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COMPARATIVE DIETARY TOXICITIES OF PESTICIDES TO BIRDS

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ABSTRACT

This report presents measurements of the lethal dietary toxicity of 89 pesticidal chemicals to young bobwhites, Japanese quail, ring-necked pheasants, and mallards. Toxicity is expressed as the median lethal concentration (LC_{50}) of active chemical in a 5-day ad libitum diet. LC_{50}'s and associated statistics are derived by methods of probit analysis. Endrin consistently was the most toxic chemical while aldrin and dieldrin were among the six most toxic chemicals of those tested on all species. In general, organophosphates were less toxic than aldrin or dieldrin, and herbicides were of a low order of toxicity. There were obvious inconsistencies in the relative sensitivity of the four species to various chemicals.

INTRODUCTION

It is now well documented in scientific literature that pesticidal contamination of ecosystems can alter the status of animal populations through diverse, often complex modes of action (see Stickel, 1968). Direct lethal toxicity is the most obvious mode of action, and laboratory measurements of lethality are basic in predicting the immediate impact of pesticides in the environment.

The following report presents measurements of the lethal toxicities of 89 pesticidal chemicals as administered for 5 days in diets of young birds of four species: bobwhite (*Colinus virginianus*), Japanese quail (*Coturnix coturnix japonica*), ring-necked pheasant (*Phasianus colchicus*), and mallard (*Anas platyrhynchos*). All work was conducted at the Patuxent Wildlife Research Center. The study was designed to provide statistically reliable estimates of the relative toxicity of any two chemicals as tested. It also permits comparisons of species sensitivity to pesticides in the diet.

None of the findings is intended to imply chemical safety beyond the scope of the study; while measurements of lethal toxicity constitute an important first step in evaluating pesticidal hazard, comprehensive estimates of total population effects require knowledge of a variety of parameters (physical, chemical, biochemical, and toxicological), involving suspect degradation products as well as parent chemicals. Such elusive, delayed effects as reproductive impairment or alterations of critical behavior patterns may be highly detrimental. Further, there is little apparent correspondence between a chemical's lethal toxicity and its capacity to induce sublethal complications. For example, DDE, a ubiquitous metabolite of DDT, is not especially toxic but produces serious reproductive effects in various species of birds (Heath et al., 1969; Wiemeyer and Porter, 1970).

PROCEDURES

All test birds were incubator-hatched progeny of breeding colonies maintained on the Patuxent Center. Bobwhites, pheasants, and mallards were phenotypically indistinguishable from wild birds; our Japanese quail colony was started from eggs obtained in 1964 from Auburn University, Auburn, Ala. All colonies were randomly outbred so that findings, although probably associated with greater variances than those among inbred strains, can be more readily related to wild populations. Tests were conducted with bobwhites, pheasants, and mallards during spring and summer; Japanese quail were tested throughout the remainder of the year.

Protocol for Determining Lethal Dietary Concentrations of Chemicals:

The protocol is essentially that described by Heath and Stickel (1965) for testing dietary toxicity of pesticides to birds, and proposed by the U.S. Department of the Interior for use in pesticide registration (ref. 19). Toxicity is expressed as a median lethal concentration (LC_{50}) of chemical in dry diet. The LC_{50} is defined herein as ppm toxicant in an ad libitum diet expected to produce 50 percent mortality among 2- to 3-week-old birds in 8 days comprising 5 days on treated diet followed by 3 days on untreated diet. The final 3 days are included to detect chemical mortality induced beyond the dosage period; otherwise LC_{50}'s would tend to be overestimated. All measurements are in terms of the active ingredient, exclusive of diluents or impurities.

Tests with gallinaceous chicks were conducted in laboratory-housed brooder units. Each unit consists of six separated tiers of four wire pens in which heat is thermostatically controlled. Tests with mallard ducklings were conducted in weatherproof wooden pens on straw-covered concrete slabs, each supplied with an infra-red heating lamp and a trough of running water.

