Bull. Environ. Contam. Toxicol. 31, 93–97 (1983)

Parathion Alters Incubation Behavior of Laughing Gulls

Donald H. White,[1] Christine A. Mitchell,[1] and Elwood F. Hill[2]

[1]*U. S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Gulf Coast Field Station, P.O. Box 2506, Victoria, TX 77902, and* [2]*Patuxent Wildlife Research Center, Laurel, MD 20708*

Organophosphate (OP) pesticides are favored for field application because they are quick-acting and relatively short-lived, and there is little bioaccumulation (STICKEL 1974). Certain of the OPs are extremely toxic to wildlife for short periods after application and reports of mortality in exposed avian populations have been well documented (ZINKL et al. 1978; STONE 1979; WHITE et al. 1979; ZINKL et al. 1981; HILL & FLEMING 1982; WHITE et al. 1982a; WHITE et al. 1982b). However, little is known of the sublethal effects of OPs on avian populations, although GRUE et al. (1982) present evidence that parental care may be significantly reduced in songbirds sublethally exposed to an OP. The primary toxic effect of OPs is inhibition of acetylcholinesterase (AChE) in the nervous system resulting in respiratory failure. In this paper we report the results of a field experiment to test the sublethal effects of parathion, an OP insecticide, on nest attentiveness of laughing gulls *(Larus atricilla)*.

MATERIALS AND METHODS

The study site consisted of a dredged material island (≃ 2 ha) near Laguna Vista (26°06' N, 97°18' W), on the extreme southern Texas Coast. About 50% of the island was covered by patches of low vegetation (<1 m) dominated by sea oxeye *(Borrichia frutescens)*, coast bacopa *(Bacopa monnieri)*, and glasswort *(Salicornia)*. At this site laughing gulls nested directly on the ground adjacent to surrounding vegetation. Nests were well constructed, mostly of dead stems of sea oxeye and saltbush *(Baccharis)*, and averaged about 5 cm high and 8 cm wide.

A pilot study was conducted to determine the proper dosage of parathion for our field experiment that would significantly inhibit brain AChE activity without overt signs of poisoning. Captive laughing gulls in groups of 2 were given oral doses of parathion in corn oil ranging from 0 to 10 mg/kg body weight, held for 20 hrs, and sacrificed for brain AChE determinations. Brains were assayed following ELLMAN et al. (1961) as modified by HILL & FLEMING (1982). We found that brain AChE activity was depressed by about 50% of normal in laughing gulls receiving 6 mg/kg parathion but evidence of intoxication was absent. On this basis, we chose 6 mg/kg body weight as our dose level.

On the morning of 3 May 1982, about a week after egg-laying began, we trapped one member of each pair of laughing gulls at 9 nests using conventional chicken wire funnel traps (WEAVER & KADLEC 1970). We had marked a series of nests with numbered stakes a week earlier so as to use pairs that were fairly synchronous in their clutch initiation. We trapped either sex for the test, since males and females share incubation duties equally (BURGER & BEER 1976). Birds were weighed, orally dosed with either 6 mg/kg parathion in corn oil or corn oil alone (controls), and marked about the neck with red dye. Five birds received parathion and 4 received corn oil. Birds were then released and their nests observed from a blind to determine if parathion altered incubation behavior. The blind had been constructed several weeks before egg-laying began so that the birds would become accustomed to it. After the birds were released, their activities in relation to predetermined behavioral parameters were recorded at 10-minute intervals. Observation periods were from 1100 to 1930 on 3 May, 0700 to 1900 on 4 May, and 0700 to 1200 on 5 May. Observational parameters included: how many birds were at the nest, which pair member was incubating, and the activities of the birds on the nest, such as preening, turning eggs, and settling on the eggs. Occasional behavioral observations also were made on marked birds when away from the nest, e.g., immediate post-release behavior and interactions with other birds in the colony.

