

South Louisiana Crude Oil and DDE in the Diet of Mallard Hens: Effects on Reproduction and Duckling Survival

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The reproductive performance of precocial birds fed an environmental contaminant often is measured in terms of egg production, fertility, hatchability, eggshell thickness, and survival of unstressed young. However, no one, to our knowledge, has examined the ability of offspring, produced by females fed an environmental pollutant, to survive under energetically poor conditions. Young precocial birds often are subjected to chilling and starvation upon hatching and the time immediately post-hatching is probably the most crucial period in the ultimate determination of the number of new individuals recruited into a population (KEAR 1965, KOSKIMIES and LAHTI 1964, MARCSTROM 1966, MEDENHALL 1979, REED 1975).

We selected crude oil and DDE as environmental pollutants for study because of their widespread occurrence and significance in the environment (OHLENDORF et al. 1979, WHITE 1979, WOLFE 1977). South Louisiana crude oil (SLCO) and DDE fed to mallard hens have been shown to cause significant declines in reproductive success by decreasing egg production, hatchability, and eggshell thickness (HEATH et al. 1969, HOLMES et al. 1978). In this paper we document, not only the effects of dietary SLCO and DDE upon the reproductive performance of mallard hens, but also the reduced ability of offspring produced by these hens to survive under energetically poor conditions.

METHODS AND MATERIALS

We maintained 67 mallard hens (McGraw Wildlife Foundation, Dundee, Ill.) in poultry batteries at 10 to 25° C with six hours light commencing at 6:30 a.m. beginning 29 January 1979. The hens were provided with free access to untreated food (Purina Game Bird Breeder Layena, St. Louis, Mo.) and water until 8 February 1979 when two groups of 21 hens were assigned at random to receive either a diet containing 10 ppm DDE or a diet containing SLCO in a concentration of 2% (Amer. Pet. Inst. Ref. Oil No. 2; DDE, Patuxent Wildlife Research Center). The remaining 25 hens continued to receive untreated food and served as controls. To induce egg production, the light cycle was increased 1 h per day beginning 17 March 1979 until the hens were exposed to 16 h light daily.

Beginning 7 April 1979, each group of hens was released with 10 untreated mallard drakes once every 7 days to insure continued egg fertility. Eggs were collected daily and identified by hen and date. Alternate eggs from each hen were kept to provide other data including eggshell thickness (measured with Starett 1010 micrometer). Eggs laid after first breeding were set weekly and candled after 7 days to provide data on fertility and hatchability. Last eggs were collected 24 May 1979. We determined the ability of ducklings to survive under energetically poor conditions by placing 1 h old ducklings in individual cages in an environmental chamber at 20° C. KOSKIMIES and LAHTI (1964) found that energy expenditure in 0-1 day-old ducklings was approximately 3.5 times basal metabolic rate at this temperature. No food or water was provided. Total time alive was recorded for each duckling.

RESULTS

Reproductive success was significantly affected by dietary treatment of hens with either SLCO or DDE (Table 1).

TABLE 1

Reproductive success of mallard hens fed either a control diet, a diet containing 10 ppm DDE or a diet containing 2% South Louisiana crude oil. Values are means \pm 1 standard error.

	Diet		
	Control	DDE (10ppm)	SLCO (2%)
Hens			
Median date of first egg	8 April	14 April*	23 April*
N (hens)	(24)	(20)	(18)
Egg production (eggs per 100 hen-days)	69.2 \pm 4.1	71.6 \pm 4.8	43.9 \pm 3.0*
N (hens:hen-days)	(24:867)	(20:521)	(18:426)
Eggs			
Eggshell thickness (mm x 10)	37 \pm 0.1	35 \pm 0.2*	31 \pm 0.3*
N (eggs)	(307)	(187)	(108)
Hatchability (% of fertile eggs hatched)	82.6 \pm 4.2	64.2 \pm 7.1*	45.5 \pm 8.8*
N (hens:eggs)	(24:109)	(19:109)	(11:44)
*P<0.05 Treated vs Control Diets			

The onset of laying was delayed for hens fed either the SLCO or DDE diets when compared with that of hens fed the control diet ($P < 0.05$ Kruskal-Wallis non-parametric analysis of variance). Hens fed the SLCO diet (SLCO hens) produced $36.6 \pm 7.4\%$ fewer eggs, but egg production by hens fed DDE (DDE hens) did not differ, when compared with hens fed the control diet (control hens). Egg fertility was not affected by either SLCO or DDE. Hatchability, however, was $22.2 \pm 10.0\%$ lower for eggs laid by DDE hens and $45.0 \pm 11.3\%$ lower for eggs laid by SLCO hens when compared with that of control hens ($P < 0.05$) (Analyses follow SNEDECOR and COCHRAN 1967:221,515). DDE and SLCO hens laid eggs with 5 and 16% thinner shells, respectively, than controls ($P < 0.05$ Duncans' multiple range test). Oil-induced eggshell thinning was greater than DDE-induced thinning ($P < 0.05$). Mallard hens fed a diet containing 3% SLCO for 150 days produced eggs with shells 33% thinner than controls (HOLMES et al. 1978). HEATH et al. (1969) reported an 8% reduction in eggshell thickness when mallard hens were fed 10 ppm DDE for one season and an 11% reduction after two seasons.

