UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

FEB 7 1990

OFFICE OF
PESTICIDES AND TOXIC SUBSTANCES

MEMORANDUM

SUBJECT: Dimethoate Data Call In Notice

FROM: for James W. Akerman, Chief
Ecological Effects Branch
Environmental Effects Division (H-7507-C)

TO: Lois Rossi, Chief
Registration Branch
Special Review and Reregistration Division (H-7508-C)

The Ecological Effects Branch (EEB) is requesting that a Data Call In Notice be sent out on dimethoate requiring ecological effects data, in particular avian field testing.

On October 1, 1987, the EEB Science Chapter was completed for the Dimethoate FRSTR. At that time EEB had identified new data requirements, which included, terrestrial field testing.

The need for field testing had been brought to our attention by the EPA- Corvallis Environmental Research Laboratory (EPA-Corvallis ERL)and the U.S. Fish and Wildlife Service, since adverse effects have been reported from the use of dimethoate in the field. In particular, there have been sage grouse mortalities associated with the use of this chemical in alfalfa fields in Idaho. In addition, through personal communications with Dr. Fairbrother, EPA- Corvallis ERL, dimethoate has also been associated with the decrease in pheasant populations in Oregon.

EEB would like a data call in notice to be sent to the registrant (s), so that at least the concerns will have been identified, and the process will have been started to investigate field effects. If you have any further questions, please feel free to contact Candy Brassard (557-0019). Thank you.

cc: Anne Barton/ EFED
Rick Tinsworth/ SRRD

1

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
ENVIRONMENTAL RESEARCH LABORATORY
200 S.W. 35TH STREET
CORVALLIS, OREGON 97333

February 5, 1990

Candy Brassard
Office of Pesticide Programs
Mail Code H-7507C
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Dear Candy:

The first phase of our study investigating the possibility of exposure of Oregon pheasants to the organophosphate insecticide dimethoate has been completed. This phase concentrated on refinement of study design and analytical techniques and was conducted using bobwhite quail as a surrogate species. The next phase of the study will be a definitive experiment with pheasants. Tissue analyses of field-collected pheasants is continuing.

Attached is a progress report summarizing results of the first phase of the study. Please feel free to contact me if you have any questions concerning this work or comments about future directions. Your interest and input are always welcome!

Sincerely,

Anne Fairbrother MAB

Anne Fairbrother, DVM, PhD
Wildlife Research Program
(503) 757-4716
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D R A F T

PHEASANT/DIMETHOATE STUDY PROGRESS REPORT

(#88-04)

January 1990

Anne Fairbrother

USEPA

Environmental Research Laboratory

200 S.W. 35th Street

Corvallis, Oregon 97333

PHEASANT/DIMETHOATE STUDY (#88-4)

Progress Report

Anne Fairbrother
USEPA
Corvallis, Oregon 97333

January 29, 1990

The first phase of the study has been completed. This report reviews the results of chemical analysis of quail tissues following exposure to dimethoate-treated feed. Similar studies with pheasant are under way to determine if species differences exist.

SUMMARY OF PROCEDURES

Quail were fed dimethoate-treated feed for 5 days at the following concentrations: 0, 20, 100, and 500 ppm. Six birds from each treatment group were euthanized at 6 hours, 1, 3, and 5 days after replacement of treated feed with clean feed. (Note: One of the birds in the 500 ppm treat-

ment group died within 8 hours after presentation of treated feed. This resulted in only five birds from this treatment group being euthanized at the 5-day post-treatment period.)

Kidney and brain tissues were removed from each bird and stored at -70°C until analyzed. Liver and kidney tissues were macerated finely, sonicated, and extracted in ethyl acetate in preparation for GC analysis using an NPD detector (specific for compounds with large numbers of phosphorus molecules). Brains were homogenized, centrifuged, and analyzed for cholinesterase activity. Brains with depressed activity were incubated with 2-PAM to determine if reactivation of the enzyme to normal values would occur. (This could then be used as a confirmation of dimethoate-induced reduction in cholinesterase activity from field-collected samples.)

