# DDE: Interference with Extra-Renal Salt Excretion in the Mallard

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One apparent common denominator among the avian species showing dramatic population declines attributed to environmental pollutants is the presence of a functional salt gland. Despite the adaptive survival value of this gland, the effects of such pollutants on avian salt gland function have not been studied.

The existence, structure, and development of the salt, or nasal, gland has been reported for a number of avian species (BOCK 1958, MARPLES 1932, SCHMIDT-NIELSEN and FÄNGE 1958, SCHMIDT-NIELSEN et al. 1958), but it was not until the late 1950's that its function as a major salt-secreting organ was described (SCHMIDT-NIELSEN et al. 1958). These paired glands are the main route of sodium chloride excretion in marine birds (SCHMIDT-NIELSEN and FÄNGE 1958). It follows, then, that suppression of salt gland function could be detrimental to survival in habitats of high salinity. Recent studies have suggested that DDT and/or its metabolites might produce such alterations. DDT was found to disrupt osmoregulatory events in marine teleosts (JANICKI and KINTER 1971 a) and eels, Anguilla rostrata (JANICKI and KINTER 1971 b). DDT also inhibited activity of adenosine triphosphatase in fish tissue homogenates (CUTCOMP et al. 1971); ATPases are believed to be important in the transport of sodium across cell membranes (SKOU 1965), thereby functioning in part to preserve tissue hypotonicity during osmoregulatory processes. The following preliminary study was, therefore, conducted to determine the effects of DDE* on salt gland function in the mallard, Anas platyrhynchos.

## Experimental

Five-month-old pen-reared male mallards from stock lines of the Denver Wildlife Research Center were maintained on either fresh water or 1 percent salt water beginning 5 days before the start of DDE feeding. Food and water were provided ad libitum throughout the study. Groups of 12 birds each were fed commercial game bird pellets to which 0, 10, 100, or 1000 parts per million (ppm) of DDE had been added. Three birds from each treatment group were then challenged with a concentrated salt

*DDE = Dichlorodiphenyldichloroethylene



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solution after 1, 3, 6, and 9 days of DDE feeding. The salt challenge was administered to each bird both intraperitoneally (12 ml of a 10 percent solution) and intravenously (3 ml of a 5 percent solution via the brachial vein). The birds were then restrained on specially designed inclined tables, and discharges from the salt glands were collected in graduated 50-ml centrifuge tubes. The amount of discharge was recorded at 15-minute intervals for 2-1/2 hours, after which each bird was weighed and killed by decapitation, and the salt glands and adrenals were removed and weighed. The entire experiment was randomized, including assignment of birds to treatments, sequence of bird selection by test day, and bird location on the restraining tables.

## Results and Discussion

The chemical composition of salt gland secretion was not altered by DDE. As in other species, this secretion was primarily sodium chloride (PEAKER 1971). Other experimental results are summarized in Table 1. Birds maintained on 1 percent salt water

TABLE 1

| DDE in diet (ppm) | Cumulative secretion by salt glands at: 1/2 hr Mean (ml) | 1/2 hr % of controls | 2-1/2 hr Mean (ml) | 2-1/2 hr % of controls | Average total weight of L and R glands (g) Salt gland | Adrenal gland | Change in body weight (g) |
|---|---|---|---|---|---|---|---|
| Fresh-water birds (n = 12 per group) | | | | | | | |
| 0 | 1.4 | 100 | 5.2 | 100 | 0.23 | 0.09 | +55 |
| 10 | <0.1 | 3 | 1.9 | 36 | 0.21 | 0.10 | -41 |
| 100 | 0.1 | 8 | 3.5 | 67 | 0.22 | 0.10 | -64 |
| 1000 | 0.5 | 36 | 2.9 | 56 | 0.20 | 0.11 | -94 |
| Salt-water birds (n = 12 per group) | | | | | | | |
| 0 | 0.8 | 100 | 9.7 | 100 | 0.42 | 0.10 | -20 |
| 10 | 1.0 | 125 | 10.0 | 103 | 0.46 | 0.11 | -28 |
| 100 | 1.0 | 125 | 7.7 | 79 | 0.43 | 0.11 | -67 |
| 1000 | 0.8 | 100 | 10.7 | 110 | 0.43 | 0.11 | -71 |

Effect of DDE on salt gland secretion and tissue weight of mallards maintained on either fresh or 1 percent salt water and challenged by inoculation with concentrated salt solutions.

before salt challenge excreted about twice as much sodium chloride and had salt glands about twice as large as fresh-water birds. DDE had no effect on the amount of sodium chloride excreted by salt-water birds, but markedly reduced excretion in fresh-water birds.

A plot of salt gland secretion in fresh-water birds (Figure 1) indicates the magnitude of suppression by DDE.