Birds were randomly assigned to study pens on the day preceeding a test and acclimated on untreated commercial diet prior to dosage. Usually 10 birds were assigned per pen, although six to 15 birds were sometimes used, depending upon availability. Dosage was never initiated before birds were 9 days old, to avoid possible interference of chemical intake by yolk sac absorption and to exclude hatchling mortality. There was no attempt to sex the birds at this early age.

To prepare test diets, chemical was dissolved in corn oil or, as necessary, in propylene glycol, and was then mixed thoroughly with dry commercial mash in a ratio of 2 parts of solution to 98 parts of feed by weight. An equal amount of pure corn oil was added to "control" diets. Thorough mixing of feed and solution was accomplished with a commercial food mixer.

Each chemical was generally administered in six dietary concentrations spaced geometrically over a span intended to produce mortality ranging from 10 to 90 percent during the 8-day period. Concentrations were selected after range finding with three widely spaced levels. One pen of birds was used per concentration, and facilities were adequate to test as many as seven chemicals (i.e., 42 concentrations) simultaneously. Included with each set of chemicals was a dieldrin "standard" of six concentrations, and three to six pens of control birds fed untreated diets. A completely randomized design was used in each experiment, with treatments and birds randomly assigned to pens.

The Probit Analysis:

Deaths in each pen were recorded daily, and total deaths during the 8-day period were used to derive percentages of mortality resulting from each dietary concentration of chemical. There were infrequent deaths among the untreated controls, and these were used to adjust the data for extraneous mortality by means of Abbott's formula (Finney, 1952).

All LC_{50}'s and associated statistics were derived by methods of probit analysis as described by Finney (1952) and programmed for computer by Daum and Killcreas (1966). The program calculates a number of maximum liklihood statistics including: the 95 percent confidence limits of each LC_{50}; the slope, and its standard deviation, of the weighted linear regression of probits on log-concentration; and the relative toxicity, with confidence limits, of any two chemicals after testing regression lines for parallelism and heterogeniety. The program fits up to 10 parallel probit regression lines simultaneously; thus chemicals tested in any one experiment were analysed as a set.

The Dieldrin Standard:

A dieldrin standard was used in every experiment, i.e., with every set of chemicals tested simultaneously. The necessity for a standard to adjust comparisons of toxicity of two or more chemicals tested at different times or locations has been discussed by various authors (Sun, 1950; Finney, 1952; Bliss, 1952). Without a standard, differences in animal sensitivity between experiments, whether due to physiological or environmental causes, can lead to biased comparisons of toxicity. Comparisons adjusted through a standard will be unbiased providing any differences in sensitivity between studies affect the toxicity estimates of the standard in the same proportion as those of the test chemicals. We selected dieldrin as a standard because it is a well-known chemical that consistently provided an acceptable probit regression line. DDT was used initially but tended to give heterogeneous results.

Not all chemicals are sufficiently toxic to be lethal at concentrations that might reasonably be expected in the environment. We therefore established a ceiling of 5,000 ppm for an LC_{50}, with occasional exceptions. Chemicals shown in range finding tests to have LC_{50}'s greater than 5,000 ppm were not tested further.

Rationale for the Protocol and the LC_{50}:

When the protocol was designed, several investigators had reported that the lethality of a pesticide mixed in the diet could differ markedly from that of the concentrate administered as a single

oral dose via capsule or gavage (Stickel et al., 1965). In human toxicology, where there is major concern with accidental or suicidal poisoning from sudden doses of concentrate, the classic single oral dose is clearly appropriate. Our particular interest, however, was in dietary toxicity because ingestion is undoubtedly the predominant route of exposure in wild species.

The primary objective in designing the protocol was to establish a dosage period short enough to approach acute exposure, yet long enough to insure adequate feeding activity by all test birds to produce sufficient mortality for meaningful determinations. The 5-day period was selected and, we believe, has proved to be satisfactory. (The important subject of chronicity, involving rates of chemical storage and excretion as well as degradation, is best studied using dosage periods longer than 5 days [Hayes, 1967; Weil and McCollister, 1963].)

