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Parathion Causes Secondary Poisoning in a Laughing Gull Breeding Colony

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Use of organophosphate insecticides as replacements for the more persistent organochlorine compounds has increased dramatically in recent years. Organophosphates are desirable for field application because they break down rapidly in the environment and do not persist in animal tissues (STICKEL 1974). Nevertheless, certain organophosphates are extremely toxic to wildlife for short periods after application and have caused widespread mortality among exposed animals (MILLS 1973, STICKEL 1974, 1975, MENDELSSOHN 1977, and ZINKL et al. 1978).

In the spring of 1978, authors White and Mitchell were studying the reproductive success of several aquatic bird species nesting in Nueces Bay, Corpus Christi, Texas. On 28 June, we found 116 dead laughing gull *(Larus atricilla)* chicks, ranging in age from a few days to several weeks, and several dead adults on 2 study islands. Upon examining the stomach contents of 2 chicks and 1 adult, we found a pasty substance containing parts of adult and larval-stage insects. Concurrently on 28 June, author King investigated a report that dead laughing gulls were seen in and near cotton fields about 3 mi from the nesting colony; he counted over 100 dead adults, most of which were on the banks of a fresh water pond adjacent to the fields. Consultation with the local landowner revealed that the cotton had been treated with parathion, an organophosphate pesticide, at the rate of 1 lb/acre (1.13 kg/ha) on 27 and 28 June for control of bollworms, budworms, and cabbage loopers. In this paper we report the results of our investigation of the laughing gull die-off. This is thought to be the first recorded instance of an organophosphate insecticide killing by means of insects carried to the young.

MATERIALS AND METHODS

We collected a sample of dead chicks and adults on 28 June in order to determine the possible cause of mortality. In addition, specimens of apparently normal chicks and adults were collected from the nesting islands in Nueces Bay on 6 July to serve as controls in the analyses. All specimens were frozen with dry ice soon after collection and transported to a laboratory freezer; the frozen specimens were later shipped in dry ice to the Patuxent Wildlife Research Center, Laurel, Maryland, for analysis.

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Brains of birds found dead and of controls were homogenized in 0.05 Tris buffer (pH 8.0) at a ratio of 100 mg/mL and then were analyzed simultaneously for cholinesterase (ChE) activity (ELLMAN 1961) with a Beckman Acta II spectrophotometer. The anterior portion (proventriculus and ventriculus) of the gastrointestinal (GI) tracts, including ingesta, were prepared and analyzed for organophosphate compounds. A 10-g portion of the homogenized GI tract was mixed with sodium sulfate in a blender and extracted with dichloromethane for 7 h in a Soxhlet apparatus. The extract was concentrated and cleaned up by gel permeation chromatography (GPC) modified by increasing the length of the GPC packing to 500 mm (LEICHT et al., ABC Labs, Inc., Columbia, MO, unpublished data). The solvent system was dichloromethane/cyclohexane, 15/85, at a flow rate of 5 mL per min. The first 150 mL containing the lipid was discarded and 150 mL was collected. This eluate was concentrated to 5 mL and analyzed by a gas chromatograph equipped with a flame photometric detector and 1.5/1.95% OV-17/QF-1 column at 200°C. The lower limit of sensitivity was 0.002 ppm wet weight. The presence of parathion in 1 adult specimen was confirmed by gas chromatography-mass spectrometry.

RESULTS AND DISCUSSION

Organophosphate pesticides are toxic because they inhibit acetylcholinesterase in the nervous system and death usually occurs from paralysis. With these compounds, a 20% inhibition of brain ChE activity indicates exposure and inhibition greater than 50% is diagnostic of death (LUDKE 1975). Although the degree of inhibition associated with lethality has varied among workers (ZINKL et al. 1977), the relationship between brain ChE activity and organophosphate exposure appears to be quite good regardless of species, age, and sex (LUDKE et al. 1975).

Results of the brain ChE activity assays are shown in Figs. 1 and 2. Brain ChE in dead adults that were analyzed was inhibited by 57 - 89%, when compared to levels in apparently normal adults, and was enough to account for death (LUDKE 1975). Brains in 4 of 9 dead chicks analyzed were inhibited by 75 - 90% below normal (Fig. 2). However, the remaining 5 chicks had ChE activities similar to the controls; we believe these birds may have died from starvation or exposure because the parents had died earlier from parathion poisoning.

Parathion was not detected in GI tract homogenates of control birds (N = 2). However, of 9 GI tracts analyzed from birds found dead (5 adults and 4 chicks), 7 (5 adults and 2 chicks) contained parathion residues ranging from <0.02 to 10 ppm, wet weight. The GI tracts of 2 chicks found dead contained no parathion; brain ChE activity was within the normal range for these birds and they probably died from starvation or exposure. Since each GI tract was homogenized with ingesta present, the residues do not reflect ppm of parathion in the food items alone but include tissues of the GI tract.

