EFFECTS OF SUBLETHAL DOSAGES OF INSECTICIDES ON *CULEX QUINQUEFASCIATUS*[1]

L. L. ROBERT[2,3] AND J. K. OLSON[2]

ABSTRACT. Groups of *Culex quinquefasciatus* were exposed as fourth instar larvae to sublethal concentrations (0.1 LC_{50} and LC_{50}) of malathion, methoprene, propoxur or resmethrin. Females exposed as larvae to an LC_{50} level of methoprene had reduced wing length and longevity. Egg production was reduced by 50% and 39% in those mosquitoes exposed to LC_{50} levels of malathion or methoprene, respectively. In contrast, egg production and egg raft size increased following treatment with 0.1 LC_{50} levels of malathion or methoprene. Females exposed as larvae to methoprene laid 30% fewer eggs per raft, and egg hatching decreased 36% compared with controls.

Females exposed as larvae to LC_{50} levels of malathion or methoprene laid shorter eggs than controls. The proportion of females in the adult population was reduced following exposure to either propoxur or resmethrin, and increased following exposure to malathion. Time to pupation and time of emergence of the adult populations were increased following exposure to most of the insecticidal treatments. These results indicated that a single, sublethal exposure to certain insecticides had a significant effect on mosquito reproduction.

INTRODUCTION

The acute toxicity of insecticides is used to produce mortality in target insect populations inside a given area. Treatment of a target species with an insecticide may be uneven due to factors such as vegetation or wind, with some individuals therefore receiving sublethal dosages of insecticides. Sublethal dosages of insecticides may affect insect populations by: 1) affecting survival, 2) affecting the reproductive ability of individuals or 3) affecting the genetic makeup of future generations (Moriarty 1969).

Before World War II it was known that arsenical and botanical insecticides had sublethal effects on insects (Hoskins 1940). At the time, these sublethal effects were viewed as inconsequential. Subsequent work with DDT, botanicals and cyclodienes has been reviewed by Ascher (1964) and Moriarty (1969). Other insecticides have been shown to affect a number of reproductive parameters in mosquitoes, including fecundity (Firstenberg and Sutherland 1981, Kelada et al. 1981, Kerdpibule et al. 1981, Liu et al. 1986), egg hatching (DeCoursey and Webster 1952, DeCoursey et al. 1953), immature development time (Wijeyaratne[4]), adult longevity (Duncan 1963) and adult size (Wijeyaratne[4], Kelada et al. 1981).

These reports indicate that in certain cases, sublethal effects of pesticides may be as important as the acute toxic effect in reducing numbers of insect pests. Knowledge of these sublethal effects of pesticides on arthropods is therefore important to the decision making processes in integrated pest management programs.

The purpose of this study was to investigate the effects of selected insecticides LC_{50} and 0.1 LC_{50} levels) upon the reproductive potential of *Culex quinquefasciatus* Say, an important urban vector of disease agents in the southern United States. The following parameters were measured to determine the effects of sublethal concentrations of insecticides on the insecticide-exposed and F_1 generations; adult longevity, adult wing length (a measure of adult body size), number of eggs per raft, egg length, number of eggs produced per female, egg hatching, sex ratio of adults and developmental time.

MATERIALS AND METHODS

The UTMB strain of *Cx. quinquefasciatus*, colonized at the University of Texas Medical Branch, Galveston, was used in this study. An insecticide-free colony of this strain has been maintained at Texas A&M University for over 10 years. It was determined to be susceptible to the insecticides used in this study.

Mosquitoes were reared at 27°C, 75–85% RH with a 14:10 hr light/dark photoperiod. Two egg rafts (ca. 200 eggs per raft) were placed in white enamel pans (36 × 23 × 5 cm) containing 1.0 liter of deionized water. Larvae hatching from these eggs were fed a diet consisting of lab chow, lactalbumin and brewer's yeast (1:1:1 ratio). Food was added quantitatively (Gerberg et al. 1969) starting on the day after the eggs were placed into the pans. Pupae were transferred to a beaker of water and placed in a wood and screen cage (50 cm). The adults emerged and fed *ad libitum* on 10% sucrose solution. The blood

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[2] Department of Entomology, Texas A&M University, College Station, TX 77843.