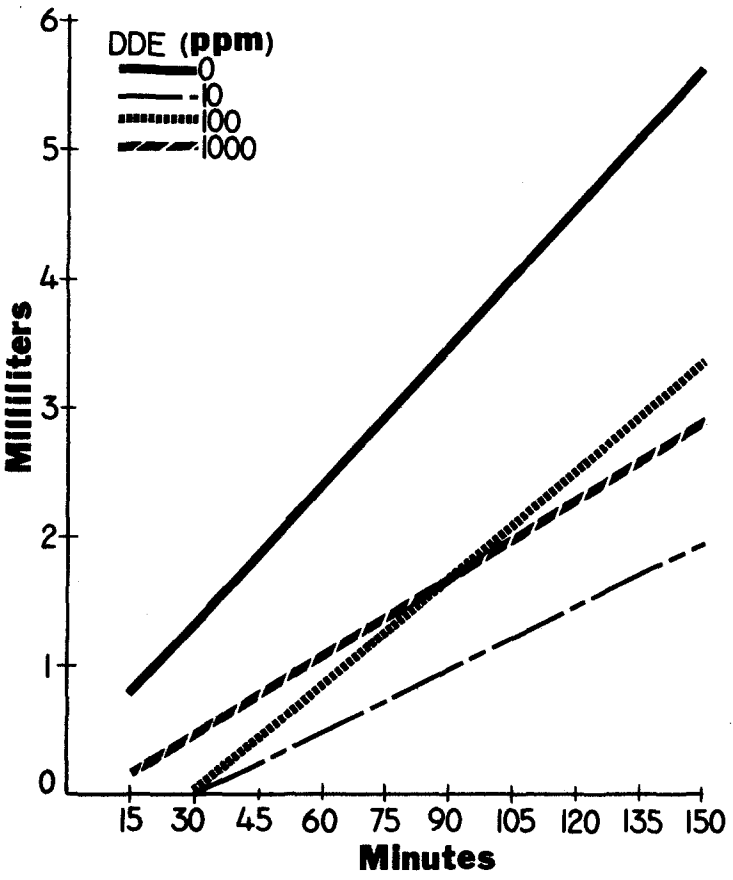


Figure 1. Rate of secretion from the salt glands of mallards maintained on fresh water and various levels of DDE in the diet (12 birds per treatment level). Accumulated secretion from each bird was measured at 15-minute intervals after salt challenge.

Orthogonal comparisons of these data showed a linear relationship between excretion and time for all treatment levels. Homogeneity of the regression coefficients for the different treatment levels was statistically rejected and the rate of salt gland secretion for control birds (0 ppm) was found to be significantly greater ($p < 0.01$) than that for birds fed any level of DDE (10, 100, or 1000 ppm). These data are interpreted to indicate an apparent all-or-none effect of DDE on salt gland function, with 10 ppm in the diet being sufficient to suppress the volume of salt gland secretion in birds not previously exposed to salt. DDE had its greatest effect during the first 30 minutes following salt challenge (Table 1).

DDE had no effect on salt gland or adrenal gland weight in either salt- or fresh-water birds, but increasing levels of DDE caused increasing losses in body weight regardless of salt exposure (0 or 1 percent). These losses (Table 1) may have been a result of reduced food consumption with increasing DDE levels, but this would not be determined since food consumption data were not recorded.

The data presented represent averages of all 12 birds in each group, even though these data were collected on four different days (1, 3, 6, and 9 days after the start of DDE feeding). With three-bird subgroups, variation among birds was great enough to mask any differences in salt gland secretion due to duration of DDE exposure; larger sample sizes will be necessary to measure this parameter.

It appears from these data that sublethal levels of DDE have no effect on extra-renal elimination of salt in mallards whose salt glands have been previously stimulated by low-level (1 percent) salt exposure. However, DDE can apparently suppress salt gland secretion in mature mallards not previously exposed to salt. The significance of these data lies in the fact that the salt gland is the major route of salt elimination for birds living in marine habitats (SCHMIDT-NIELSEN and FÄNGE 1958). Interference with its function in juveniles or other birds not previously (or perhaps not recently) exposed to salt could result in inability to eliminate toxic levels of salt taken in while feeding (invertebrates, for example, have salt concentrations equal to their surroundings). This type of phenomenon may be responsible for some of the recent unexplained die-offs of marine birds in which the evidence has failed to incriminate either infectious disease or environmental pollutants (BOURNE 1971, WATSON 1970).

## Summary

The effects of DDE on the function of the salt gland, the main route of sodium chloride excretion in marine birds, were investigated in mature mallards, Anas platyrhynchos, maintained

on either fresh water or 1 percent salt water and given 0, 10, 100, or 1000 ppm DDE in the diet (12 birds per level). The rate of sodium chloride excretion by the salt gland following injections of concentrated salt solutions was not reduced (from that of controls) in DDE-treated birds maintained on salt water, but was significantly reduced in DDE-treated birds not previously given salt.


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