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SHORT-TERM EFFECTS OF OIL INGESTION ON AMERICAN KESTRELS (*FALCO SPARVERIUS*)

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Abstract: The Mexican Ixtoc oil well blowout resulted in extensive oil contamination along the Texas Gulf coast. This oil posed a potential hazard to migrating birds including the endangered peregrine falcon (*Falco peregrinus*). Laboratory tests with the American kestrel (*Falco sparverius*) indicated that the oil:water mixture gathered at the surface of the blowout site posed little acute hazard to falcons.

INTRODUCTION

The Ixtoc I well blowout on 3 June 1979 in the Bay of Campeche, Mexico, resulted in contamination of the Texas Gulf coast, and Padre Island in particular, with crude oil. Since this is a major migratory route and resting area for North American peregrine falcons (*Falco peregrinus*), there was concern regarding the potential hazard to migrating peregrines which might consume oiled prey items.

An evaluation of the effects of crude oil ingestion on birds of prey has not been done, but toxicity studies with such diverse species as Cassin's auklets, *Ptychoramphus aleuticus* (Ainley and Morrel, 1978), Pekin ducklings (Crocker et al., 1974), domestic chickens, domestic ducklings, and young herring gulls, *Larus argentatus* (Gorman and Sims, 1978) have resulted in no mortality. Hartung and Hunt (1966) tested several industrial oils on ducks kept under optimal conditions and reported that only a cutting oil additive caused mortality.

Several workers have reported sublethal effects of oil ingestion in birds. Szaro et al. (1978) fed up to 5% south Louisiana crude oil to mallard (*Anas platyrhynchos*) ducklings from hatching to 8 weeks of age. They reported hepatocyte hypertrophy, bile duct proliferation, renal tubular degeneration, elevated plasma alanine amino-

transferase and ornithine carbamyl transferase activities, and reduced packed cell volumes. Refined oils tested by Hartung and Hunt (1966) produced lipid pneumonia, gastrointestinal irritation, fatty livers, adrenal cortical hyperplasia, and reduced packed cell volumes. Holmes et al. (1978) found that stressed Pekin ducks fed petroleum-contaminated food exhibited involution of lymphoepithelial tissues.

The present study was initiated to determine the hazards of Ixtoc I oil ingestion to peregrine falcons through the use of a surrogate species, the American kestrel (*Falco sparverius*). The kestrel is closely related and has served as a model in previous studies. Mortality, histopathological, hematological and clinical pathological effects were studied.

MATERIALS AND METHODS

Forty juvenile kestrels from the captive colony maintained at the Patuxent Wildlife Research Center (20 of each sex) were randomly assigned to 1 m³ suspended vinyl-coated wire cages and acclimated for 2 weeks. Birds were then randomly assigned to one of three diets: 3.0% crude oil (eight males, eight females), 0.3% crude oil (eight males, eight females), control (four males, four females). Diets were prepared by mixing Ixtoc I oil (IXO) obtained from the U.S. Environmental Protection Agency (collected at the sur-

face of the blowout; about 50% seawater) in a commercial bird of prey diet.^[1] This diet is known to be adequate for kestrel survival and reproduction and is nutritionally complete. The amount of salt contributed by the seawater (maximum of 0.05%) was considered inconsequential. Control diets were fed as supplied by the manufacturer. Ambient temperatures were monitored with a minimum-maximum thermometer located at a nearby pen area.

Ten birds (two control and four from each oil diet) were killed at the end of weeks 1, 2, and 4. At the end of week 4, the remaining 10 birds were placed on untreated (control) diets, maintained for an additional 4 weeks, then killed (week 8). Birds were weighed weekly (except for week 6) and food consumption was measured daily for weeks 1-5. Food consumption was not determined during week 6 and on alternate days during weeks 7 and 8. Fresh food was provided each morning and food remaining from the previous day was weighed and removed. Food was provided in 50g amounts 4 days per week and 75g amounts the other 3 days. Water was available *ad libitum*.

Before killing, birds were weighed and bled by jugular venipuncture with sodium heparinized 5cc syringes and 2.54cm 23 gauge needles. The heparinized blood was transferred to 5cc partially evacuated tubes^[2] and a portion was sent to a commercial laboratory^[3] for automated erythrocyte count (RBC). Packed cell volume (PCV) was determined by the microhematocrit method and hemoglobin (Hb) was measured by

the cyanomethemoglobin procedure.^[4] The remaining whole blood was centrifuged at 1325 g for 15 minutes and the plasma harvested. Plasma was stored at -85 C before determination of uric acid and alanine aminotransferase (ALT) by using a centrifugal analyzer.^[5]

Kestrels were killed with Fluothane[®]^[6] and examined at necropsy. Brain, liver, and kidney were weighed and the following tissues fixed in 10% buffered formalin: brain, esophagus, trachea, cardiac muscle, skeletal muscle, liver, kidney, adrenal, spleen, proventriculus, stomach, duodenum, pancreas, and Meckel's diverticulum. Tissues were sent to a commercial laboratory^[7] for histologic processing and staining with hematoxylin and eosin (H & E). Oil red O, Mallory's iron, and Von Kossa stains were used on duplicate sections to confirm the presence of fat, iron, and calcium, respectively. Tissues from control birds and birds fed 3.0% crude oil diets were examined microscopically.