[3] Present Address: Department of Entomology, Walter Reed Army Institute of Research, Washington, DC 20307-5100.

[4] Wijeyaratne, P. M. 1976. Effects of sub-lethal larvicide exposure on population and flight characteristics of *Culex pipiens quinquefasciatus*. Ph.D. Dissertation. Univ. of Texas Health Science Center at Houston. 245 pp.

meal source consisted of á restrained chick placed in the cage 3 days after emergence.

The following insecticides were used: 1) malathion (95.9%) (American Cyanamid Co., Wayne, NJ); 2) propoxur (99.0%) (Mobay Chemical Corp., Kansas City, MO); 3) resmethrin (86.8%) (Penick-Bio UCLAF Corp., Lyndhurst, NJ); and 4) methoprene (89.25%) (Zoecon Corp., Dallas, TX). Acetone was used as a solvent to dilute all chemicals to the appropriate concentrations.

Immatures were exposed to the various insecticides as early fourth instar larvae. Controls for each experimental group were treated with acetone and handled in the same manner as the insecticide exposed groups. Fifty larvae were placed in 100 ml of deionized water in a 355-ml disposable plastic cup. Those larvae treated with malathion, resmethrin or propoxur were exposed for a 24-hr period without food. After exposure, the larvae were removed from the insecticide solution, rinsed, placed in fresh water and fed larval diet. Larvae exposed to methoprene were kept in the insecticidal solution until adult emergence with food added after the first 24-hr period. This procedure was followed because mature fourth instar larvae are most susceptible to juvenile hormone mimics (Spielman and Skaff 1967) and exposure during this period is critical.

Three days after emergence, replicates from each insecticide treatment consisting of 3 groups of 10 males and 10 females were placed in wood and screen cages (30 × 22 × 22 cm). Females were given the opportunity to feed on a restrained chick. Egg rafts were collected daily. The procedure was repeated after 8 days to allow for the completion of a second gonotrophic cycle.

Analysis of variance was performed using the ANOVA and GLM procedures, depending on whether the experiment involved a balanced or unbalanced design (SAS 1985). The data from the egg hatching studies, expressed as percentages, were transformed using the arcsin angular transformation to normalize the distribution before testing by ANOVA (Sokal and Rohlf 1981). Individual means were compared using least significant difference (LSD). This procedure was used because the comparison-wise error rate from each pairwise comparison was considered to be more important than the experiment-wise error rate (Jones 1984). This allows all possible comparisons between all experimental groups.

RESULTS AND DISCUSSION

The insecticides used in this study varied in their effectiveness in killing early fourth instar larvae of *Cx. quinquefasciatus* (Table 1). Comparison of the 3 insecticides which cause larval mortality indicated resmethrin was the most toxic (LC_{50} = 4.3 ppb) to larvae, followed by malathion (LC_{50} = 35 ppb) and propoxur (LC_{50} = 287 ppb). These LC_{50} values are generally consistent with those previously reported (Wijeyaratne[4], El-Khatib and Georghiou 1985).

The highest concentration of methoprene used was not lethal to larvae but was highly effective in preventing adult emergence, as evidenced by the low LC_{50} (0.1 ppb). The LC_{50} value for methoprene was similar to that reported earlier for *Cx. quinquefasciatus* (Schaefer and Wilder 1972).

Egg production was significantly reduced ($P < 0.01$) during the first gonotrophic cycle by

Table 1. Mean LC_{50} (ppb), LC_{90} (ppb) and slope values for *Culex quinquefasciatus* larvae exposed to insecticides, serially diluted with acetone in 100 ml of deionized water.*

Chemical	LC_{50}	LC_{90}	Slope
malathion	35.0	51.0	3.6
methoprene	0.1	0.5	2.3
propoxur	287.0	439.0	6.6
resmethrin	4.3	7.0	5.9

* Average of 3 consistent replicates.

Table 2. Mean (±SE) number of eggs per female and eggs per raft for *Culex quinquefasciatus* exposed as larvae to insecticides.