The administration of toxicant blended in the diet provides in a sense, an "applied" measurement of toxicity, in that incorporated in each measurement are not only the digestive factors that determine uptake of chemical from the feed into the system, but also the propensity of the animal to ingest food containing the pesticide. Therefore, we have expressed lethality in terms of "ppm chemical in feed" rather than attempting to convert to a ratio of unit of chemical per unit of body weight, such as mg/kg.

Further reasons made such conversions impractical. Food consumption could not be measured accurately, since feed scattered by birds became mixed with litter and droppings, and its weight could only be estimated. Each bird in a given pen actually ingested a slightly different amount of toxicant, and there is no assurance that if each bird had consumed the average mg/kg for the pen the same percentage of mortality would have resulted. A conversion to mg/kg/day would provide a better measurement than mg/kg but would not overcome the problem of food spillage. At best it would be a rough measurement based on pen averages and would take additional time to compute. For these reasons conversions were not attempted.

Proper measurements of mg/kg/day require that birds be caged individually, weighed daily, their dosage predetermined and diet prepared daily, and a procedure utilized to insure complete ingestion of the preparation. Stress caused by excessive handling might conceivably lead to anomalous results.

Food consumption, corrected for estimated spillage, was measured to detect reductions in intake due to treatment. Repellency was rarely demonstrated (as substantiated by satisfactory probit regression lines) except occasionally at the higher dosage levels. There was some reduction in average daily consumption associated with high mortality, probably due more to intoxication than repellency.

TOXICITY STATISTICS

An alphabetical listing of common plus chemical names of compounds tested comprises table 1. Detailed toxicity statistics for each chemical and species are given in table 2, while table 3 ranks chemicals by order of toxicity for each species. Table 4 compares species sensitivity to several classes of compounds. Detailed descriptions are included with each table.

Relative Toxicities:

The term "relative toxicity," as used in this study, is the ratio of the dietary concentrations of two chemicals expected to produce a given percentage of mortality, as tested, in a specified population of birds. For example, if twice the concentration of chemical B is needed to produce the same percentage of mortality as a given concentration of chemical A, then chemical A has twice the lethal toxicity of chemical B. In general, the relative toxicity of chemical A to chemical B (at a given level of mortality) is the required concentration of B divided by that of A.

The relative toxicity of two chemicals may be expressed unconditionally as the ratio of their LC_{50}'s provided (1) the level of tolerance of the test subjects did not differ between the experiments in which the chemicals were tested, and (2) the two probit regression lines are parallel. The first condition can be assumed valid only for chemicals tested in the same completely randomized experiment. However, adjustments for tolerance differences between experiments can be made through the ratio of the LC_{50}'s of the respective dieldrin standards. The second condition, parallelism, is assumed if the slopes of the regression lines are not shown to be different at a specified level of statistical significance. If the lines are not parallel, relative toxicity will vary with dietary concentration and therefore cannot be expressed as a constant.

The relative toxicity of dieldrin (RTD) to each chemical tested is presented for each species in table 2, with a notation if parallelism was rejected. To calculate the relative toxicities of other pairs of chemicals, adjusted through the dieldrin standard, we propose using the data in table 2 according to the procedure presented in appendix 1.

There is perhaps a tendency to suppose that if one chemical is, say, twice as toxic as another, it will cause twice the mortality at a given dosage. Such a relationship does not hold true, however, as can readily be demonstrated by constructing two hypothetical dosage-mortality lines on log-probability paper. The valid comparison of toxicity is between the amounts of chemicals required to produce the same effect, and not between the percentages of mortality resulting from equal dosages.