Ducklings produced by DDE (DDE ducklings) or SLCO hens (SLCO ducklings) survived 5.1 and 9.7 h less, respectively, than did ducklings produced by hens fed the control diet ($P < 0.05$ analysis of variance). A significantly greater proportion of ducklings from hens fed the treated diets were unable to initiate body temperature regulation ($P = 0.001$) ($\chi^2 = 13.573$) (Table 2). We could class a duckling as to its thermoregulatory ability within 1 h after being placed into the environmental chamber. Ducklings that were classed as thermoregulators had cloacal temperatures between $34-38^\circ\text{C}$, were active and vocal, and had "fluffed" down feathers in response to the cold. By contrast, ducklings classed as non-thermoregulators had cloacal temperatures between $22-25^\circ\text{C}$, exhibited a complete loss of coordinated motor activity, were non-vocal, and down feathers, although dry, remained matted together and failed to develop normally.

Both survival time and the rate of body weight loss were heterogeneous with respect to thermoregulatory ability and treatment (Table 2). Among all three groups, ducklings able to thermoregulate lost weight faster and survived longer than ducklings unable to thermoregulate. The SLCO and DDE ducklings that did thermoregulate, however, lost weight faster and died sooner ($P < 0.05$) than control ducklings able to thermoregulate.

DISCUSSION

These data confirm the previously established results that crude oil and DDE affect the onset of laying, egg production, hatchability, and eggshell thickness. Previous studies, however, have not shown that either crude oil or DDE decreases duckling survival.

TABLE 2

Survival time and rate of body weight loss according to thermoregulatory ability of ducklings hatched from eggs laid by hens fed either a control, SLCO (2% w/w) or DDE (10 ppm) diet. Ducklings were weighed when 1 h old and placed in an environmental chamber in individual cages at 20° C. No food or water was provided. Values are the mean \pm 1 standard error.

Diet of Hen	DID NOT THERMOREGULATE				DID THERMOREGULATE			
	N	Body Weight Loss (g/h)	Survival Time (h)		N	Body Weight Loss (g/h)	Survival Time (h)	
Control	27	0.068 \pm 0.007	44.55 \pm 2.53		63	0.252 \pm 0.004	65.60 \pm 0.98	
SLCO (2%)	14	0.067 \pm 0.011	46.26 \pm 5.00		6	0.288 \pm 0.025	57.53 \pm 3.81*	
DDE (10 ppm)	35	0.074 \pm 0.007	47.75 \pm 2.54		35	0.277 \pm 0.008*	60.64 \pm 2.11*	

*P<0.05 Treated vs Control Diets

Our data show that either SLCO or DDE fed to mallard hens produced two effects in their ducklings: 1) a greater proportion failed to initiate maintenance of a functional body temperature in response to temperature stress and 2) the metabolic rate of ducklings that did maintain a functional body temperature was increased. Both of these effects resulted in a decrease in survival time. The mechanisms for these effects are unknown. Perhaps SLCO or DDE slows the development of neural and endocrine processes, processes necessary for an initial endothermic response. The inability of 30% of our control ducklings to thermoregulate indicates that normal ducklings do not hatch with equal endothermic ability. HEINZ (1976) found that unstressed ducklings from parents fed 3 ppm DDE were hyper-responsive to maternal calls and traveled shorter distances from frightening stimuli. These abnormal behavioral responses indicate that neural processes were affected by DDE. Mallard ducklings fed diets containing 0.25, 2.5, or 5.0% SLCO also traveled shorter distances from frightening stimuli (SZARO et al. 1978).

Both DDT and SLCO have been shown to increase the activity of avian hepatic mixed function oxidase enzymes (MILLER et al. 1978, LUSTICK et al. 1972). SLCO in the diet of Pekin ducks has also been shown to cause hypertrophy of the thyroid gland (HOLMES et al. 1978). Perhaps these kinds of physiological responses are responsible for the greater metabolic rate of thermoregulating ducklings hatched from eggs laid by treated hens. DDT (10 ppm) in the diet of bobwhite quail (Colinus virginianus) has been shown to increase their metabolic rate (LUSTICK et al. 1972, PETERLE et al. 1974).

Whatever the mechanisms, these data demonstrate that the effects of DDE and SLCO on reproductive output of birds in natural systems may be more severe than that demonstrated by previous studies.

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