		Number of eggs per female		
G.C.[1]	Insecticide	Control group	0.1 LC_{50} group	LC_{50} group
1	malathion	132 (15)	141 (11)	66 (19)*
1	methoprene	183 (15)	194 (25)	111 (19)*
1	propoxur	153 (9)	144 (14)	168 (16)
1	resmethrin	146 (12)	136 (2)	148 (4)
2	malathion	128 (7)	88 (7)*	94 (5)*
2	methoprene	203 (11)	161 (25)*	143 (5)**
2	propoxur	198 (29)	161 (23)	173 (7)
2	resmethrin	124 (6)	130 (5)	117 (10)
		Eggs per raft		
G.C.[1]	Insecticide	Control group	0.1 LC_{50} group	LC_{50} group
1	malathion	144 (8)	156 (8)	152 (7)
1	methoprene	215 (10)	227 (12)	150 (8)*
1	propoxur	204 (5)	202 (5)	193 (4)
1	resmethrin	152 (8)	160 (7)	174 (6)
2	malathion	165 (8)	157 (8)	155 (7)
2	methoprene	200 (7)	205 (5)	155 (8)*
2	propoxur	198 (8)	194 (6)	179 (7)
2	resmethrin	151 (8)	156 (7)	171 (5)

* Value is significantly different from control, $P < 0.01$.

** Value is significantly different from control, $P < 0.001$.

[1] Indicates first or second gonotrophic cycle.

50% and 40%, and in the second gonotrophic cycle by 30% and 40% following exposure to the LC_{50} treatment of malathion or methoprene, respectively (Table 2). In contrast, the 0.1 LC_{50} treatments of malathion or methoprene each had a slightly stimulatory effect on egg production in the first gonotrophic cycle. Both concentrations of malathion and methoprene resulted in significantly reduced ($P < 0.01$) egg production during the second gonotrophic cycle. However, none of these effects were observed in the F_1 generation.

Other investigators have reported reduced egg production following larval exposure to sublethal concentrations of insecticides. Fecundity was reduced in *Cx. quinquefasciatus* following larval exposure to selecting concentrations of dimilin, methoprene, dichlorvos, malathion (Wijeyaratne[4]) or temephos (Ferrari and Georghiou 1981). Various insecticides also reduced egg production in *Aedes aegypti* (Linn.) (Firstenberg and Sutherland 1981) and *Cx. pipiens* Linn. (Gaaboub and Dawood 1974).

The slight increase in egg production during the first gonotrophic cycle following exposure to 0.1 LC_{50} treatments of either malathion or methoprene is also consistent with some studies using low (causing little or no mortality) insecticidal dosages. *Culex pipiens* females that survived various concentrations of insecticides which inhibited the emergence of 25% of adults had an increase in basal follicle numbers (Saleh and Aly 1987, Saleh 1988). Sutherland et al. (1967) indicated that maximum sublethal dosages of DDT, dieldrin and malathion resulted in significantly increased basal follicle number in female *Ae. aegypti*. In contrast, Firstenberg and Sutherland (1981) reported decreased fecundity in *Ae. aegypti* populations following exposure to 0.1 LC_{50} dosages of malathion or temephos.

Conflicting results as to the effects of insecticides on mosquito egg production appear to be related to the dosages used. Our data (and that of most of the studies cited) indicate that females which are exposed as larvae to selecting doses (causing some mortality in the population) of insecticides have reduced egg production and females exposed as larvae to nonselecting doses (causing little or no mortality) may have either increased or decreased egg production (Table 3). Although some discrepancies occur, selecting doses of insecticides generally increase egg production in adult mosquitoes.

The LC_{50} treatment of methoprene resulted in egg rafts that were significantly smaller ($P < 0.01$) than those produced by controls (Table 2). However, the 0.1 LC_{50} treatment resulted in slightly more eggs per raft than controls during

Table 3. Summary of previous research on egg production in adult mosquitoes following larval exposure to selecting and nonselecting insecticidal doses.

