no resistant offspring. These data indicate that the DDT-resistance derives from a single recessive factor (Davidson, 1958b). The resistant homozygotes contain much DDT-dehydrochlorinase activity, the susceptibles none (C. W. Kearns—unpublished report of WHO consultant, 24 July 1957).

The DDT-resistance of *Aëdes aegypti* from Trinidad extends to DDD, but not to dieldrin or gamma-BHC (Fay, 1958). The resistance of three different strains to the various DDT analogues is directly proportional to their ease of dehydrochlorination by alkali (Busvine & Coker, 1958). The Trinidad strain converts much DDT to DDE *in vivo*, while a normal strain converts none (Brown & Perry, 1956); but no DDT-dehydrochlorinase activity could be shown *in vitro* by methods which demonstrated it in

resistant houseflies (Brown, 1956). In crosses between the Trinidad strain and a susceptible strain, the genetic segregation shown in the F_2, and in the offspring of a back-cross of the F_1 with the susceptible strain, indicate that a single factor is mainly concerned in the DDT-resistance (Coker, 1957).

A contribution to the understanding of other causes of resistance has been made by Hoskins & Gordon (1956), who introduced the term " vigor tolerance " to cover instances resulting from extra vigour of the strain rather than from any specific defence mechanisms. Such vigour tolerance could account for the slightly enhanced cross-tolerances shown between one specific type of insecticide-resistance and another. It would appear likely that its inheritance is polygenic, that it involves no change in the slope of the dosage-mortality regression line, and that reversion to susceptibility will occur on release from insecticide pressure. Vigour tolerance could well be involved in the moderate decreases in susceptibility levels of *Anopheles* observed during or after control programmes (WHO Malaria Section, 1957) or even in the high increase of tolerance in *A. sacharovi*, as suggested by Davidson (1958a).

There is good evidence that increased DDT-tolerance in *A. atroparvus* is associated with an inversion in regions 44 to 48 of the sinistral arm of the third chromosome. Larvae homozygous or heterozygous for this inversion survive a level of DDT exposure that kills the homozygotes with the non-inverted normal arrangement (D'Alessandro, Frizzi & Mariani, 1956). Selection pressure from DDT can greatly increase the frequency of this inversion, the heterozygotes occurring in greater proportion than expected, with the over-all DDT-tolerance of the population increasing (D'Alessandro, Frizzi & Mariani, 1958). Since the inverted length of chromosome is inherited *en bloc*, it would simulate a single gene. It is of interest that the dieldrin-resistant *A. gambiae* of Northern Nigeria show a great variety of inversions in high frequency (Frizzi & Holstein, 1956).

The research findings during the period just past confirm the distinctness of two specific types of resistance to chlorinated-hydrocarbon insecticides—namely, resistance to DDT and its analogues (type 1) and resistance to dieldrin and other cyclodiene derivatives extending to gamma-BHC (type 2); this separation was first discovered in houseflies and now has been shown to hold good for *Anopheles*, *Aëdes* and *Culex* mosquitos and for bed-bugs. Type 1 resistance has been traced to a single genetic origin in *Anopheles sundaicus* and *Aëdes aegypti*, and in both cases it has been associated with the detoxification of DDT by a dehydrochlorinating enzyme process, as in houseflies. Type 2 resistance has been shown to originate in a single genetic factor in *Anopheles gambiae*, but its mechanism is at present unknown. During the period under review, it has become abundantly clear that insecticide-resistant strains are developed by Darwinian selection of individuals carrying the genetic pre-adaptations for the specific defence mechanisms. The insecticide cannot change the insects; it can only select survivors by

killing the others. There is one exception, however, which has arisen in connexion with the effect of dieldrin in multiplying housefly populations. Afifi & Knutson (1956) have reported that the offspring of flies that have survived dieldrin intoxication lay 70% more eggs than normal flies. This instance of an induced change that is passed on to the offspring is of the greatest interest and practical importance.

The most promising lead for countermeasures against the resistance problem lies in the possibility that there are chemical compounds which are more toxic to housefly strains that are resistant to the normal insecticides than to those that are not. An impure sample of diisopropyl tetrachloroethylphosphate was found to be almost three times as toxic to the DDT-resistant Orlando-Beltsville strain as to the normal NAIDM strain (Mitlin, Babers & Barthel, 1956). When a DDT-resistant strain containing 5% susceptible individuals was submitted to selection pressure from this material for three generations, it was transformed into a DDT-susceptible strain (C. W. Kearns—personal communication to WHO, 27 July 1957). It was discovered by Ascher that bromoacetic acid showed greater knock-down properties against resistant than against susceptible strains both of *M. d. domestica* and of *M. d. vicina*, and that many of its esters showed the same property (Ascher, 1957b). Cetyl bromoacetate, the ester with the longest residual action, was twice as effective as normal against strains developed from selection pressure by chlordane, DDT and other chlorinated hydrocarbons, diazinon or chloroacetic acid (Ascher, 1958).