One-way analysis of variance and Duncan's multiple Range test (Steel and Torrie, 1960) were used for statistical interpretation of hematology and plasma chemistry; results from males and females were combined. Data for food consumption and body weights were analyzed by repeated measures analysis of variance (Winer, 1971) and Bonferroni multiple comparison procedures (Neter and Wasserman, 1974).

RESULTS

Males were significantly lighter ($P < 0.05$) than females at the beginning

[1] Nebraska Brand, Central Nebraska Packing, North Platte, Nebraska 69101, USA.

[2] Vacutainer[®], Becton-Dickinson, Rutherford, New Jersey 07070, USA.

[3] Bionetics Medical Laboratories, Kensington, Maryland 20795, USA.

[4] Hycel Kits 116 and 117, Hycel Inc., Houston, Texas 77052, USA.

[5] CentriChem[®], Union Carbide Corporation, Rye, New York 10580, USA.

[6] Ayerst Laboratories, Inc., New York, New York 10001, USA.

[7] American Histolabs, Inc., Rockville, Maryland 20850, USA.

of the acclimation period and at the initiation of the study, but this difference was not detectable after 1 week on experimental diets, so data for sexes were combined. No significant differences ($P < 0.05$) existed with respect to initial body weight or food consumption or between birds killed at a given time interval and those not killed. Only those birds alive at week 4 ($N=20$) and at week 8 ($N=10$) were used in the analyses or reported in the figures.

Two birds (both males) died after 2 weeks on 3.0% IXO diets and were included as part of the group killed at 2 weeks. Both had lost weight (21% and 31%) and died during the night following a 24 C drop in temperature from the previous day's high. All birds maintained on 3.0% IXO diets lost significantly ($P < 0.05$) more weight than controls or birds fed 0.3% diets, whereas no differences

($P > 0.05$) were detected between controls and the 0.3% treatment group (Fig. 1). When placed on untreated food, the birds fed 3.0% diets gained weight and by the end of week 7 there were no significant differences ($P > 0.05$) in body weight between treatment groups (Fig. 1). After week 1, birds fed 3.0% diets ate significantly ($P < 0.05$) more food than birds in other treatment groups (Fig. 2). The birds fed 0.3% diets also ate slightly more than controls, but the difference was not significant ($P > 0.05$). Food wastage was minimal and equal between groups. The drop in food consumption by controls after week 1 probably reflected the stabilization of their body weights. Up to that point they were gaining weight.

Mean Hb of kestrels fed 3.0% IXO was significantly less ($P < 0.05$) than controls after 1 week on treatment (Table 1).

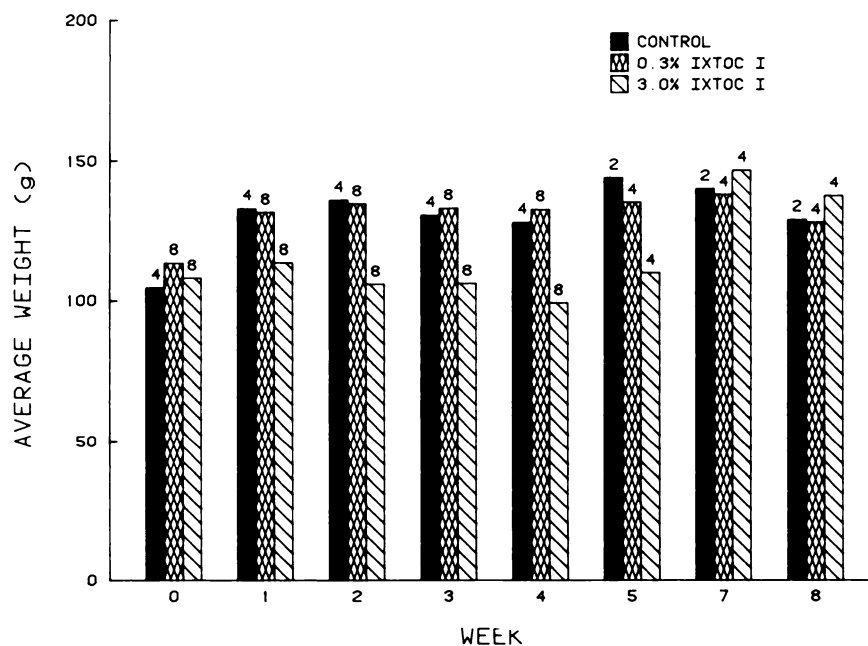


FIGURE 1. Average weekly body weights of American kestrels at the start of the acclimation period (week 0), during the time fed Ixtoc I crude oil in the diet (week 1-4), and while on untreated diets (week 5-8). Sample size is indicated over each bar.