Chemical	Dose	Species	Effect*	Reference
		Selecting doses		
Altosid	LC_{50-90}	*Ae. aegypti*	−EP	Naqvi et al. 1976
methoprene	LC_{50}	*Ae. aegypti*	−EP	Firstenberg and Sutherland 1981
Abate				
Penfluron	0.1–1.0 ppb	*An. stephensi*	−EP	Saxena and Kaushik 1986
Puryltriazine	5–10 ppm			
DDT	20 ppm	*Cx. quinquefasciatus*	−EP	Kerdpibule et al. 1981
temephos	LC_5, LC_{50}	*Cx. quinquefasciatus*	−EP	Ferrari and Georghiou 1981
malathion	LC_{50}	*Cx. quinquefasciatus*	−EP	Wejeyaratne[4]
dimilin				
dichlorvos				
methoprene				
DDT	LC_{90}	*Cx. pipiens*	−EP	Gaaboub and Dawood 1974
Altosid	0.1–1.0 ppb	*Cx. pipiens*	+FN	Gaaboub et al. 1981
dimilin				
IGRs	IC_{25}	*Cx. pipiens*	+FN	Saleh and Aly 1987
chlorpyrifos	IC_{25}	*Cx. pipiens*	+FN	Saleh 1988
sumithion				
		Nonselecting doses		
DDT	0.1 ppm	*Ae. aegypti*	+EP	Havertz and Curtin 1967
6 IGRs	sublethal	*Cx. pipiens*	+EP	Kelada et al. 1981
methoprene	0.1 LC_{50}	*Ae. aegypti*	−EP	Firstenberg and Sutherland 1981
Abate				
temephos	0.01 ppm	*Cx. quinquefasciatus*	−EP	Ferrari and Georghiou 1981
DDT, dieldrin, malathion	sublethal	*Ae. aegypti*	+FN	Sutherland et al. 1967

* EP, egg production; FN, follicle number; −, decrease; +, increase.

both gonotrophic cycles. Wijeyaratne[4] also reported that female *Cx. quinquefasciatus* surviving exposure to LC_{50} levels of dichlorvos, dimilin, malathion or methoprene consistently laid fewer eggs per raft than controls. When exposed to doses of 20 ppm DDT, *Cx. quinquefasciatus* produced fewer eggs per raft for 49 consecutive generations (Kerdpibule et al. 1981). Similar results were reported for *Cx. pipiens* exposed as larvae to 6 different IGRs (Kelada et al. 1981).

The LC_{50} treatment of methoprene resulted in a significant reduction ($P < 0.01$) in egg hatching (Table 4); however, egg hatching was not affected in the progeny of the untreated F_1 offspring. This result is in contrast with previous studies that report methoprene treatments have no effect on egg hatching in *Cx. pipiens* (Kelada et al. 1981) or *Ae. aegypti* (Firstenberg and Sutherland 1981).

Egg hatching was not affected by larval exposure to malathion, propoxur or resmethrin. This is consistent with the results of previous studies on cyclodiene exposure of *Anopheles quadrimaculatus* Say (DeCoursey et al. 1953) and *Ae. aegypti* (Duncan 1963). In addition, it has been reported that malathion exposure of larvae does not result in reduced egg hatching of *Cx. pipiens* (Gaaboub and Dawood 1974) or *Ae. aegypti* (Firstenberg and Sutherland 1981).

Egg rafts produced by females exposed to the LC_{50} treatment of methoprene had a different appearance than did control rafts. The eggs in these rafts were not in orderly rows or as closely packed as the eggs in control rafts (Fig. 1). In addition, many of the eggs were laid singly on the surface of the water. Eggs were also generally lighter in color and had a streaked appearance. Upon dissection, these eggs contained apparently fully-developed embryos; indicating that embryogenesis was not inhibited by the insecticidal exposure. We hypothesize that the LC_{50} treatment of methoprene resulted in a disruption in the way the eggs are held together (i.e., the waxy coating of the eggs) in orderly, tight rafts. This was simulated by individually separating eggs from control egg rafts containing fully developed embryos. These eggs were placed horizontally on the surface of the water. Egg hatching was reduced by 50%, suggesting that the eggs must remain upright in the raft for normal hatching to occur.

