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Physiologic and Clinicopathologic Effects Of Crude Oil On
Loggerhead Sea Turtles

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Abstract The physiologic and clinicopathologic effects of weathered South Louisiana crude oil exposure were studied in the laboratory in juvenile loggerhead sea turtles. Sea turtles ingested oil incidentally, and oil was observed clinging to the nares, eyes, and upper esophagus, and was found in the feces. Oiled turtles had up to a four-fold increase in white blood cell counts, a 50% reduction in red blood cell counts, and red blood cell polychromasia. Most serum blood chemistries (e.g. BUN, protein) were within normal ranges, although glucose returned more slowly to baseline values than in controls. Gross and histologic changes were present in the skin and mucosal surfaces of oiled turtles, including acute inflammatory cell infiltrates, dysplasia of epidermal epithelium, and a loss of cellular architectural organization of the skin layers. The cellular changes in the epidermis are of particular concern because they may increase susceptibility to infection. Although many of the observed physiological insults resolved within a 21-day recovery period, the long-term, biological effects of oil on sea turtles remain completely unknown.

Introduction

Sea turtle populations around the world coexist with oil and gas exploration and development activities. This association is not without risk, as evidenced by accounts of turtles harmed by oil in the Gulf of Mexico (Hall et al. 1983), the Atlantic (Witham 1978), the Gulf of Iraq (Hutchinson and Simmonds 1992), the Mediterranean (Grammentz 1988), and in the Red Sea (Frazier and Salas 1984). Sea turtles also consume and become fouled in oil and tar balls (Witham 1978; Witham et al. 1986), raising the possibility of bioaccumulation over their potentially long lifespan.

Only one previous study concerned the effects of oil on sea turtles, documenting egg

and hatchling mortality following direct application of crude oil (Fritts and McGehee 1981). The vulnerability of juvenile and adult sea turtles was unassessed. Yet the potential risk for turtles encountering oil at sea is high. In an oil slick the turtles' habit of surfacing to breathe ensures a continued contact with the oil. There is also evidence that sea turtles feed selectively in surface convergence lines which could also result in prolonged contact with viscous weathered oil (Witham 1978; Hall et al. 1983). Some species are indiscriminate feeders, and foreign material that they consume may remain in the digestive track for periods of weeks to several months (Lutz 1990).

The primary intent of this research, mandated by the U.S. Minerals Management Service (MMS), was to determine if exposure to crude oil is detrimental to the health of sea turtles. Since little was known about the toxic effects of oil in marine or freshwater turtles, we reasoned that a broad survey approach might increase our chances of identifying susceptible physiological systems. Those organ systems that had been previously shown in marine birds and mammals to be impacted by oil were chosen for initial study (Geraci and St. Aubin 1985; Leighton 1986).

Methods

Animals Loggerhead turtles (*Caretta caretta*) ranging from 8-12 kg and approximately 15-18 months old were obtained from mixed clutches of Florida headstarted i.e., hatchery-reared turtles. Turtles were kept outdoors in large 1.2 X 1.8 m fiberglass tanks supplied with seawater continuously pumped from Biscayne Bay and turtles were fed only pellet chow (#45 sea turtle chow, Purina, Inc.) ad libitum, 3-4 X per week. Sea turtles were brought indoors, weighed and held in a 1000 L tank filled with fresh unfiltered seawater at room temperature

(22-24° C) 24 h before initiating data collection.

South Louisiana crude oil supplied by the MMS was weathered outdoors over aerated seawater for 48 h in a wide glass container, and then was poured over the surface of the tank. An oiling level was chosen that was the minimum practical oil layer thickness (0.5 mm, 96 h) that would ensure that the surface of tank was covered completely by oil. Five sea turtles each were used in the oil exposed and control group, respectively.

Each time oiled turtles were removed from the tank for collection of blood samples, some of the oil adhering to the body was unavoidably removed by sorbent towels used to hold the turtles for blood sampling. Other than this, we made no attempt to remove oil until the recovery period. Following the 96th hour sampling point we used the oil sorbent sheets to remove oil adhering to the skin of the turtle. The mouth cavity and eyes were also blotted clean but no "detergents" were used to clean the turtles. Turtles were then removed to large outdoor holding tanks until the next sampling period and at this time had access to turtle chow. Day zero served as a baseline for both oiled and control turtle groups.