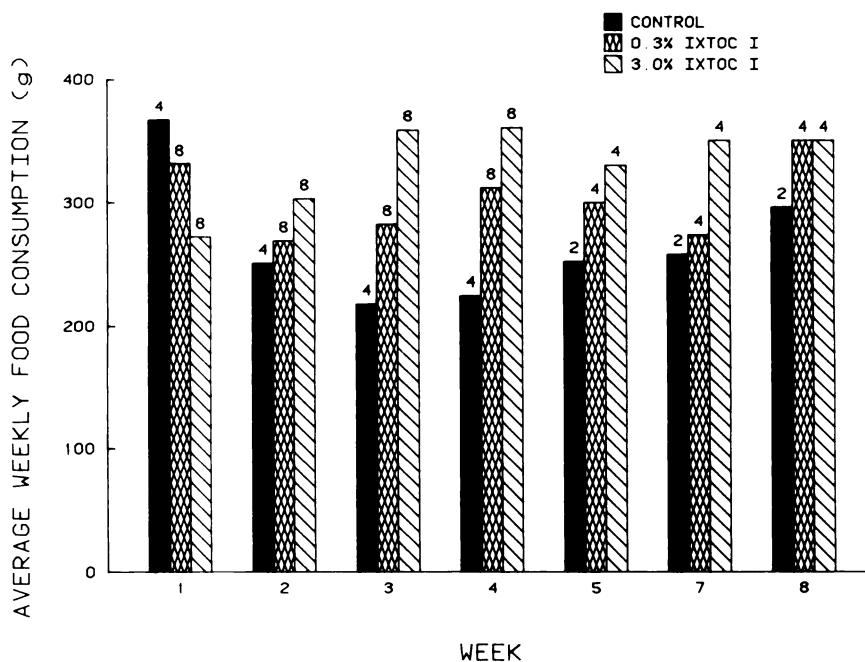


FIGURE 2. Average weekly food consumption of American kestrels fed Ixtoc I crude oil in the diet (week 1-4), and while on untreated diets (week 5-8). Sample size is indicated over each bar.

TABLE 1. Hematological results from American kestrels fed Ixtoc I (IXO) crude oil (means \pm SD).

Week of killing	Treatment	N	Packed Cell Volume (%)	Hemoglobin (g/dl)	Erythrocyte Count ($10^6/\mu\text{l}$)
1	Control	2	45.8 \pm 1.80	14.2 \pm 0.60A ^a	3.00 \pm 0.18
	0.3% IXO	4	45.1 \pm 3.10	13.9 \pm 0.50A	3.06 \pm 0.26
	3.0% IXO	4	39.3 \pm 3.40	11.8 \pm 1.10B	2.63 \pm 0.24
2	Control	2	39.5 \pm 2.80	12.9 \pm 0.60	2.62 \pm 0.04
	0.3% IXO	4	46.5 \pm 2.30	15.0 \pm 1.10	3.01 \pm 0.23
	3.0% IXO	2	35.8 \pm 11.00	11.7 \pm 3.70	2.50 \pm 0.93
4 ^b	Control	2	42.5 \pm 2.10	13.5 \pm 0.90	2.68 \pm 0.01
	0.3% IXO	4	41.3 \pm 6.60	12.6 \pm 2.00	2.70 \pm 0.40
	3.0% IXO	4	39.0 \pm 4.70	12.4 \pm 1.60	2.51 \pm 0.30
8	Control	2	46.0 \pm 0	14.3 \pm 0	2.99 \pm 0.04
	0.3% IXO	4	42.0 \pm 3.60	13.3 \pm 0.90	2.89 \pm 0.22
	3.0% IXO	4	43.8 \pm 1.70	13.5 \pm 1.10	2.95 \pm 0.13

^aMeans which do not have a letter in common were significantly different ($P < 0.05$).

^bAt the end of 4 weeks all birds were provided with untreated feed.

Although mean Hb was not significantly different throughout the remainder of the study, it remained lower in kestrels fed 3.0% IXO than in controls. There were no significant differences in PCV or RBC, but both were consistently less in high-dose birds than in controls (Table 1).