Adults exposed as larvae to either malathion or methoprene produced significantly shorter ($P < 0.001$) eggs than did controls during the first 2 gonotrophic cycles (Table 5). The greatest reductions in egg length occurred during the first gonotrophic cycle. Although several studies have

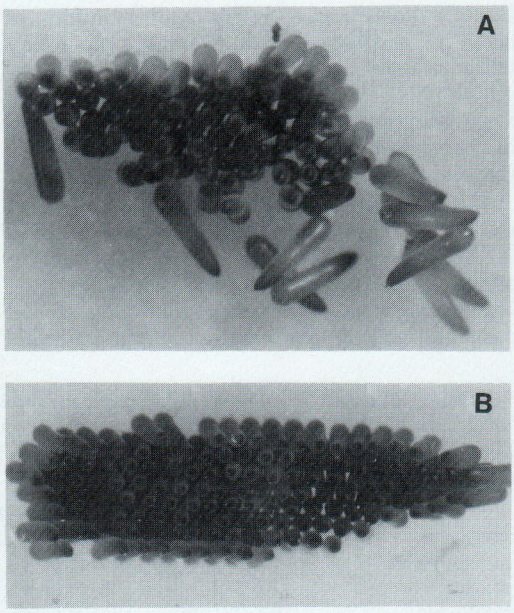

Fig. 1. Photomicrographs of egg rafts produced by *Culex quinquefasciatus* females exposed as larvae to: A) an LC_{50} level of methoprene, and B) acetone only.

Table 4. Mean (±SE) percentage egg hatch of eggs laid by *Culex quinquefasciatus* females exposed as larvae to insecticides.

G.C.[1]	Insecticide	Percentage egg hatch (%) Control group	0.1 LC_{50} group	LC_{50} group
1	malathion	95.9 (1.9)	93.9 (2.5)	95.5 (1.1)
1	methoprene	95.6 (0.8)	94.0 (0.9)	60.8 (1.9)*
1	propoxur	92.4 (3.2)	83.0 (4.1)	80.0 (3.5)
1	resmethrin	92.3 (2.0)	92.4 (2.3)	94.2 (1.2)
2	malathion	93.9 (0.9)	88.4 (6.3)	93.6 (1.6)
2	methoprene	91.3 (2.9)	88.5 (9.3)	65.1 (7.8)*
2	propoxur	88.9 (1.1)	90.2 (1.2)	89.9 (1.8)
2	resmethrin	91.5 (0.6)	90.6 (3.6)	89.6 (2.1)

* Value is significantly different from control, $P < 0.01$.
[1] Indicates first or second gonotrophic cycle.

Table 5. Mean (±SE) length of eggs from *Culex quinquefasciatus* females exposed as larvae to insecticides.

G.C.[1]	Insecticide	Mean egg length (mm)		
		Control group	0.1 LC_{50} group	LC_{50} group
1	malathion	0.71 (0.03)	0.70 (0.04)	0.66 (0.03)**
1	methoprene	0.72 (0.02)	0.71 (0.03)	0.67 (0.03)**
1	propoxur	0.71 (0.02)	0.71 (0.02)	0.70 (0.02)
1	resmetrin	0.72 (0.03)	0.70 (0.03)	0.70 (0.03)
2	malathion	0.73 (0.03)	0.71 (0.03)	0.69 (0.02)**
2	methoprene	0.69 (0.01)	0.68 (0.02)	0.67 (0.01)*
2	propoxur	0.70 (0.02)	0.70 (0.01)	0.70 (0.02)
2	resmethrin	0.69 (0.02)	0.69 (0.02)	0.69 (0.02)

* Value is significantly different from control, $P < 0.01$.
** Value is significantly different from control, $P < 0.001$.
[1] Indicates first or second gonotrophic cycle.

Table 6. Mean (±SE) adult wing length and longevity of *Culex quinquefasciatus* exposed as larvae to insecticides.

Sex	Insecticide	Wing length (mm)		
		Control group	0.1 LC_{50} group	LC_{50} group
M	malathion	2.29 (0.03)	2.31 (0.03)	2.37 (0.05)
M	methoprene	2.61 (0.02)	2.71 (0.03)*	2.59 (0.02)
M	propoxur	2.65 (0.01)	2.66 (0.01)	2.63 (0.03)
M	resmethrin	2.42 (0.06)	2.36 (0.04)	2.36 (0.02)
F	malathion	2.87 (0.03)	2.85 (0.03)	2.82 (0.05)
F	methoprene	3.19 (0.03)	3.22 (0.03)	3.10 (0.03)*
F	propoxur	3.04 (0.01)	3.04 (0.03)	3.05 (0.01)
F	resmethrin	2.84 (0.06)	2.77 (0.08)	2.84 (0.02)
		Adult longevity[1]		
Sex	Insecticide	Control group	0.1 LC_{50} group	LC_{50} group
M	malathion	23.0 (3.0)	22.3 (0.7)	21.0 (1.2)
M	methoprene	26.3 (2.3)	20.3 (1.8)*	16.3 (2.8)*
M	propoxur	21.0 (2.5)	26.3 (4.5)	21.7 (2.2)
M	resmethrin	18.3 (1.3)	19.3 (0.9)	20.0 (2.1)
F	malathion	28.7 (2.7)	29.6 (3.2)	29.0 (1.0)
F	methoprene	28.0 (1.0)	25.0 (3.1)	16.0 (4.4)*
F	propoxur	28.7 (4.8)	30.7 (1.5)	31.7 (2.0)
F	resmethrin	22.0 (1.0)	25.7 (3.5)	24.3 (3.0)

