# Pesticide Effects on the Fecundity of the Gray Partridge

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Chlorinated hydrocarbons and organophosphates have been considered a primary factor in the decline of various animal species. Reproductive success, as measured by progeny production, appears to be depressed in affected species (1).

This investigation was designed to determine the influence of subtoxic levels of the pesticides, parathion and dieldrin, on fecundity, embryonic development, hatchability, growth and livability of Gray partridge (Perdix perdix); pesticides were administered in the feed prior to and during the breeding season.

Rudd and Genelly (2) reported that all species of birds that had been observed were sensitive to the pesticide dieldrin. They also reported that sensitivity varied with the age of the animal within the species. The lethal dosage of dieldrin varies widely from one animal to another (3, 12). DeWitt (4) observed that sensitivity to dieldrin differed between sexes within a species.

Dieldrin is deposited in the body fat (lipid fraction) of egg laying species (5) with residues being proportional to the level and duration of exposure and the fat content of the tissue. DeWitt (6) noted that 20 ppm dieldrin added to the diet of quail caused a reduction of feed consumption in some groups.

Stadelman et al. (7) observed that Leghorn hens fed 0.1 to 0.15 ppm of dieldrin in the diet for 14 days had no significant residues in their eggs or body fat. Cannon et al. (5) reported that Leghorn

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The Shell Chemical Company of New York supplied technical dieldrin, and Velsicol Chemical Corporation of Chicago provided analytical grade parathion.

laying hens receiving 0.1 ppm dieldrin in their diets had residues of dieldrin in their eggs which were barely measurable after 9 weeks, but after 12 weeks the fat in these hens showed high levels of dieldrin. Stadelman et al. (7) maintained Leghorn layers on 10-15 ppm of dieldrin for 5 days and found high residue levels in the fat and eggs for 26 weeks after termination of the pesticide treatment.

Eggs from quail fed 10 ppm dieldrin had 50 percent lower hatchability than control eggs (4); however, egg production and fertility were not markedly influenced. Chick survival from hatch to three weeks of age was 43.9 and 87.5 percent for dieldrin and control groups, respectively.

Chronic poisoning from parathion is seldom observed as the residues of parathion quickly disappear and continued ingestion at sublethal levels does not produce serious injury (2). Normal feed consumption, weight gain and survival were observed in rats fed diets containing 100 ppm parathion. Barnett (8) reported that pheasants fed diets containing 100 or 1,000 ppm of parathion declined in total body weight and exhibited decreased gonadal activity. Parathion was not detectable in the liver after 39 days of these treatments. Similar weight loss symptoms were observed (9) when Japanese quail (Coturnix coturnix Japonica) were fed 5.0 ppm of the organophosphate Azodrin.

## METHODS AND MATERIALS

During late April mature Gray partridge were permitted to self-select mates, then transferred to 2.5 x 5m turf floor breeding pens. The breeding pen complex consisted of 32 pens, arranged in paired adjacent units of 16 pens; each pen opened into a 5.0 x 20m alley. The entire pen complex was covered with poultry netting and snow fencing to provide shade and protection. Each breeding pen was equipped with 0.2 x 1.0m shelter board. To minimize interpen interference, a 25.4 cm high solid wood divider formed the perimeter of each pen.

On May 1, 11 of the 32 pens were randomly selected and placed on a game bird ration containing 3 ppm of technical dieldrin. Another group of 11 randomly selected pairs was fed the same ration containing 8 ppm of reference analytical grade ethyl parathion. The remaining 10 pens were maintained on the basal ration, constituting the control. All rations were pelleted into 3.2 mm pellets. Feed and water were supplied ad libitum. Dietary pesticide levels were determined via feeding trials by the Bureau of Sport Fisheries and Wildlife, Denver Wildlife Research Center, Denver, Colorado, as those 90-day dietary levels which did not produce changes in body weight, feed consumption nor external physical symptoms indicative of toxicity.

Eggs were analyzed for residues by the procedure described in FDA Pesticide Analytical Manual, 1969, Volume 1, part 162. Qualitative and quantitative measurements were obtained by gas liquid chromatography employing both electron capture and flame photometric detectors for parathion and electron capture for dieldrin.

Egg production, fertility and hatchability records were maintained for each pair of birds. Eggs were gathered daily, marked for identification and stored at 16±2° C. Eggs were incubated weekly at 37.5±0.5° C. with a relative humidity of 53 percent. After 20 days of incubation the eggs were transferred to a hatcher. Temperature was reduced to 36.9° C. and the relative humidity increased to 64 percent. Hatches were removed at the end of the 24th day of incubation.

All unhatched eggs were opened and examined. Eggs lacking development of the area pellucida and area opaca were classed as infertile. The stage of development of all dead embryos was estimated; 1-13 day embryos were classified as "early dead"; 14-24 day embryos that did not pip were classed as "dead in shell." Hatchability was expressed as the percentage of fertile eggs set.

Hatched chicks were weighed, wing banded, randomized by parental treatment and placed in brooding batteries in groups of 25 to 30 chicks each. Temperature was initially maintained at 35° C. and lowered 2.8° C. weekly for four weeks. The chicks were fed turkey starter until six weeks of age when they were fed pelleted game bird ration. Weekly individual body weight records were maintained for the first six weeks for 63 controls, 43 dieldrin and 43 parathion chicks.