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Estimating LC's for the General Percentage of Response:

Lethal concentrations for percentages of mortality other than the median can be estimated from the data in table 2. The computations involve transforming the LC_{50} to its common logarithm and the desired percentage of mortality to its probit, the probit of 50 percent being 5. If we let k equal the new percentage of mortality for which we wish to estimate the lethal dietary concentration (i.e., the LC_k), and b equal the particular slope value from table 2, then

$$\log LC_k = \log LC_{50} + (\text{probit } k - 5)/b$$

The antilog of $\log LC_k$ is, of course, the desired estimate. Tables for transforming percentages to probits can be found in various statistical texts, including Finney (1952).

In this study, there appeared to be little advantage to estimating response dosages other than the LC_{50}. It can be estimated more precisely than any other value, per given effort, and serves to provide efficient estimates of relative toxicity. At times, estimates of extreme values, such as the LC_5 or LC_{99}, may be important; however, such estimates are best determined from especially designed experiments, since extrapolation from a standard probit regression line can be misleading if the true regression equation has some curvature (Finney, 1952).

Utility of Toxicity Statistics:

The immediate utility of the toxicity statistics lies, perhaps, in their providing readily calculable comparisons of the short-term dietary toxicity of any two of the chemicals to a given species. Further, the slopes of the probit regression lines indicate the rate of increase in lethal hazard resulting from a proportional increase in exposure, i.e., the steeper the slope, the more rapid the increase in lethality.

Often, pesticidal contamination in the natural diet can be measured by chemical analysis of field samples. When this is possible, the degree of hazard may be predicted by direct comparison of ppm chemical in the natural diet with the lethality of dietary concentrations in laboratory studies.

When residues in the natural diet cannot be determined, short-term hazard may be estimated, as mentioned by Tucker and Crabtree (1970), by comparing the test chemical (T) with a chemical (K) known to produce mortality in the field when applied at a given rate. For example, if in the laboratory T is twice as toxic as K (i.e., the LC_{50} of T is one-half that of K), then we should expect T to produce mortality in the field if applied at one-half or more the

stipulated rate for K. Expressions of hazard that relate quantities of chemical lethal in laboratory doses to quantities of chemical applied per unit area (say, per square foot) have been suggested in the literature. The approach is necessarily a manipulation of the one immediately above; and while convenient, it cannot offer greater accuracy. Clearly, the accuracy of all such predictions will depend upon how representative the laboratory measurements are of toxicity in the field.

Multiple, interacting factors (physical, chemical, biochemical, and toxicological) determine the total hazard of a pesticide once released into the environment, as outlined in detail by Kenaga (1968). Thus findings herein should not be extrapolated beyond the limits of the protocol without caution and qualification.

DISCUSSION

Toxicity Comparisons:

More than 4500 intra-species estimates of relative toxicity can be derived from the data in table 2 in addition to those involving the dieldrin standard. Although it would be impractical to list them in this report, certain general comparisons should be noted.

Endrin was consistently the most toxic chemical as tested. It was virtually twice as toxic as any other chemical with the exception of DRC-1339 (Starlicide), which was tested only on Japanese quail. Dieldrin and the closely related aldrin were always among the six most toxic chemicals of those tested on all species, and to pheasants they were the second and third most toxic chemicals.

Organophosphates generally proved to be less toxic in the diet than aldrin or dieldrin. Of 22 organophosphates tested on bobwhite, only three (phosphamidon, fenthion, and Dasanit) were more toxic; of 23 tested on Japanese quail, only parathion and methyl parathion were more toxic; and of 23 tested on mallards, only Dasanit and diazinon were more toxic. None of 21 organophosphates was more toxic to pheasants than aldrin or dieldrin.

Data from Tucker and Crabtree (1970), who present a variety of estimates of acute toxicity using encapsulated doses, show that when chemical concentrate is administered as a single oral dose, organophosphate pesticides are generally more toxic to mallards and pheasants than either aldrin or dieldrin. Limited data for Japanese quail suggest the same tendency, but there are too few comparable data for bobwhite.

