RICHARD J. NEVES

Please make copies for Jim Fleming TN Tech 1992
10/92 Dick Biggins USFWS
Jim Lewis Corps
John Jenkinson TVA

UNITED STATES GOVERNMENT

memorandum

DATE: September 23, 1992

REPLY TO ATTN OF: Jim Fleming

SUBJECT: Progress Report on freshwater mussel response to cholinesterase inhibiting pesticides

TO: Dick Biggins, John Fridell

We have just completed the first round of our testing to examine the responses of freshwater mussels to acephate and aldicarb. Our first test was to try to establish a dose response curve for both lethality and cholinesterase activity. We used *Elliptio complanata* as for the test species. We prepared a control and 5 geometrically arranged concentrations of each of the two compounds (based on preliminary work we conducted earlier this summer). Three mussels were added to each experimental chamber which contained 2 liters of the test solutions. Mussels were kept in the solutions for 96 hrs, then sacrificed by freezing. Observations of mussel behavior during the test period indicated that in the highest dose groups, slight gaping of the shell occurred during the first 24 hours, but was not observed as the test continued. Siphoning was observed in all test subjects throughout the test period.

Our maximum doses of 320 ppm acephate and aldicarb did not cause any mortality during the test period. This was not expected as we had previously observed highly significant cholinesterase inhibition at 1 and 10 ppm, and calculated that higher doses would likely be lethal.

Cholinesterase activity of the mussels in this test responded significantly to the exposures (Figures 1,2). However, it may be significant that once experimental concentrations reached 5 ppm, further reduction of cholinesterase activity was not noted at higher concentrations.

Concurrently with the mussel tests, we exposed *Corbicula* to the same test concentrations and protocols. However, for *Corbicula*, we used the entire body (less shell) of the animal in the cholinesterase test. Again, no mortality was observed in *Corbicula* during the 96 hr test period. However, in contrast to the adductor

OPTIONAL FORM NO. 10
(REV. 1-80)
GSA FPMR (41 CFR) 101-11.6
5010-114
* U.S. GPO: 1987 — 181-247

muscle of *E. complanata*, cholinesterase activity did not respond significantly to test concentrations (Figures 3,4). This either suggests that cholinesterase activity of the "meat" of *Corbicula* is too low to measure or is not responsive. At any rate, it does not appear that *Corbicula* will serve as a useful surrogate for mussels in a biomonitoring program. Therefore, we will discontinue work with *Corbicula*.

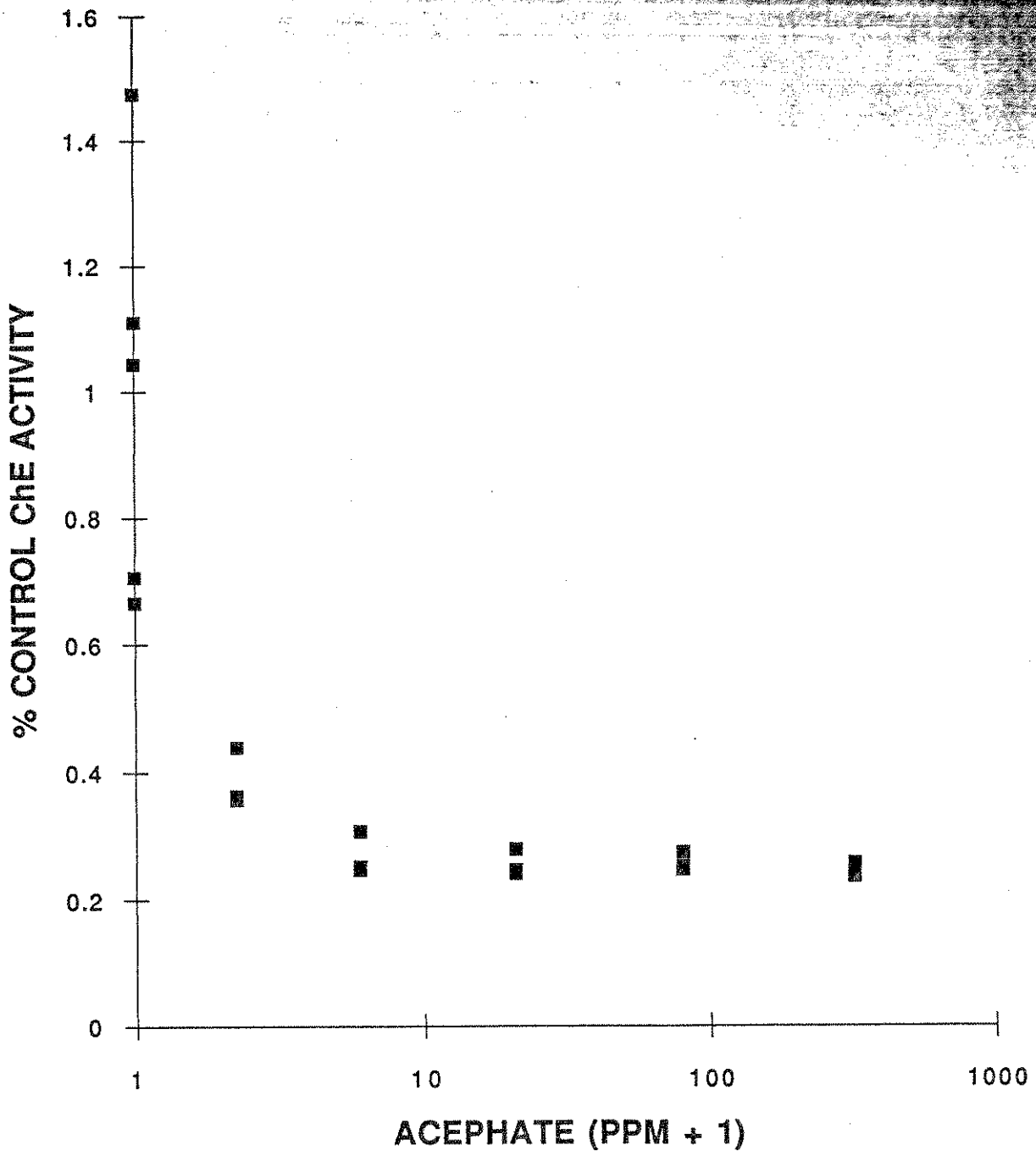
Our next test, which is to be completed next week, will attempt to determine if temperature of the water significantly enhances the toxicity of acephate and aldicarb. It is easy to speculate that for cold-blooded animals, that toxicity might be related to metabolic rates which increase with temperature.