* Value is significantly different from control, $P < 0.01$.
[1] Expressed as LT_{50}, the number of days to 50% mortality.

reported detrimental insecticidal effects on insect ovarioles and ovariole development (Zaghloul and Brown 1968, Judson and de Lumen 1976, Saleh 1988), none reported any effect on egg length.

Adult wing length was influenced as a result of exposure to methoprene (Table 6). Males exposed to the 0.1 LC_{50} treatment of methoprene had significantly longer wings ($P < 0.01$) than controls. Females exposed to the LC_{50} treatment of methoprene had significantly shorter wings ($P < 0.01$). In contrast, Wijeyaratne[4] reported that larval treatments with methoprene had no effect on wing length. However, Wijeyaratne[4] did find that dimilin, dichlorvos and malathion treatments cause reduced wing length in females. Conversely, Kelada et al. (1981) reported that LC_{50} treatments of 6 IGRs, including methoprene, caused increased wing length in female *Cx. pipiens*. They also found that the LC_{10} treat-

Table 7. Mean (±SE) days to pupation and adult emergence of *Culex quinquefasciatus* exposed as larvae to insecticides.

Chemical	Dose	Pupation time[1]	Emergence time[2]	
			Males	Females
Control	0	0.7 (0.06)	2.3 (0.20)	2.7 (0.27)
malathion	0.1 LC_{50}	1.1 (0.12)*	3.0 (0.25)*	3.1 (0.15)*
malathion	LC_{50}	0.9 (0.11)*	2.4 (0.07)	2.9 (0.14)
methoprene	0.1 LC_{50}	1.0 (0.14)*	2.6 (0.16)*	3.2 (0.18)*
methoprene	LC_{50}	0.9 (0.11)*	2.9 (0.23)*	3.0 (0.14)*
propoxur	0.1 LC_{50}	0.9 (0.08)*	2.4 (0.17)	2.5 (0.10)
propoxur	LC_{50}	0.7 (0.09)	2.5 (0.10)	2.7 (0.20)
resmethrin	0.1 LC_{50}	0.9 (0.07)*	2.7 (0.14)*	2.7 (0.06)
resmethrin	LC_{50}	0.9 (0.08)*	2.6 (0.14)*	2.6 (0.10)

[1] Time (days) from insecticidal treatment to pupation.
[2] Time (days) from insecticidal treatment to adult emergence.
* Value is significantly different from control, $P < 0.05$.

ment of methoprene resulted in reduced female wing length. Abu-Hashish[5], using LC_{50} and LC_{75} levels of 6 synthetic pyrethroids, reported no significant changes in wing dimensions of *Cx. pipiens*.

Only methoprene-exposed mosquito populations showed a significant reduction ($P < 0.01$) in longevity compared with controls (Table 6). Both exposure levels of methoprene resulted in reduced longevity in males and females. This result contrasts with the findings of Wijeyaratne[4] who suggests that methoprene does not affect longevity of *Cx. quinquefasciatus*. However, Arias and Mulla (1975), working with *Cx. tarsalis* Coq. and the chemicals methoprene and dimilin, found decreased adult mortality rates after larval exposure to 0.1 and 1.0 ppb.

Larval exposure to malathion, propoxur and resmethrin did not significantly affect adult longevity (Table 6). Likewise, larval exposure to malathion did not affect adult longevity in *Cx. pipiens* (Gaaboub and Dawood 1974), *Cx. quinquefasciatus* (Wijeyaratne[4]) or *Ae. aegypti* (Firstenberg and Sutherland 1981). Sublethal dosages of DDT (Gaaboub and Dawood 1974, Kerdpibule et al. 1981) and resmethrin (Focks 1984) did not affect adult longevity in various mosquito species.