We can only theorize about the differences due to the two methodologies. Apparently organophosphate degradation in treated

feed prior to consumption was not a factor: virtually identical results were obtained when Dasanit and demeton concentrates were prepared and mixed in feed daily and when the diets were prepared at the onset of the 5-day period. It has been reported, however, that absorption of some organochlorine compounds through the gastrointestinal wall is more efficient when chemical is blended in the diet than when administered as a single dose of concentrate (Stickel et al., 1965). With organophosphate compounds we doubt that this difference is pronounced. Moreover, exposure tends to be gradual when toxicant is dispursed throughout the diet, often giving the system time to degrade the relatively unstable organophosphate compounds but not the notably stable organochlorine compounds.

Dietary LC_{50}'s for certain of the chemicals have been estimated previously. Those for six Aroclor products (polychlorinated biphenyl mixtures) given by Heath et al. (1970) were derived from the same data used herein, and slight changes represent computerized refinements. The same is true for several organochlorine pesticides, excepting technical DDT, presented by Heath et al. (1965); however, DDT estimates are derived from subsequent tests, and differences appear to be largely the result of a change in source of technical DDT. Further, poor reproducibility (heterogeniety) of DDT results may have contributed to the differences.

Gill et al. (1970) have given 5-day dietary LC_{50}'s of technical and p,p'-DDT, p,p'-DDD, and p,p'-DDE for pheasant chicks. We have tested technical DDT, a technical DDD marketed as Rhothane, and p,p'-DDE, always in the same completely randomized experiment for a given species. Gill's LC_{50}'s of DDD and DDE (522 and 1086 ppm) corresponded quite well with ours for pheasants (579 and 841 ppm), but theirs of technical DDT (935) is higher than we present here (311 ppm). We suspect a difference between the technical DDT formulations, since our technical DDT was consistently more toxic than either Rhothane or DDE to all species. Their LC_{50} of 550 ppm for p,p'-DDT corresponds better to ours for technical DDT.

DDE is generally reported to be much less toxic than DDD; and although it is probably the most abundant pesticide residue in the environment, DDE is thought to present little hazard in a lethal sense. With the exception of pheasants, we found this toxicity relationship not to hold true. Testing both chemicals simultaneously in completely randomized experiments, we derived the following LC_{50}'s for DDE and DDD: bobwhite, 825 and 2178 ppm; Japanese quail, 1355 and 3165 ppm; and mallards, 3572 and 4814 ppm. Thus DDE was about 2 1/2 times as toxic as DDD to both bobwhite and Japanese quail and almost 1 1/2 times as toxic to mallards.

There appears to be a definite reversal in the toxicity of diazinon and Guthion to birds compared to mammals. Both in dietary and in encapsulated doses (Tucker and Crabtree, 1970) diazinon was from seven to 38 times as toxic as Guthion to mallards and pheasants; however, oral LD_{50}'s for rats (Gaines, 1960 and 1969) show Guthion to be at least 19 times as toxic as diazinon.

Herbicides were generally of a low order of toxicity. Only diquat and paraquat consistently produced mortality, the latter being the most toxic herbicide tested. Otherwise most LC_{50}'s were greater than 5000 ppm, and in many tests there was no mortality at that concentration.

Comparisons of Species Sensitivity:

There were obvious inconsistencies in the relative sensitivity of the four species to various chemicals, as is shown in table 4. Mallards generally tolerated the highest dietary concentrations, but to toxaphene, terepene polychlorinates, and Ceresan M, the mallard was the most sensitive species. Bobwhites, Japanese quail, and pheasants were similar in overall sensitivity to organochlorine compounds; but to most organophosphates, bobwhites and Japanese quail were more sensitive than pheasants. There was a major difference in the sensitivity of bobwhites and Japanese quail to the Aroclors, bobwhites being the most sensitive of the four species and Japanese quail the least sensitive, as reported earlier (Heath et al., 1970); otherwise there was no discernible difference in the overall sensitivity of the two species.

