Dicrotophos Poisoning of Great-tailed Grackles in Texas

Authors: Mitchell, Christine A., White, Donald H., Kolbe, Elizabeth J., and Biever, Ronald C.

Source: Journal of Wildlife Diseases, 20(3) : 256-257

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-20.3.256

Journal of Wildlife Diseases, 20(3), 1984, pp. 256–257

Dicrotophos Poisoning of Great-tailed Grackles in Texas

Christine A. Mitchell, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, P.O. Box 2506, Victoria, Texas 77902, USA; **Donald H. White,** U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, School of Forest Resources, University of Georgia, Athens, Georgia 30602, USA; **Elizabeth J. Kolbe,** U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland 20708, USA; **and Ronald C. Biever,** Texas Parks and Wildlife Department, 1601 E. Crest Drive, Waco, Texas 76705, USA

On 20 April 1983 a bird die-off at the rodeo and fair grounds at West, Texas, 26 km north of Waco, was investigated. Approximately 30 great-tailed grackles, *Quiscalus mexicanus* (Gmelin), and one rock dove, *Columba livia* Gmelin, were found dead and dying. Necropsies were performed on four specimens. Nineteen fresh dead birds were collected, placed in plastic bags, and frozen. The carcasses were sent on dry ice to Victoria, Texas where they were kept frozen until 10 May 1983 when they were prepared for chemical analysis and brain acetylcholinesterase (AChE) assays were run. Three grackles, which had been collected near Fort Hancock, Texas on 7 January 1983, were kept frozen until 10 May and used as controls.

Brain AChE activity was determined colorimetrically on a random sample of five grackles and on the three controls 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). The assays were run at room temperature. The upper gastrointestinal (GI) tracts with ingesta were individually analyzed for organophosphate insecticide residues at the Patuxent Wildlife Research Center. Residues were determined on a gas chromatograph equipped with a flame photometric detector and a 1% Reoplex 400 column. Residues in one sample were confirmed by mass spectrometry. The lower limit of reportable residues was 0.5 ppm.

Received for publication 13 December 1983.

Brain AChE activity, expressed as micromoles of acetylthiocholine hydrolyzed per min per g of brain tissue, was 14.9, 11.1, and 12.8 ($\bar{x}$ = 12.9) for controls and 1.9, 1.4, 1.9, 1.1, and 1.4 for birds found dead. The average depression was 88% and ranged from 85–91%. This is in excess of the 50% depression level considered indicative of a lethal exposure to an AChE inhibitor (Ludke et al., 1975, Arch. Environ. Contam. Toxicol. 3: 1–21). Sorghum seeds were found in four of the five GI tracts. Two of the GI tracts contained detectable levels of dicrotophos (3-hydroxy-*N*,*N*-dimethyl-*cis*-crotonamide dimethyl phosphate), an AChE inhibiting chemical, at 16 and 34 ppm on a wet weight basis. It is not uncommon in a die-off of this sort for some of the GI tracts to contain no detectable chemical residues or food (Hill and Fleming, 1982, op. cit.; White et al., 1983, J. Wildl. Dis. 19: 373–375). Since death is not necessarily immediate (Hill and Camardese, 1982, Amer. Soc. Testing and Materials Special Tech. Rep. 757: 41–65) a bird can complete digestion of the food in its GI tract or consume uncontaminated food before dying. The grackles which were necropsied showed no signs of gross pathology and their body condition was good with adipose tissue present in the body cavity. This is similar to other die-off situations (Felton et al., 1981, Vet. Rec. 108: 104), but contrasts with the experimental work done by Grue (1982, Arch. Environ. Contam. Toxicol. 11: 617–626) on common grackles, *Quiscalus quiscula* L., which died with little body fat after exposure to dicrotophos in their diets.

The much reduced brain AChE levels coupled with the AChE inhibiting chemical in the GI tracts allow us to establish the cause of death as dicrotophos poisoning. Because dicrotophos (EPA reg. No. 201-274) is not registered for use on any grain crop, either intentional poisoning or gross misuse is indicated. Although this is not the first reported mortality in bird populations due to dicrotophos poisoning (U.S. Dept. Interior, 1969, Bureau of Sport Fish. Wildl. Resource Publ. 74: 55), it is the first which substantiates it with biological and chemical analyses.

We thank Dick Crick for conducting necropsies and pathological examinations of specimens, and Charles Henny, Gary Heinz, and Clementine Glenn for assistance in the preparation of the manuscript.