RÉSUMÉ

Le développement de la résistance des insectes aux insecticides observé dans le monde de juillet 1956 à novembre 1957 fait l'objet de cet article. L'auteur y expose en outre le résultat des recherches sur l'origine et le mécanisme des divers types de résistance.

Au cours de la dite période, on a découvert trois nouvelles espèces résistantes (*Anopheles subpictus, Chrysomya putoria, Ripicephalus sanguineus*). Huit espèces qui résistaient déjà à un insecticide donné étaient devenues résistantes à des composés d'autres groupes: 2 au DDT, 4 à l'HCH et à la dieldrine et 2 aux composés organo-phosphorés. Au surplus, l'aire de dispersion des populations résistantes s'est considérablement étendue dans le cas de 11 espèces examinées. Les niveaux de sensibilité normale ont été établis pour un grand nombre d'espèces, ce qui permet de déceler la résistance sitôt qu'elle apparaît.

Les connaissances sur l'origine et le mécanisme de la résistance ont beaucoup progressé. Chez les mouches, la résistance aux composés organo-phosphorés implique une résistance croisée aux insecticides à base d'hydrocarbures chlorés. Plus une souche de mouches est résistante au HCH, moins elle en absorbe par la cuticule et plus facilement elle métabolise celui qu'elle absorbe. Quant au DDT, il se vérifie qu'une enzyme détoxicante, la déchlorase du DDT, est le facteur qui confère aux mouches une résistance au DDT. Sa production est liée à un facteur héréditaire. Cette enzyme protège le système nerveux central. Des recherches sont en cours pour la concentrer et la purifier. D'autres espèces de mouches résistantes contiennent plus de lipoïdes totaux et de lipoïdes tarsiens que les mouches sensibles, ce qui permet la dissolution d'une plus grande quantité de DDT. Les croisements effectués au sein d'une souche de mouches homozygotes pour la résistance

au DDT, obtenue après plusieurs années de sélection, ont montré que la résistance était liée à un gène unique, dominant. La résistance d'*Anopheles gambiae* dans la Nigeria du Nord s'étend à l'aldrine, au chlordane et au HCH gamma, mais non au DDT. Elle est liée à un allèle qui n'est pas dominant. Les homozygotes supportent 4% de dieldrine et les hétérozygotes 0,4%. De tels hétérozygotes existent normalement dans 0,04-6,0% de la population des mouches de la Nigeria du Nord, dans des régions où aucune pulvérisation de dieldrine n'a encore été effectuée. Chez *Aëdes aegypti*, la résistance au DDT semble également liée à un seul facteur génétique.

Certains auteurs ont introduit la notion d'une résistance due à la vitalité de l'insecte (« vigor tolerance ») pour expliquer certains aspects de la résistance qui ne semblent pas relever d'un mécanisme spécifique de défense. Les recherches récentes ont également confirmé l'existence de deux types de résistance aux hydrocarbures chlorés: *a*) résistance au DDT et à ses analogues observée chez *Anopheles*, *Aëdes*, *Culex*, et chez des punaises; *b*) résistance à la dieldrine et à d'autres dérivés du cyclodiène, s'étendant à l'HCH, observée chez *A. gambiae*. La résistance de type *a* est sous la dépendance d'un seul gène chez *Anopheles sundaicus* et *Aëdes aegypti*, et, chez ces deux insectes a pu être attribuée à une détoxication, comme chez les mouches.

Les recherches ont montré à l'évidence que la résistance est la conséquence d'une sélection darwinienne d'individus porteurs de pré-adaptations génétiques correspondant aux mécanismes de défense. Les insecticides ne provoquent pas de changement à proprement parler chez les insectes. Ils opèrent une sélection en détruisant les individus sensibles. Une exception cependant est à relever. On a constaté que les descendants d'une certaine souche de mouches ayant survécu au traitement à la dieldrine pondaient 70% de plus d'œufs que les mouches normales. Ce fait est important du point de vue théorique et pratique.

Des observations récentes laissent entrevoir un moyen de contrecarrer la résistance des insectes aux insecticides. On a constaté que certaines souches résistantes au DDT étaient plus sensibles à certains autres composés chimiques — en l'occurrence le tétrachloroéthylphosphate de diisopropyle non purifié — que les souches sensibles (au DDT). Une souche de mouches résistantes au DDT comprenant 5% d'individus sensibles, soumise à la pression sélective du composé chimique précité pendant trois générations, est redevenue sensible au DDT. Des observations analogues ont été faites avec l'acide bromacétique et certains de ses dérivés.

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