PAPERS AND PROCEEDINGS OF THE 22ND ANNUAL MEETING

of the AMERICAN MOSQUITO CONTROL ASSOCIATION

Co-sponsored by The Communicable Disease Center, Public Health Service, U. S. Dept. Health, Education and Welfare, and the Chatham County Mosquito Control Commission, Savannah, Georgia

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Part II

SYMPOSIUM ON INSECTICIDE RESISTANCE IN MOSQUITOES

INTRODUCTORY REMARKS

DON W. MICKS

University of Texas Medical Branch, Galveston, Texas

May I take this opportunity to welcome each of you to this symposium on insecticide resistance. As you read through your programs, you undoubtedly took note of the fact that we have been fortunate in collecting together such a group of experts on various facets of the insecticideresistance problem, particularly as it relates to mosquitoes. This is a far-ranging symposium in that it extends from California on the West to Florida on the East and up to the northern states. It will deal not only with the biochemical and genetic aspects of the problem of resistance to chlorinated hydrocarbon and organophosphorus insecticides, but also with chemical counter-measures as well.

It seems appropriate this morning for us to recall that almost twenty years have elapsed since insecticide-resistance in mosquitoes was first discovered. Mosna, in Italy, in 1947, noted that only one year after house-spraying had been initiated in Latina, *Culex molestus* adults were resting on DDT-treated walls. Even more spectacular was his finding that these mosquitoes could survive 3 days of continuous contact with DDT deposits. The serious-

ness of this phenomenon was demonstrated more forcefully when resistance was observed for the first time in anopheline mosquitoes. Following on the heels of a highly successful, nation-wide malaria control program in Greece, which had begun in 1946, *Anopheles sacharovi*, to a large extent, developed outside resting habits. By 1950, adults were observed resting in houses on DDT-sprayed walls, and control failures were evident in several areas by 1952.

About the same time Mosna noted DDT-resistance in Culex molestus, workers in the United States, a number of whom are here this morning, found likewise that DDT was failing to control Culex tarsalis, Aedes nigromaculis and Aedes dorsalis in California, and Aedes sollicitans and Aedes taeniorhynchus in Florida. With these early observations it was clear that the floodgates of resistance were open and that the problem could be expected to extend to other mosquitoes in these and other areas where insecticides such as DDT would be used for control.

During the past decade, beginning with 1956 when 17 species of mosquitoes had

developed resistance to at least one group of insecticides, the magnitude of the problem has increased enormously. By 1960, this number of resistant species had doubled and at the present time has more than tripled that original number.

Whereas these figures are impressive, it should be remembered that the actual and potential threat posed by insecticideresistance in mosquitoes is greater than it appears on the surface even without extending to any additional species. Many of us have been prone to think of this phenomenon as one thing which occurs in fairly circumscribed areas where populations of vectors and people are not very mobile. An examination of the record of the past five years shows that such populations are anything but stable. Rapidly increasing urbanization throughout the world has involved an extensive segment of the human population which in turn has been accompanied by major extensions of the two most highly domesticated species, Aedes aegypti and Culex fatigans (quinquefasciatus).

These important disease vectors, especially the latter, have already shown themselves capable of developing multiple resistance. Furthermore, their spread into new urban foci has already created serious new problems which are sure to worsen before they get better. For example, the introduction of *A. aegypti* into many new areas of Southeast Asia such as Manila, Bangkok, Singapore and Penang has been associated with recent epidemics of hemorrhagic fever with fatality rates as high as 10

percent. This disease is caused by a modified dengue virus, transmitted by *A. aegypti*, and has now become a major public health problem in Southeast Asia.

Another evidence of vector dynamics and accompanying problems of insecticide-resistance relates to *Culex fatigans*. This species, which is resistant to DDT and dieldrin, presently comprises about 90 percent of the mosquito population of Rangoon. In 1945, the incidence of filariasis there was about one percent at a time when the population was 300,000. Today, Rangoon has a population of approximately 700,000, with a filariasis rate ranging from 8 to 15 percent. With increasing urbanization and slum areas which follow, the vector is becoming more abundant.

In tropical Africa, *C. fatigans*, until relatively recently, was scarce in some cities. Now it is present in large numbers in almost all population centers and is extending into the rural areas of several states. This development coupled with the widespread distribution of Bancroftian filariasis in tropical Africa may already be responsible for what appear to be new foci of the disease.

These examples are intended only to emphasize the increasing challenge of insecticide resistance, which as our symposium summarizer, Dr. Brown, has stated it, "is as many-sided as biology itself." We now have the privilege of hearing of some of the more recent developments in some of these "sides" and something of what the future may hold.

TITLES OF PAPERS FOR 1967 ANNUAL MEETING

The official call for papers will probably already be in the hands of AMCA members by the time this notice is read. This is a reminder that **the deadline**, as given to the editor, **is December 15**, and the program chairman is T. D. Mulhern, 5545 E. Shields Ave., Fresno, California. The meeting will be a joint meeting with the California Mosquito Control Association, at the Sheraton Palace Hotel, San Francisco, California, Feb. 5–6–7–8, 1967.