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differentiated adipose cells, but were not well circumscribed and lightly encapsulated as are lipomas in mammals. In these respects they resembled, but were less invasive than, infiltrative lipomas previously described in man (Dionne et al., 1974, *Cancer* 33: 732-738; McChesney et al., 1980, *Vet. Pathol.* 17: 316-322) and in dogs (McChesney, 1980, op. cit.). These fish were from commercial and experi-

mental catfish production ponds that were not known to contain chemical carcinogens or carcinogenic pollutants.

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Secondary Poisoning of Franklin's Gulls in Texas by Monocrotophos

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On 4 June 1983, 45 dead Franklin's gulls (*Larus pipixcan*) were found at a freshwater pond on the Santa Ana National Wildlife Refuge (NWR) near Alamo, Texas. The birds were collected by Refuge personnel and frozen in polyethylene bags. Also, a sample of small cicadas (Cicadidae), probably regurgitated earlier by the dying gulls, was collected at the pond near the gulls and frozen. On 8 June, five healthy Franklin's gulls were shot at the Refuge and frozen to serve as controls in determining the cause of mortality. Brain acetylcholinesterase (AChE) assays were conducted on a sample of the birds found dead plus controls on 17 June and the proventriculi were shipped frozen to Patuxent Wildlife Research Center for chemical analysis of contents. Apparently, the die-off occurred over several days, for only seven birds found dead were suitable for brain assays and none of these were saved for necropsy.

AChE determinations were made using

the method of Ellman et al. (1961, *Biochem. Pharmacol.* 7: 88-95) as modified by Hill and Fleming (1982, *Environ. Toxicol. Chem.* 1: 27-38). Contents of individual proventriculi were analyzed for organophosphate and carbamate insecticides following White et al. (1982, *J. Field Ornithol.* 53: 22-27). The lower limit of quantification was 0.1 ppm, wet weight. Insecticide concentrations were confirmed by mass spectrometry in two samples.

AChE activities in brains of birds found dead and of controls and insecticide concentrations in their proventricular contents are shown in Table 1. AChE activity in birds found dead was extremely inhibited, averaging 95% below the average for controls, and was well below the 50% depression level indicative of death from an AChE inhibitor (Ludke et al., 1975, *Arch. Environ. Contam. Toxicol.* 3: 1-21). The proventriculi of all birds found dead contained small cicadas; one specimen had 83. Cicadas also were present in proventriculi of control birds. Monocrotophos [(E)-phosphoric acid, dimethyl (1-methyl-

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TABLE 1. Brain acetylcholinesterase (AChE) activities and monocrotophos concentrations in proventricular contents of Franklin's gulls, Santa Ana, NWR, Texas.

Group	(No.)	AChE activity ^a	% Inhibition ^b	Monocrotophos (ppm)
Control	(1)	14.5	0	ND ^c
	(2)	13.5	0	ND
	(3)	17.8	0	NA ^d
	(4)	26.8	0	NA
	(5)	14.9	0	NA
Found dead	(1)	0.3	98	1.6
	(2)	2.5	86	ND
	(3)	0.8	95	0.6
	(4)	0.3	98	ND
	(5)	0.8	95	ND
	(6)	0.3	98	1.0
	(7)	0.5	97	1.1

^a AChE activity expressed as micromoles acetylthiocholine hydrolyzed per min per g brain tissue.

^b AChE activities of individuals found dead were compared to the average AChE activity of controls.

^c ND = not detected at limit of quantification (0.1 ppm).

^d NA = no analysis.

3-(methylamino)-3-oxo-1-propenyl) ester] was detected in stomach contents of four of seven birds found dead, ranging from 0.6–1.6 ppm, wet weight, but was not present in stomachs of control birds (Table 1). The sample of cicadas collected on the pond's shore had 0.2 ppm monocrotophos. Only a small amount of food material was present in dead birds 4 and 5 (Table 1), thus this may have accounted for the absence of monocrotophos in the food material, or it may have been present in amounts below the 0.1 ppm quantification limit. It is common in similar avian die-offs for some stomach samples to contain no detectable chemical residues even though brain AChE activity may be greatly inhibited (Hill and Fleming, 1982, op. cit.; White et al., 1983, J. Wildl. Dis. 19: 373–375). Monocrotophos is highly toxic to birds in controlled laboratory studies (Hudson et al., 1984, U.S. Fish Wildl. Serv., Resour. Publ. No. 153, 90 pp.) and also has been responsible for mortality in wild populations (Mendels-

sohn and Paz, 1977, Biol. Conserv. 11: 163–170; White et al., 1983, op. cit.). All birds that we examined were in good flesh and had abdominal fat, although detailed necropsies were not performed.

Discussions with a local pesticide applicator revealed that monocrotophos had been applied to a sugarcane field adjacent to the Refuge for control of sugarcane beetles (*Euethiola rugiceps*) a few days before the dead gulls were discovered. Cicadas were not the target organisms. We visited the field on 8 June and found only a few emerging cicadas, but shed nymphal skins were abundant on the cane stalks and on the ground among the rows. Franklin's gulls were observed capturing cicadas over the field during our visit. Apparently, the main cicada emergence coincided with the spraying of the field, resulting in the deaths of the gulls. Only one additional Franklin's gull was found dead at the pond after 4 June (2 days later) suggesting that the toxic properties of the monocrotophos application had subsided by this time.

We conclude that anti-AChE poisoning due to monocrotophos was the cause of death in 45 Franklin's gulls found at a pond on the Santa Ana NWR, Texas. AChE inhibition in brains averaged 95% and the ingesta of some affected birds contained monocrotophos residues that were confirmed by mass spectrometry. The deaths of more secretive wildlife species may have gone undetected. Also, two endangered cats, the ocelot (*Felis pardalis*) and jagaurundi (*Felis yagouaroundi*), which occur on the Santa Ana NWR could have been exposed since several poisoned gulls found at the pond had been partially eaten by predators or scavengers.

Avian poisonings from organophosphate or carbamate insecticides occur frequently, and in some instances, are done intentionally (Stone, 1979, N.Y. Fish Game J. 26: 37–47; Hill and Fleming, 1982, op. cit.; White et al., 1983, op. cit.; Stone et

al., 1984, *Condor*, 86: 333–336). This investigation further demonstrates the dangers of using a highly toxic organophosphate insecticide such as monocrotophos near areas where wildlife congregate, especially near wildlife refuges, which often are the only remaining suitable habitat for wildlife in agricultural areas.

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