During the course of the experiments samples were obtained of tank water, the oil surface layer, and turtle feces; these were submitted for hydrocarbon analysis. Hydrocarbons in tank water for one experiment declined from 164.2 to 62.4 mg L⁻¹ between days 1 and 4. Additional data on hydrocarbon levels are published in detail elsewhere (Van Vleet and Pauly 1987). No sea turtles deaths or permanent disabilities were believed to have occurred as a direct result of experimental procedures.

Exposure Studies At least one hour before baseline measurements were begun, a lid was placed over the seawater in the tank so that the turtle could only surface to breathe in a 600

ml glass funnel respirometer, described previously (Lutcavage and Lutz 1991). Once turtles became habituated to breathing in the funnel (1-3 h) we recorded ventilation rate (f) for 60-90 min with a digital pneumotachograph (Size #1, Hewlett Packard, Inc.) attached to the funnel with a one-way respiratory valve (Rudolph, Inc).

Venous blood was sampled aseptically from the dorsal cervical sinus and whole blood was used for determination of pH, blood gases, cell counts, hematocrit, and hemoglobin analysis. Venous blood pH, oxygen (pO_2), and carbon dioxide partial pressure (pCO_2) were determined immediately with a digital acid-base analyzer (Mk 2, Radiometer, Inc.) thermostatted to room temperature. Serum and plasma were separated from whole blood and used for determination of blood glucose, serum protein, and selected serum chemical compounds. Serum protein and blood glucose were determined photometrically with Sigma Chemical kits. Clinicopathologic parameters measured included hemograms, selected serum chemical compounds, fecal blood, and dermopathologic evaluation. Cloacal fecal samples were obtained via gentle swabbing with a culturette lubricated with sterile K-Y jelly. Hemograms were calculated from EDTA anticoagulated blood and included red blood cell indices and white blood cell total differential counts. Red and white blood cells were obtained by conventional methodology. Oropharyngeal and fecal microbiologic flora were collected with sterile swabs in transport media and results are reported elsewhere (Bossart 1986). Selected serum chemical compounds were determined with a multifilter photometer (450-610 nm) with commercially obtained individual blood chemistry kits (Mallinckrodt, New York). The presence of fecal blood was determined by a commercially prepared strip colorimetric test (Multistix, Ames, Inc).

Histopathology Skin biopsies were obtained following intradermal injection of a local anaesthetic (2% Lidocaine HCL). Skin biopsies were collected aseptically via an incisional or a 5 mm punch technique. The sites biopsied included the dorsal flipper mid zone and the ventrolateral neck skin. Tissues for histologic evaluation were fixed immediately in 10% neutral buffered formalin and processed routinely. Specimens were cut at 5 μ m and stained with hematoxylin and eosin.

Data Analysis All values are expressed as mean $\pm$ 1 standard error of the mean (SEM). After testing for normality, statistical comparisons among mean data groups were made with one-way anova at each sampling point with mean separation performed with the Fisher's least significant difference t-test. The confidence level was selected at $P < 0.05$.

Results

Ventilation and Submergence Loggerheads spent approximately two thirds of their time submerged, with no significant differences between oiled turtles and controls (Figure 1a). The presence of oil had no consistent effect on breathing frequency although during recovery there was some indication of reduced ventilation rate (Figure 1b).

Blood Gases, Hematology, and Serum Chemistry Venous pO_2 remained almost constant throughout the sampling period, showing no disturbance in tissue oxygen uptake (Figure 1c). In both control and oiled groups, venous pCO_2 values were low, and pH values high (Figure 1d), suggesting that hyperventilation caused respiratory alkalosis. The oiled group had slightly but not significantly more acidic values.

Hemoglobin concentration did not show a statistically significant change during oil exposure but there was an increasing trend during oiling. Hematocrit was relatively

constant throughout sampling (Figure 2). Red blood cell counts in control and oiled turtles initially declined, but in the oiled group were reduced by almost 50% and did not return to baseline values during the recovery period (Figure 2c). Peripheral blood smears from oiled sea turtles exhibited red blood cell polychromasia. There was a rapid and significant rise in white blood cell count which in oiled turtles persisted beyond a week of recovery (Figure 2d). By day 3 of oil exposure, white blood cell counts had increased four-fold, indicating that the turtles' immune response probably had been activated by oil exposure.