RESULTS

The immediate behavior upon release of birds that were dosed with either parathion or corn oil was quite variable and unpredictable. We were not able to make extensive observations during this period since birds were dosed and released one at a time from within the blind, but a brief account of how some of the birds reacted follows. We saw a control (CO) and a parathion-treated bird (PT) fly directly to the water near the shoreline and begin to bathe, possibly in reaction to the dye on their necks; we did not see them drink. One CO circled the island several times and landed about 1 m from its nest, but made no attempt to replace its mate on the nest. Two PTs flew to the shoreline and stood motionless in a few inches of water; they did not bathe or drink while we observed them. Another PT flew directly to its nest upon release and began incubating. One CO flew directly to an adjacent island (100 m) and landed, but was not present at that location when we looked again about 15 minutes later. About 5 minutes elapsed from the time each bird was dosed and when it was released; during this period no birds regurgitated and none were seen regurgitating after being freed.

Observations and recording of the activities at the 9 study nests took less than 2 minutes at each 10-minute interval. There were 1152 observations recorded during the 3-day study period. At least 1 member of a pair was observed incubating the clutch 87% (998/1152) of the time and both members were seen on the nest 2% (26/1152) of the time. There was no difference ($\underline{P}>0.3$, Chi-square test) in the amount of time (11%) spent off the nest between groups. While incubating, birds usually sat on the eggs facing straight ahead in no particular direction ($\underline{P}>0.05$, Chi-square test). There were only 5 observations of birds turning or probing the eggs, 4 of settling on the eggs, 4 of actual nest exchange, and 1 of preening.

On the day of treatment, there was no difference ($P>0.05$, Chi-square test) in the proportion of time spent on the nest between PT and CO birds (Table 1). The stress of trapping and handling probably altered incubation behavior in both groups the first day. However, birds dosed with 6 mg/kg parathion spent significantly less time incubating on days 2 ($P<0.001$) and 3 ($P<0.05$) than did birds receiving only corn oil. By noon of the third day, sharing of nest duties between pair members in the PT group approached that of the COs, indicating that the parathion intoxication had subsided.

TABLE 1. Percentage of observation periods on nest: marked vs. unmarked birds[a].

	Time	CONTROL (n=4)			TREATED (n=5)		
Date	Period[b]	unmarked		corn oil	unmarked		parathion
3 May 82	1100-1930	68	: (n=193)[c]	32	74	: (n=248)	26
4 May 82	0700-1200	32	: (n=107)	68	61	: (n=135)	39
	1300-1900	25	: (n=57)	75	72	: (n=80)	28
5 May 82	0700-1200	47	: (n=94)	53	58	: (n=110)	42

[a] Observations when birds were off the nest were not included in the calculations.
[b] Observations were not continuous during each period.
[c] Observations at 10-minute intervals per period.

DISCUSSION

LUDKE et al. (1975) suggested that brain AChE inhibition in birds greater than 50% could be diagnostic of death. Therefore, the 50% that we induced in our treated birds probably represented severe, but sublethal, intoxication because adult laughing gulls killed by parathion had brain AChE inhibition of 57 to 89% (WHITE et al. 1979).

There are few published studies that implicate sublethal organophosphate poisoning in reproductive impairment of birds. WHITE et al. (1979) reported heavy mortality in a laughing gull breeding colony from parathion exposure. Adults had gathered poisoned insects from nearby cotton fields and fed them to their young. About 25% of the chicks and over 100 adults in the colony died. However, about half the chicks found dead that were analyzed had normal brain AChE activity. This suggested that the dead chicks with normal AChE levels probably died from starvation or exposure because their parents were dead or inattentive, having been exposed to parathion (either lethally or sublethally) in treated fields. In an experimental study, GRUE et al. (1982) dosed nesting wild female starlings *(Sturnus vulgaris)* with 2.5 mg/kg dicrotophos (an OP) and a control group with corn oil. They found that females receiving dicrotophos made fewer sorties to feed their young and remained away from nest boxes for longer periods than did controls. Also, nestlings of dicrotophos-treated females lost significantly more weight than did nestlings of controls. McEWEN & BROWN (1966) observed disruption of lek display behavior and weakened defense of breeding territories in wild sharp-tailed grouse *(Tympanuchus phasianellus)* given single oral doses of malathion.