Such inconsistencies in relative sensitivity among four species, especially when three are gallinacious species, strongly suggest that certain ones not tested will be much more sensitive to the different chemicals. Studies using mature house sparrows, blue jays, cardinals, and bobwhites (Hill, 1971) also show considerable variation in species sensitivity.

The 8-Day Mortality Pattern:

Death rarely occurred during the first day of dosage and only occasionally on the second day. Mortality tended to accelerate from the third through fifth day of dosage, subsided on the first day of untreated diet (i.e., the sixth day), and deaths rarely occurred thereafter. The pattern of mortality was generally similar for all effective dosages, the level of mortality being a function of a particular dosage.

Two very toxic chemicals - Ceresan M and Dasanit - produced notable exceptions to this pattern, especially in tests with mallards. Ceresan M produced little mortality during the 5 days of dosage, although the birds had become severly intoxicated by the fifth day. Mortality began on the first day of untreated diet and was so heavy during the second and third days that mortality on untreated diet was recorded for an additional six days. Only one of eight birds survived at 60 ppm (active ingredient), and none at levels of 74 ppm and higher. There were no deaths, however, at 20 ppm or among controls.

The response to Dasanit was completely opposite to that of Ceresan M. Virtually all mortality occurred during the first day, the extent of mortality depending upon dosage level; if a bird survived the initial day of dosage, it apparently became tolerant of the chemical, regardless of dosage level, and survived. As shown earlier, chemical breakdown in feed was definitely not a factor.

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REFERENCES

1. Bliss, C. I.
   1952. The Statistics of Bioassay. (Reprinted, with additions, from Vitamin Methods, Vol. II.) Academic Press, New York. Pp. 445-628.

2. Daum, R. J., and W. Killcreas.
   1966. Two computer programs for probit analysis. Bulletin of the Entomological Society of America 12 (4): 365-369.

3. Finney, D. J.
   1952. Probit Analysis, 2d ed. Cambridge University Press. 318 p.

4. Gaines, T. B.
   1960. The acute toxicity of pesticides to rats. Toxicology and Applied Pharmacology 2 (1): 88-99.

5. _____
   1969. Acute toxicity of pesticides. Toxicology and Applied Pharmacology 14 (3): 515-534.

6. Gill, J. A., B. J. Verts, and A. G. Christensen.
   1970. Toxicities of DDE and some other analogs of DDT to pheasants. Journal of Wildlife Management 34 (1): 223-226.

7. Hayes, W. J., Jr.
1967. The 90-dose LD_{50} and a chronicity factor as measures of toxicity. Toxicology and Applied Pharmacology 11 (2): 327-335.

8. Heath, R. G., J. W. Spann, and J. F. Kreitzer.
1969. Marked DDE impairment of mallard reproduction in controlled studies. Nature 224 (5214): 47-48.

9. _____, _____, _____, and C. Vance.
1970. Effects of polychlorinated biphenyls on birds. Proceedings XV International Ornithological Congress, The Hague, 30 August - 5 September, 1970. Edited K. H. Voous. E. J. Brill Publishers, Leiden.

10. _____, _____, and C. Vance.
1965. Pen studies of avian toxicology at Patuxent. P. 23-24 in U.S. Fish and Wildlife Circular 226.

11. _____, and L. F. Stickel.
1965. Protocol for testing the acute and relative toxicity of pesticides to birds. P. 18-21 in U.S. Fish and Wildlife Circular 226.

12. Hill, E. F.
1971. Toxicity of selected mosquito larvicides to some common avian species. Journal of Wildlife Management 35 (4): 757-762.

13. Kenaga, E. E.
1968. Guidelines for evaluating the properties of pesticides for safe use in the wildlife environment. Down to Earth 23 (4): 11-18.

