Toxicity of Chlorpyrifos to Mallard Ducks

Stuart H. Hurlbert
Department of Biology
San Diego State University
San Diego, Calif. 92182

In a recent review article on toxicity of chlorpyrifos to birds, KENAGA (1974, p. 28) devotes a full page to criticizing an experimental study in which we found that treatment of shallow fenced ponds with this compound resulted in mortality of young mallard ducks (HURLBERT et al., 1970).

Kenaga's critique represents a condensation of an earlier unpublished one (KENAGA and DOWELL 1970) to which we had responded privately (HURLBERT 1970) but apparently unconvincingly. As their criticisms are strong and as our study remains, even after nine years, the only carefully controlled and replicated field investigation of DURSBAN ®'s effects on any waterfowl species, we feel obliged to respond. So that we may avoid excessive quotation, the interested reader should refer to KENAGA's (1974) article.

The body of Kenaga's criticism is presented as a numbered list of seven "important factors which [we] ignored or omitted from consideration." Without repeating these, we will respond to them individually. We will show that the intensity of Kenaga's criticism reflects only on the alarm with which Dow Chemical viewed our results and their possible influence on DURSBAN ®'s registration and not on the validity of those results. Our observation of post-treatment duck mortality of 42% on treated ponds and 0% on control ponds and our attribution of the mortality to the DURSBAN ® applications remain effectively unchallenged.

1. As stated in our paper (HURLBERT et al., 1970), two ducks died before the first application of DURSBAN ® and these were birds penned on ponds that subsequently were treated. However, since a total of eight ponds (40 ducks) were treated whereas only two ponds (ten ducks) were kept as controls, this pre-treatment mortality hardly constituted evidence that the ponds assigned for treatment were less salubrious than the control ponds in some way. The difference in pre-treatment mortality rates between control ponds (0%) and treated ponds (5%) was so obviously non-significant statistically that it seemed unnecessary to elaborate the point.

2. We stated (p. 44) that the experimental ponds had been used earlier for field tests with five organophosphorus compounds and cited a paper by KEITH and MULLA (1966) that provided details

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of those tests. Since the last of these tests were performed more than three years before the DURSBAN ® experiment and since there were abundant data indicating that such organophosphorus compounds effectively disappear within several days to several weeks after application in the field, it was our judgement that residue analyses for these other compounds were unnecessary. We see no reason to question the soundness of that judgement.

3. Contrary to their statement, the total amount of chlorpyrifos actually applied to each pond is easily calculated from the data we provided on application rates and pond dimensions. In fact, KENAGA and DOWELL (1970) themselves carried out these calculations and presented them in their unpublished report! If the implication is that we made mathematical errors at the time fo filling our spray cans--well, who knows? KENAGA (1974, p. 28, 34) himself errs in stating that our most heavily treated ponds received, in total, 400 times more insecticide than our most lightly treated ponds (the actual difference was 100-fold). But we have no reason to think that such errors intruded at any point in our own study.

4. Since KENAGA's (1974) item 4 is a straightforward paraphrasing of our own statements, it is unclear what we "ignored or omitted."

5. Conventional toxicological frameworks such as dose-response and time-response curves provide a poor foundation for understanding the effects of pesticides in natural or semi-natural ecosystems. In such contexts the doses--the amounts actually ingested or absorbed by the organisms--are not known; and so many factors other than the rate of application operate to determine dose that the correlation between rate and dose cannot be presumed a priori to be high. For example, the limited solubility of a given insecticide and the presence of large amounts of insecticide-adsorbing suspended silt could effectively set an upper limit to the dose received by a certain aquatic invertebrate; thus over a certain range of application rates, mortality in natural populations of that invertebrate could remain essentially constant. In the case of organisms such as ducks there are even more possibilities of this sort. In our study, perhaps the ducks continued feeding until they felt a certain intensity of discomfort from the ingested chlorpyrifos. Ducks exposed to the high application rates might thus have ceased feeding after some minutes while those exposed to the lower rates might have continued feeding for one or more hours. The amounts of chlorpyrifos ingested per duck under those circumstances would have been rather similar for the different application rates. Such a mechanism could help account for the rate independent duck mortality, as could the mechanism suggested in our original paper (HURLBERT et al., 1970). Both are speculative, of course, and there are many other possibilities; we did not pretend to determine the mechanism whereby DURSBAN ® had its effect. It may even have been that DURSBAN ® caused duck mortality indirectly, e.g., by

stimulating the development of toxic blue-green algae populations. DURSBAN® applications had that very effect in an experiment the following year in the same ponds (HURLBERT et al., 1972). Kenaga apparently feels that these sorts of relationships could account for rate independent mortality over, at most, a 10-fold range of application rates; we feel certain the range can be much greater.

6. and 7. Few autopsies and residue analyses were made on ducks, as Kenaga correctly states. Live ducks could not be sacrificed without critical reduction in sample sizes. Ducks that died were recovered after variable periods of time during which they were subject to rapid decomposition in very hot weather; it is unlikely that the presence, absence, or concentrations of the rapidly metabolized chlorpyrifos in their tissues could have told us much. We regret not having examined the ducks' gut contents; these might have provided a better idea of the mechanism of DURSBAN®'s effect. However, it is difficult to imagine circumstances under which the suggested autopsies or residue analyses would have provided data serving to exculpate DURSBAN® as the immediate cause of duck mortality on the treated ponds.

It is conceivable that under less severe conditions (e.g., cooler weather, more food), fewer or no ducks might have died on treated ponds. There is, however, very little doubt that under the conditions that did exist, DURSBAN® applications caused duck mortality. Carefully designed experimental studies of DURSBAN®'s effects on ducks under other semi-natural conditions are desirable and may demonstrate this insecticide to be less hazardous than do our data.

References

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