Following this test, we will proceed to determine recovery responses of ChE activity in mussels so that we can define the "window of sampling opportunity." This will define the basis for employing these techniques in a mussel biomonitoring program.

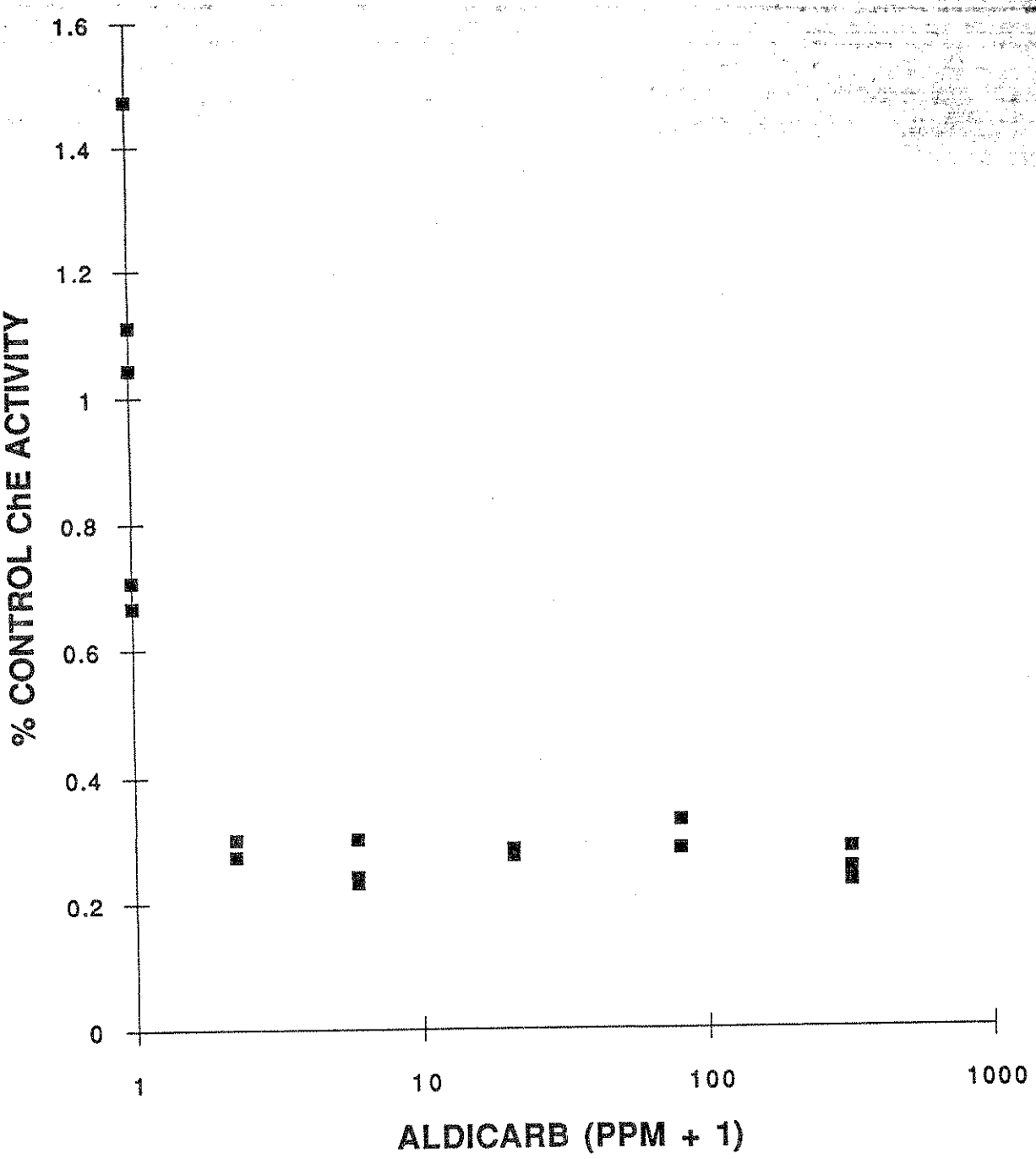
We will keep you informed of our progress.