14. Snedecor, G. W., and W. G. Cochran.
1967. Statistical Methods, 6th ed. Iowa State Univ. Press, Ames. 593 p.

15. Stickel, L. F.
1968. Organochlorine pesticides in the environment. U.S. Bureau of Sport Fisheries and Wildlife, Special Scientific Report -- Wildlife No. 119. 32 p.

16. Stickel, W. H., W. E. Dodge, W. G. Sheldon, J. B. DeWitt, and L. F. Stickel.
1965. Body condition and response to pesticides in woodcocks. Journal of Wildlife Management 29 (1): 147-155.

17. Sun, Yun-Pei.
1950. Toxicity index -- an improved method of comparing the relative toxicity of insecticides. Journal of Economic Entomology 43 (1): 45-53.

18. Tucker, R. K., and D. G. Crabtree.
1970. Handbook of toxicity of pesticides to wildlife. U.S. Bureau of Sport Fisheries and Wildlife, Resource Publication No. 84. 131 p.

19. U.S. Fish and Wildlife Service.
1964. Procedures for evaluation of toxicity of pesticides to wildlife. Pesticides Review Staff. 77 p.

20. Weil, C. S., and D. D. McCollister.
1963. Relationship between short-and long-term feeding studies in designing an effective toxicity test. Journal of Agricultural and Food Chemistry 11 (6): 486-491.

21. Wiemeyer, S. N., and R. D. Porter.
1970. DDE thins eggshells of captive American kestrels. Nature 227 (5259): 737-738.

Table 2. Median lethal concentrations (LC_{50}'s) of pesticidal chemicals in 5-day diets of second-week birds, with relative toxicities of dieldrin (RTD) when tested concurrently as a standard--cont.

CHEMICALS Species	No. concen-trations	(Birds per conc.)	LC_{50}:[a] ppm chem. in feed	(95% conf. limits)	Slope: probit on log conc.	(St. dev.)	Relative[b] toxicity of dieldrin (RTD)	(95% conf. limits)	Toxic[c] rank no.
Aroclor 1254									
Bobwhite	5	(10)	604	(410- 840)	6.379	(1.848)	20.4	(15.0 -27.7)	33
Jap. quail	8	(10)	2898	(2598-3241)	5.772	(1.364)	46.3	(39.4 -54.5)	45
Pheasant	5	(10)	1091	(968-1228)	12.174	(2.431)	21.3	(18.2 -25.0)	32
Mallard	6	(10)	2699	(2159-3309)	6.674	(1.263)	16.7	(12.7 -22.0)	31
Aroclor 1260									
Bobwhite	5	(10)	747	(577- 937)	6.211	(1.631)	25.2	(18.9 -34.4)	37
Jap. quail	7	(10)	2186	(1917-2478)	7.444	(1.439)	34.9	(29.3 -41.3)	41
Pheasant	6	(10)	1260	(1106-1433)	5.421	(2.715)	24.6	(20.8 -29.1)	35
Mallard	5	(10)	1975	(1363-2749)	4.054	(1.759)	12.2	(8.93-16.3)	27
Aroclor 1262									
Bobwhite	5	(10)	871	(702-1069)	4.037	(1.584)	29.4	(22.1 -40.8)	39
Jap. quail	7	(10)	2291	(2038-2575)	7.552	(1.501)	36.6	(31.0 -43.2)	43
Pheasant	5	(10)	1234	(1086-1402)	13.518	(2.547)	24.1	(20.5 -28.5)	34
Mallard	6	(10)	3008	(2461-3634)	2.351	(1.226)	18.6	(14.2 -24.5)	36
atrazine									
Bobwhite	3	(10)	>5000[d]						
Jap. quail	3	(14)	>5000	7% mort. @ 5000					
Pheasant	3	(8)	>5000						
Mallard	3	(10)	>5000	30% mort. @ 5000					

Table 2. Median lethal concentrations (LC_{50}'s) of pesticidal chemicals in 5-day diets of second-week birds, with relative toxicities of dieldrin (RTD) when tested concurrently as a standard--cont.