Because of the small sample sizes in this study, we were unable to statistically compare reproductive success at nests of PT and CO birds. Even with larger sample sizes, it is doubtful that hatching success would have differed between groups since we only dosed one member of a pair. Our data show that the undosed members compensated and incubated the clutch in the absence of the treated pair members. Seldom was a nest left unattended in either group. However, our findings do indicate that a single oral dose of parathion significantly altered incubation behavior in those pair members receiving treatment. This effect of OPs could be catastrophic to birds where only one sex incubates the clutch, or if both sexes are exposed in those species sharing nest duties, thus making the clutch more susceptible to predation or egg failure.

SUMMARY

One member of each pair of incubating laughing gulls at 9 nests was trapped, orally dosed with either 6 mg/kg parathion in corn oil or corn oil alone, and marked about the neck with red dye. Each nest was marked with a numbered stake and the treatment was recorded. A pilot study with captive laughing gulls had determined the proper dosage of parathion that would significantly inhibit their brain AChE activity (about 50% of normal) without overt signs of poisoning.

After dosing, birds were released and the nests were observed for 2½ days from a blind on the nesting island. The activities of the birds at each marked nest were recorded at 10-minute intervals. Results indicated that on the day of treatment there was no difference ($\underline{P}>0.05$, Chi-square test) in the proportion of time spent on the nest between treated and control birds. However, birds dosed with 6 mg/kg parathion spent significantly less time incubating on days 2 and 3 than did birds receiving only corn oil. By noon on the third day, sharing of nest duties between pair members in the treated group had approached normal, indicating recovery from parathion intoxication. These findings suggest that sublethal exposure of nesting birds to an organophosphate (OP) insecticide, such as parathion, may result in decreased nest attentiveness, thereby making the clutch more susceptible to

predation or egg failure. Behavioral changes caused by sublethal OP exposure could be especially detrimental in avian species where only one pair member incubates or where both members are exposed in species sharing nest duties.

ACKNOWLEDGMENTS

We thank Lowell C. McEwen and Harry M. Ohlendorf for critical reviews of an earlier draft and Gloria Aguayo for typing the manuscript.

REFERENCES

BURGER, J. and C. G. BEER: Behavior 55, 301 (1976).
ELLMAN, G. L., K. D. COURTNEY, V. ANDRES, JR., and R. M. FEATHERSTONE: Biochem. Pharm. 7, 88 (1961).
GRUE, C. E., G. V. N. POWELL, and M. J. McCHESNEY: J. Appl. Ecol. 19, 327 (1982).
HILL, E. F. and W. J. FLEMING: Environm. Toxicol. Chem. 1, 27 (1982).
LUDKE, J. L., E. F. HILL, and M. P. DIETER: Arch. Environm. Contam. Toxicol. 3, 1 (1975).
McEWEN, L. C. and R. L. BROWN: J. Wildl. Manage. 30, 611 (1966).
STICKEL, W. H.: Annu. Conf. West. Assoc. State Game and Fish Comm. 53, 484 (1974).
STONE, W. B.: New York Fish and Game J. 26, 37 (1979).
WEAVER, D. K. and J. A. KADLEC: Bird-Banding 41, 28 (1970).
WHITE, D. H., K. A. KING, C. A. MITCHELL, E. F. HILL, and T. G. LAMONT: Bull. Environm. Contam. Toxicol. 23, 281 (1979).
__________., C. A. MITCHELL, E. J. KOLBE, and J. M. WILLIAMS: J. Wildl. Dis. 18, 389 (1982a).
__________., C. A. MITCHELL, L. D. WYNN, E. L. FLICKINGER, and E. J. KOLBE: J. Field Ornithol. 53, 22 (1982b).
ZINKL, J. G., J. RATHERT, and R. R. HUDSON: J. Wildl. Manage. 42, 406 (1978).
__________., D. A. JESSUP, A. I. BISCHOFF, T. E. LEW, and E. B. WHEELDON: J. Wildl. Dis. 17, 117 (1981).
Accepted March 3, 1983