## RESULTS AND DISCUSSION

Average egg production during the laying season for the control, dieldrin and parathion groups was 17.9, 13.6 and 18.3 eggs per hen, respectively. Maximum production from a single hen was 54 eggs; four of the 32 pairs failed to produce any eggs. Depressed egg production as observed for birds fed dieldrin is in agreement with Rudd and Genelly (2) who reported markedly lowered egg production from pheasant hens fed 25 to 50 ppm dieldrin. Although dieldrin produced an average of 4.3 eggs less per pair than controls, within group variances precluded statistically significant differences (P=.05).

The mean fertility for all groups and all hatches was 86.6 percent. Average fertility of control, dieldrin and parathion eggs for the season was 85.5, 83.2 and 90.0 percent, respectively (Table 1). These fertility percentages did not differ statistically from each other. Fertility was not significantly affected by dieldrin or parathion ingestion at the levels employed.

TABLE 1

The influence of feeding parathion or dieldrin on fertility and hatchability of Gray partridge eggs.

| Treatment | No. of pairs tested | No. of eggs set | % Fertility | % Early dead | % Pips | % Dead in shell | % Hatch-ability (fertile eggs) |
|---|---|---|---|---|---|---|---|
| Control | 10 | 159 | 85.5 | 7.3 | 6.5 | 4.4 | 81.8 |
| Dieldrin | 11 | 107 | 83.2 | 6.8 | 7.5 | 12.2 | 73.5 |
| Parathion | 11 | 197 | 90.0 | 10.2 | 7.5 | 14.4 | 67.9 |

Total prenatal mortalities of 18.2, 26.5 and 32.1 percent were determined for fertile control, dieldrin and parathion eggs.

Percent pipped, but unhatched, eggs varied slightly between the three treatment groups (Table 1). The average percent pipped but unhatched eggs for control, dieldrin and parathion groups were 6.8, 7.5 and 7.5, respectively. This uniformity of results suggests that pesticide treatments were not accountable for any significant decrease in the ability of viable full-term embryos to emerge from the shell.

Hatchability of fertile eggs (HFE) for control, dieldrin and parathion groups was 81.8, 73.5 and 67.9 percent, respectively. The average HFE for all groups was 74.7 percent. Chi square comparisons of these hatchability data approached, but did not attain, statistical significance ($P<.05$).

Although not significantly different from control data, depression of embryo survival and hatching ability, attributable to the progressive debility of embryos produced by pesticide treated parent stock, is indicated in these responses (Table 1).

Eggs from the dieldrin treatment did not show an increased percentage of early dead embryos (Table 1), but the percent dead in the shell was quite high and could be attributed to the dieldrin residues found in the eggs (Table 2). The decline in hatchability of the dieldrin group was attributed to dieldrin and DDE residue (Table 2).

TABLE 2

Pesticide residue (ppm) in egg contents of Gray partridge fed dieldrin or parathion.[1]

| Pesticide treatment | Egg number | DDE | Dieldrin | Parathion |
|---|---|---|---|---|
| Control | 1 | 0.1 | 0.1 | Not Found |
| | 2 | 0.1 | 0.1 | Not Found |
| | 3 | 0.1 | Not Found | Not Found |
| Dieldrin 3.0 ppm | 1 | 0.2 | 2.0 | No Analysis |
| | 2 | 0.1 | 1.0 | No Analysis |
| | 3 | 0.1 | 1.6 | No Analysis |
| Parathion 8.0 ppm | 1 | No Analysis | No Analysis | Not Found |
| | 2 | No Analysis | No Analysis | Not Found |

[1] Residue analysis conducted by Denver Wildlife Research Center, Bureau of Sport Fisheries and Wildlife, Denver, Colorado.

Parathion residues were not detected in the eggs; therefore, any comment concerning its possible effect on embryo viability is speculative. However, this treatment group had high percentages of both early and late dead embryos. At 17.5 days of development, the domestic chick embryo reduces its metabolic rate, which is later accelerated (10). Oxygen uptake and carbon dioxide output data demonstrate increased metabolic activity from the 18th day of incubation through hatching (11). Any modification or impairment of these innate metabolic rate changes by internal or external factors is detrimental to embryo viability. It is hypothesized that unidentified parathion degradation products present in the eggs had an adverse effect on this metabolism and are accountable for the high percentage of "dead in shell" embryos. It is also possible that parathion may have caused aberrant metabolism in the formation of the eggs which impaired embryo viability during late stages of development. Similar effects earlier in embryonic development could account for early embryonic deaths.

Average weekly body weights of 63 control, 43 dieldrin and 43 parathion chicks hatched from the second through the seventh hatches were tabulated according to parental pesticide treatments. No alterations in growth rate, attributable to parental pesticide treatment, were indicated in these comparisons. Mortality to six weeks of age for the combined hatches was 7, 6 and 5 percent for control, parathion and dieldrin treatment progeny. These low mortality levels and comparable weight gains in all groups indicate that the levels of dieldrin and contaminant DDE residue determined in the eggs were not detrimental to livability or growth of surviving chicks.

Although few of the individual factors investigated attained statistical levels of significance, progressive debility of

physiological fitness, particularly progeny production, is strongly indicated in these responses to pesticide ingestion at sublethal levels.

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