There were no significant differences between controls and kestrels fed 3.0% IXO with respect to plasma uric acid values or ALT activities. Uric acid in six control and five birds fed 3.0% diets was 14.5 ± 5.8 and 13.3 ± 4.8 mg/dl (mean \pm SD), respectively, and mean ALT activity was 25.8 ± 2.2 in four controls and 25.7 ± 7.9 IU/l in four birds fed 3.0% diets.

There were no statistically significant differences ($P > 0.05$) between treatment groups with respect to absolute brain, liver, or kidney weights. Liver and kidney weights were also examined as functions of brain weight and initial body weight; again, no statistically significant differences ($P > 0.05$) were noted.

Microscopic examination of tissues revealed accumulation of hemosiderin in hepatocytes and Küpffer cells in livers from eight of 16 birds fed 3.0% diets and one of eight controls. Splenic hemosiderosis was also more prevalent in oil-fed birds (six of 16 compared with one of eight controls). Circulatory hemosiderosis, evidenced by the presence of hemosiderin in vessels of tissue sections, was seen in two of 16 oil-fed birds and no controls. Diffuse hepatic fatty change was observed in three controls and three oil-fed birds. Several calcified casts were noted in medullary collecting ducts and thick segments of medullary loops in the kidney of one bird fed 3.0% oil. No significant lesions were observed in other tissues examined.

DISCUSSION

The two birds fed 3.0% diets probably died as a result of weight loss followed by cold stress. Ambient temperatures fluctuated throughout the study, but

maximum-minimum differences were usually less than 16 C, so the 24 C drop may have been a significant stress to birds in poor flesh. Holmes et al. (1979) found that in mallards, ingestion of petroleum increased the probability of death in birds already stressed by cold and seawater-adaptation.

The inability of the birds fed 3.0% diets to maintain their body weight despite increased food consumption is particularly noteworthy. This trend was evident in the birds fed 0.3% diets as well, but these birds were apparently able to compensate with a minor increase in food consumption. Holmes et al. (1978) reported hyperphagia in ducks consuming petroleum-contaminated food, and suggested it might have been the result of higher than normal levels of adrenocortical activity, or possibly some direct effect of petroleum on absorptive properties of mucosal cells in the small intestine. Miller et al. (1978) found that herring gull chicks which had been dosed with crude oil consumed more food than controls, but gained less weight, due in part to reduced nutrient transport in the intestine.

The oiled food was avoided the first week, particularly the 3.0% diet. Engel et al. (1978) observed a similar depression in food intake in Coturnix quail (*Coturnix coturnix*) following a single oral dose of Prudhoe Bay crude oil. However, an alternate choice of untreated food was not available to the kestrels and after an initial adjustment period they accepted the oiled food. It is probable that in a free-choice situation they would avoid oiled food.

Hartung and Hunt (1966) reported decreased PCV values in ducks fed fuel oil, and Szaro et al. (1978) found similar reductions in mallard ducklings fed south Louisiana crude oil. In the present study, kestrels fed 3.0% oil had consistently lower PCV, Hb and RBC values than controls, but the only statistically significant difference was in Hb at 1 week. Because of the low magnitude of

this Hb reduction and the absence of significant differences in other erythrocyte data, kestrels receiving 3.0% oil were not considered anemic.

The significance of hepatic hemosiderosis in oil-fed birds is unknown, but has been reported in birds following the ingestion of fuel oil (Snyder et al., 1973). It may have been related to excessive hemolysis of erythrocytes (Smith et al., 1972), since birds fed 3.0% diets had consistently lower Hb, PCV, and RBC values, and circulatory hemosiderosis was present in two oil-fed birds. Since fatty change of similar severity was present in the livers of controls as well as oil-fed birds, it was apparently unrelated to petroleum ingestion. The extent of renal calcinosis observed in one oil-fed bird was probably not sufficient to cause compromise of kidney function. Absence of significant

liver and kidney damage is further supported by the similarity in ALT activity and uric acid levels between dosed and undosed birds.

Dietary Ixtoc I crude oil evidently poses little hazard to free-ranging American kestrels. High level continual exposure (equalling 0.5% of the birds' body weight) caused death after two weeks when combined with severe cold stress, but lower level exposure was apparently harmless. Such high level exposure is improbable in nature because of avoidance of the oiled food and the amount of time required for such exposure to occur. Even if such exposure occurs, survival is good (88%) and recovery rapid. It is therefore unlikely that the Ixtoc I oil spill posed any acute toxic hazard to peregrine falcons. However, long-term effects of such exposure were not determined, nor were the effects of other types of oils evaluated.

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