cc. Tom Augspurger

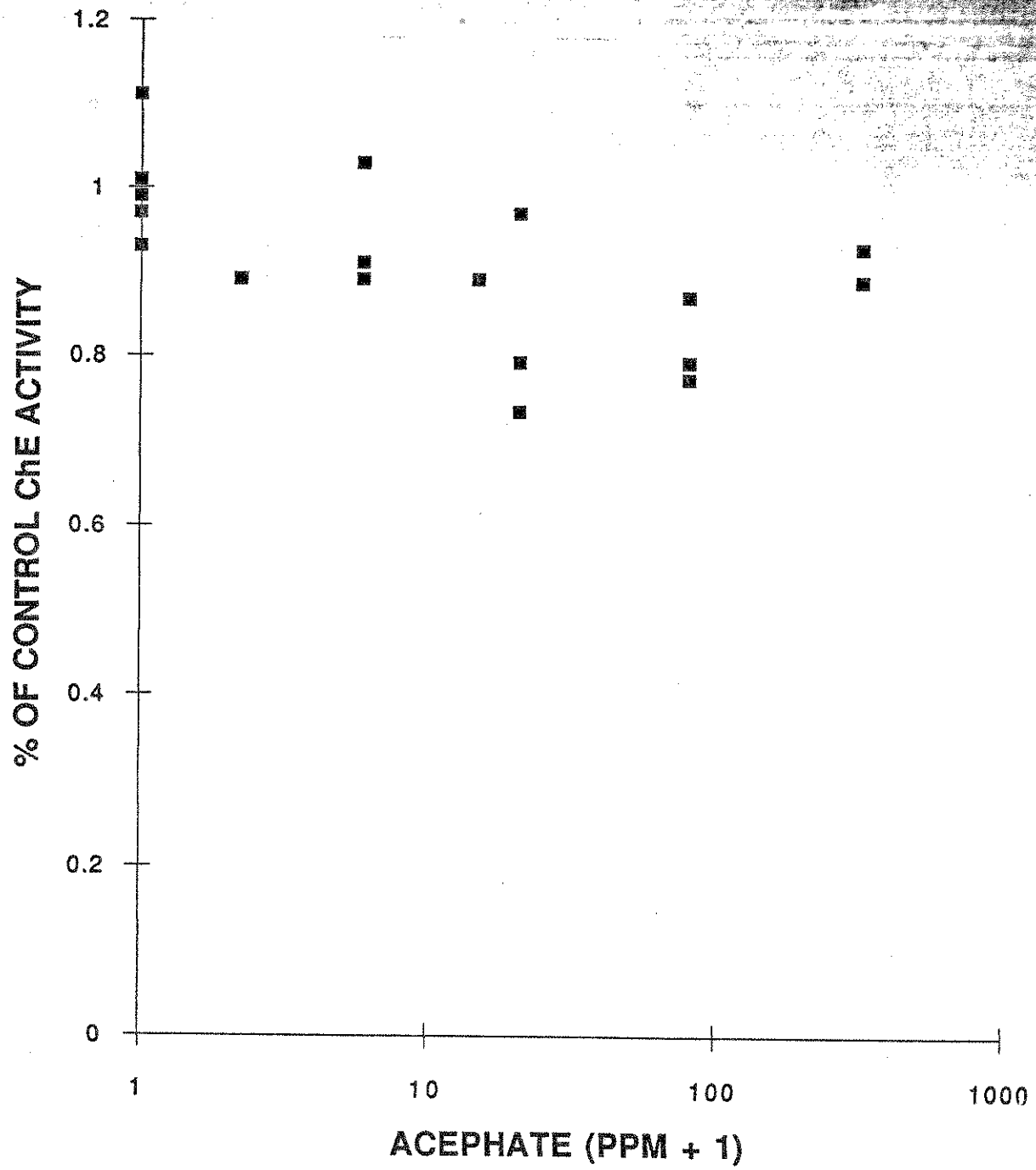
ChE RESPONSE OF MUSSELS TO ACEPHATE



ChE RESPONSE OF MUSSELS TO ALDICARB



ChE RESPONSE OF CORBICULA SP. TO ACEPHATE



ChE RESPONSE OF CORBICULA SP. TO ALDICARB