Except the LC_{50} treatment of propoxur, both concentrations of all insecticides resulted in significantly lengthened ($P < 0.05$) time to pupation (Table 7). The time needed for males of insecticide-exposed mosquito populations to emerge was greater than controls for the malathion, methoprene and resmethrin treatments. Females of the populations exposed to the 0.1 LC_{50} treatments of malathion or to either treatment of methoprene also took a significantly longer time ($P < 0.05$) to emerge than did controls (Table 7). Increased larval and pupal periods resulting from insecticidal treatments is consistent with the earlier research of Wijeyaratne[4] who reported that LC_{50} levels of dimilin, dichlorvos, malathion and methoprene prolong the larval development period of *Cx. quinquefasciatus*. Lengthened larval development time was reflected in an increased generation time, particularly with dimilin and methoprene. This pattern was also reported with various dosages of other insecticides in autogenous *Cx. pipiens* (Farghal[6]).

Adult sex ratios were affected by exposure to methoprene, propoxur or resmethrin (Table 8). The proportion of adult females in the insecticide-exposed populations were significantly reduced ($P < 0.01$) following either propoxur or resmethrin exposure. In contrast, methoprene exposure resulted in a significantly increased ($P < 0.01$) proportion of females in the insecticide-exposed and untreated F_1 population. These effects were not observed in the F_2 population. Wijeyaratne[4] also observed similar sex ratio effects associated with methoprene treatments on *Cx. quinquefasciatus*. Gradual increases in methoprene concentrations were likewise accompanied by gradual increases in female:male ratio in autogenous *Cx. pipiens* (Farghal and Temerak 1981).

[5] Abu-Hashish, T. A. 1978. Morphological and biological studies in Egyptian mosquitoes under the action of synthetic pyrethroids with a new order of activity. Ph.D. Thesis. Faculty of Agriculture, University of Alexandria, Egypt.

[6] Farghal, A. I. 1979. Recent studies in culicine mosquito control. Ph.D. Thesis. Assuit University, Egypt.

Table 8. Sex ratio of adult *Culex quinquefasciatus* exposed as larvae to insecticides.

Chemical	Dose	Sex ratio (Female:Male)	
		Insecticide-exposed population	F_1 population
control	0	1:1.63	1:1.87
malathion	0.1 LC_{50}	1:1.82	1:1.61
malathion	LC_{50}	1:1.83	1:1.50
methoprene	0.1 LC_{50}	1:0.90*	1:1.16*
methoprene	LC_{50}	1:0.61**	1:1.15*
propoxur	0.1 LC_{50}	1:2.29*	1:1.56
propoxur	LC_{50}	1:2.22*	1:1.50
resmethrin	0.1 LC_{50}	1:1.69	1:1.63
resemthrin	LC_{50}	1:2.67*	1:1.79

* Values are significantly different, $P < 0.01$.
** Values are significantly different, $P < 0.001$.

CONCLUSIONS

Sublethal treatments (0.1 LC_{50} and LC_{50}) of either malathion, methoprene, propoxur or resmethrin to larvae profoundly affected a variety of reproductive parameters in *Cx. quinquefasciatus*. Methoprene exposure resulted in reduced egg production, egg raft size, egg hatching, egg length, wing length and adult longevity. Exposure to this chemical also resulted in slower development time of immature stages and a change in the sex ratio. Malathion exposure resulted in reduced egg production and egg length, and an increase in the development time of the immature stages. Propoxur and resmethrin exposure in the larval stage resulted in increased development time of immatures and a change in the sex ratio.

Our data clearly indicate that egg production in mosquitoes is adversely affected by selecting dosages of insecticides. The reason for this detrimental effect is unclear; however, larval nutrition, general metabolic poisoning or hormonal disturbances may affect egg production. In contrast, studies on the effects of nonselecting dosages of insecticides on mosquito production report a variety of results. These nonselecting dosages of insecticides may either increase or decrease egg production. The mechanism responsible for these differences in response resulting from larval exposure to low dosages of insecticides is unknown.

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