RESULTS

GC analysis of liver and kidney tissues produced a peak with a retention time similar to that produced by the dimethoate standard. However, the peak shape differed in that the base was much broader than the base of

the dimethoate peak. When the machine was reconfigured to slow down passage of the sample through the column, the retention time of the peak was between that of dimethoxon and dimethoate. This unidentified peak was observed in both liver and kidney tissues from birds in all treatment groups including controls that were euthanized at all time intervals; however,m not all samples had a measurable peak. Therefore, we concluded that this peak represents some compound not related to dimethoate or its metabolites. We plan to use a GC-mass spectrometer to try to identify the compound responsible for producing the peak.

Brain cholinesterase activity was reduced 20-50% in quail exposed to dimethoate (Table 1). Incubation with 2-PAM did not increase activity of depressed samples. This is consistent with results reported by Zech et al. (Biochimica et Biophysica Acta 128:363-371, 1966). Brain tissue from methyl parathion-treated birds had cholinesterase activity boosted to normal levels when treated with 2-PAM (run as a positive control).

DISCUSSION

The quail study was very useful for establishing procedures and specific protocols for feeding and chemical analyses. In particular, we discovered the unidentified peak on the chromatogram that could potentially confound interpretation when looking for residues of dimethoate or its metabolites. We will reexamine the chromatographs of tissues from pheasants collected last spring to see if the "dimethoate" peak is truly the OP-compound of if it has a shape similar to the unidentified peak discovered in these studies. The additional work with field-collected and captive pheasants will also help determine if this is an anomaly unique to quail or if it occurs is pheasants as well. In any case, this has important ramifications for all analytical chemists analyzing field-collected tissue samples for dimethoate residues.

The lack of response of dimethoate-depressed brain cholinesterase activity to 2-PAM treatment is very significant. Many diagnostic laboratories are beginning to use this reactivation technique as part of their diagnostic procedure for organophosphate exposure. The results with dimethoate suggest that a negative result of the 2-PAM procedure

would not be very informative. Additionally, 2-PAM is used therapeutically in human and veterinary medicine to treat sublethally exposed individuals. Obviously, in the case of dimethoate poisoning, it is contraindicated and atropine would be the treatment of choice.

CONTINUING STUDIES

Liver, crop, and crop contents from pheasants collected in December and January are currently being analyzed. These birds were collected in areas treated with dimethoate within 14 days prior to harvesting the birds. Brain cholinesterase activity also will be determined for these birds.

Feeding studies with 39 female pheasants will follow a similar protocol as the quail study. Dimethoate-treated feed at 0, 100, and 500 ppm will e presented to the birds for 5 days during the first week of February. Birds will be euthanized at 6 hours, 1 day, and 3 days post-exposure. Tissue analyses should be completed by the end of the month. 2-PAM reactivation will be attempted on pheasant brains that have reduced cholinesterase activity.

Table 1. Mean ± standard error brain cholinesterase activity (umoles AChE hydrolyzed per gram brain tissue) of Northern Bobwhite exposed to dimethoate-treated feed for five days.

Day Post-Treatment	Dimethoate Concentration in Feed (ppm) 0	20	100	500
0[1]	---	---	---	1.4 ± --- (1)[3]
0.25[2]	6.4 ± 0.6 (2)	5.6 ± 0.1 (3)	4.0 ± 0.2 (3)	3.6 ± 0.3 (3)
1	5.8 ± 0.2 (2)	5.6 ± 0.2 (3)	5.2 ± 0.2 (3)	3.4 ± 0.8 (4)
3	5.4 ± 0.1 (2)	6.0 ± 0.04 (3)	4.8 ± 0.2 (3)	5.0 ± 0.4 (2)
5	5.7 ± 1.2 (2)	6.2 ± 0.3 (3)	5.3 ± 0.3 (3)	5.4 ± --- (1)

1 Bird died 6-8 hours after presentation of treated feed.
2 7 hours post-treatment.
3 Numbers in parentheses indicate sample size.