CHEMICALS Species	No. concen-trations	(Birds per conc.)	LC_{50}:[a] ppm chem. in feed	(95% conf. limits)	Slope: probit on log conc.	(St. dev.)	Relative[b] toxicity of dieldrin (RTD)	(95% conf. limits)	Toxic[c] rank no.
2,4-DB									
Bobwhite	3	(10)	>5000	40% mort. @ 5000					
Jap. quail	3	(12)	>5000[d]						
Pheasant	3	(10)	>5000						
Mallard	—								
DDD									
Bobwhite	5	(7)	2178	(1835-2584)	9.379	(2.497)	59.2	(47.7 -74.2)	45
Jap. quail	4	(12)	3165	(2534-3978)	4.613	(1.780)	56.2	(43.0 -74.0)	47
Pheasant	4	(7)	579	(499- 668)	11.956	(3.360)	13.5	(11.1 -16.4)	26
Mallard	6	(10)	4814	(3451-7054)	3.455	(1.343)	24.7	(17.9 -36.1)	40
DDE									
Bobwhite	5	(7)	825	(697- 976)	8.132	(2.436)	22.5	(18.1 -28.0)	35
Jap. quail	6	(12)	1355	(1111-1648)	6.469	(1.205)	24.1	(18.6 -31.0)	36
Pheasant	5	(7)	841	(731- 967)	12.198	(2.969)	19.6	(16.3 -23.7)	29
Mallard	6	(10)	3572	(2811-4669)	3.709	(1.069)	18.4	(13.6 -25.7)	35
DDT									
Bobwhite	5	(7)	611	(514- 724)	7.357	(2.489)	16.6	(13.4 -20.8)	28
Jap. quail	6	(12)	568	(470- 687)	4.770	(1.367)	10.1	(7.86-13.0)	28
Pheasant	4	(7)	311	(256- 374)	10.982	(4.644)	7.27	(5.88- 8.9)	16
Mallard	6	(10)	1869	(1500-2372)	3.896	(0.996)	9.60	(7.14-13.3)	23

Table 2. Median lethal concentrations (LC_{50}'s) of pesticidal chemicals in 5-day diets of second-week birds, with relative toxicities of dieldrin (RTD) when tested concurrently as a standard--cont.

CHEMICALS Species	No. concen-trations	(Birds per conc.)	LC_{50}:[a] ppm chem. in feed	(95% conf. limits)	Slope: probit on log conc.	(St. dev.)	Relative[b] toxicity of dieldrin (RTD)	(95% conf. limits)	Toxic[c] rank no.
trichlorfon									
Bobwhite	5	(10)	720	(591- 871)	5.604	(2.677)	18.3	(14.5 -23.3)	30
Jap. quail	6	(10)	1901	(1601-2255)	4.898	(1.108)	35.6	(28.0 -45.4)	42
Pheasant	–								
Mallard	–								
Zectran									
Bobwhite	–								
Jap. quail	3	(14)	≈500	–			≈ 8.9	–	27
Pheasant	5	(9)	846	(724- 985)	6.558	(1.936)	17.2	(14.0 -21.0)	28
Mallard	6	(11)	334	(268- 412)	3.041	(0.921)	2.39	(1.79- 3.21)	10

[a] LC_{50}: ppm chemical in ad libitum diet expected to produce 50% mortality in 8 days comprising 5 days of toxic diet followed by 3 days of untreated diet.

[b] Relative toxicity of dieldrin (RTD) read: "Dieldrin is x times as toxic as the given chemical as tested." See text for use of RTD's to compare toxicities of any two chemicals.

[c] Toxic rank number: Numerical position in listing of chemicals ranked in descending order of toxicity (see table 3). The larger the toxic rank number or the RTD, the less toxic the chemical.

[d] No mortality at 5000 ppm unless specified.

[e] Relative toxicity of dieldrin applies only at LC_{50}, since probit slope is significantly different (P=0.05) from that of dieldrin.