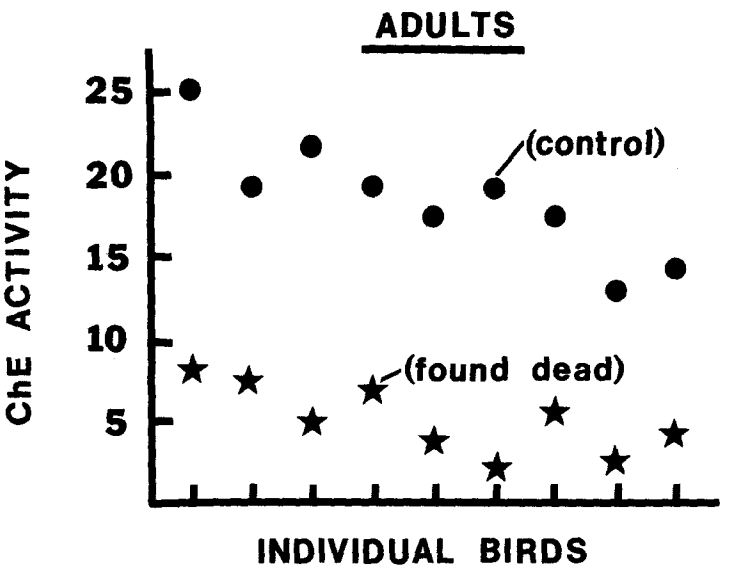

Figure 1. Brain ChE activity in adults found dead and in apparently normal specimens. ChE activity expressed as micromoles acetylthiocholine hydrolyzed per min per g brain tissue.

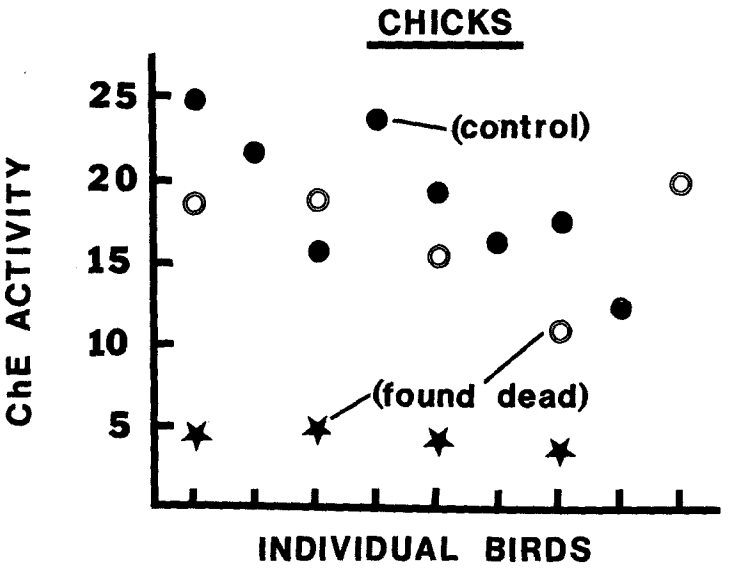

Figure 2. Brain ChE activity in chicks found dead and in apparently normal specimens. Open circles represent chicks that probably died from starvation or exposure rather than parathion poisoning.

We conclude that parathion applications to cotton fields near Corpus Christi, Texas, on 27 and 28 June 1978 were responsible for extensive mortality among laughing gulls, both adults and nestlings. Apparently the adults died from ingestion of poisoned insects gathered from the sprayed fields. Chicks in the nesting colony probably died from 2 causes: either from ingestion of contaminated food regurgitated by the parents, or from starvation or exposure because their parents were killed earlier. From our reproductive success data we estimated that approximately 25% of the immature population on the study islands died either directly or indirectly from the parathion. We did not attempt to estimate the extent of mortality in the adult population since many of the dead adults probably were not found.

The recommended application rate of parathion to cotton is ½ lb/acre (Victoria County Agricultural Extension Agency); the cotton fields discussed in this report received twice that amount. As STICKEL (1974) points out, many of the organophosphates known to be highly toxic to wildlife can be used safely in the environment if recommended formulations are applied. Although organophosphate pesticides kill primarily by depressing acetylcholinesterase, the effects are reversible; a sickened animal may completely recover and breed successfully. However, indiscriminate use of these compounds in areas of breeding colonies may have significant impacts, especially in areas occupied by threatened or endangered species. Our data demonstrate that parathion, a non-persistent organophosphate, can have adverse effects on breeding laughing gulls.

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REFERENCES

ELLMAN, G. L., K. D. COURTNEY, V. ANDRES, and R. M. FEATHERSTONE.: Biochem. Pharmacol. 7, 88 (1961).

LUDKE, J. L., E. F. HILL, and M. P. DIETER.: Arch. Environ. Contam. Toxicol. 3, 1 (1975).

MENDELSSOHN, H.: Biol. Conserv. 11, 163 (1977).

MILLS, J. A.: Proc. N. Z. Ecol. Soc. 20, 65 (1973).

STICKEL, W. H.: Proc. Annu. Conf. Western Assoc. State Game and Fish Comm. 11-13, 484 (1974).

STICKEL, W. H.: *In* Ecological Toxicology Research. Plenum Press, New York. eds. A. D. McIntyre and C. F. Mills (1975).

ZINKL, J. G., C. J. HENNY, and L. R. DEWEESE.: Bull. Environ. Contam. Toxicol. 17, 379 (1977).

ZINKL, J. G., J. RATHERT, and R. R. HUDSON.: J. Wildl. Manage. 42, 406 (1978).