Serum glucose declined in both oiled and control groups (range 76-138 mg 100 ml⁻¹), and was probably a function of starvation during confinement. The decline was reversed with feeding but the rate of recovery in oiled turtles was slower than that of controls. Blood urea nitrogen (range 55-115 mg 100ml⁻¹) decreased during the exposure period in both groups and may have been related to starvation. However, values in controls showed an immediate increase with feeding while in oiled turtles mean values continued to decline for ten days. Serum protein was relatively constant in both groups (range 2.7-3.4 g 100 ml⁻¹). Occasional traces of both hemolyzed and non-hemolyzed blood in feces were present in control and oiled animals. This trace amount probably reflects a dietary origin, but may also have originated from minor trauma occurring during cloacal fecal specimen sampling.

Dermatopathology and Histopathology Each time the turtles surfaced oil residue was observed around their eyes and in the nasal and oral cavities, and their tongues were darkly stained with oil. In all of the loggerheads exposed to oil, the skin began to slough off in layers especially around the neck and flipper area. The exposed soft skin was reddened and lacked the leathery texture of the original epidermis. Skin continued to peel off for 1-2

weeks after removal from the oil and only recovered its normal leathery texture only after about a month following oil exposure. Consistent gross and histologic changes were present in oiled turtles that did not occur in controls. After exposure to oil had ceased (day 5) marked microscopic changes were observed in the skin. In the epidermis, extensive numbers of acute inflammatory cells, primarily heterophilic granulocytes, diffusely infiltrated the cellular layers beneath the outer epidermal layer (stratum corneum, Figure 3a). Associated with the acute inflammatory cell infiltrates was moderate to extensive, multifocal epithelial cell necrosis. These necrotizing changes were characterized by loss of intercellular bridges and cellular cohesion, with shrunken, thickened hyperchromatic nuclei. In addition, there was moderate dysplasia of epidermal epithelium characterized by loss of individual cell uniformity, cellular architectural orientation, and atypical proliferative changes (Figure 3b). During this time period there was also marked diffuse acanthosis and hyperkeratosis.

The superficial dermis was characterized by moderate to severe diffuse edema with associated diffuse infiltrates of heterophilic granulocytes and occasional plasma cells, histiocytic cells, and small lymphoid cells. There was also moderate multifocal, superficial dermal hemorrhage. Changes were more extensive in the soft pliable skin of the neck and less severe in the hard, heavily keratinized skin of the flippers. Depending on the biopsy site these lesions were still apparent seven to nine days after oil exposure had ceased.

Nine to 13 days after oiling, epidermal inflammatory cell infiltrates, necrosis, and dermal hemorrhage were no longer present in the skin biopsies. There was still moderate acanthosis and hyperkeratosis with marked sloughing of the cornified layer of the epidermis. Inflammatory cells had mostly disappeared from the dermis although there was still mild

superficial dermal edema. At this time and up to 21 days after exposure to oil had ceased marked grossly observable sloughing of the skin and exposed mucous membranes occurred, involving primarily the skin of the neck, top and sides of the head, and inguinal and axillary regions. Sloughing mucous membranes involved the periorbital conjunctiva, nasal mucosa, and oral mucosa, and consisted of coagulative necrosis admixed with extensive amounts of keratin and heterophilic granulocytes.

Discussion

Ventilation and Submergence Ventilation and submergence patterns of oiled loggerhead turtles were not significantly different than unoiled turtles. Both oiled and control turtles spent at least three times their usual amount of time at the surface than they typically do in large outdoor tanks. Turtle breathing patterns were undoubtedly influenced by the conditions of confinement in the experimental tank and the imposition of breathing in the funnel. Nevertheless, a few individuals seemed to be disturbed even more so by the presense of oil. These turtles either reduced the amount of time spent at the surface, possibly avoiding oil, or became more agitated and had short submergence intervals. Although consistent changes were not evident under the conditions of this study, it is likely that the presence of oil could alter ventilatory patterns of turtles surfacing to breathe in oil slicks.

In oiled turtles there was no evidence of reduced oxygen uptake by the blood or tissues. Ventilatory activity was high in both groups, and it is possible that the increased ventilation was sufficiently great to mask any effect or graded response that oil might have had on oxygen uptake across the lungs. In terrestrial mammals, petroleum vapor inhalation alters behavior and causes pathological changes in the respiratory system (Nau et al. 1966).

Sea otters oiled as a result of the Exxon Valdez spill suffered higher incidences of emphysema and petroleum hydrocarbon toxicosis (Williams et al. 1990). Inhalation of toxic oil fumes is also believed to pose serious health hazards to whales and dolphins (Geraci and St. Aubin 1982; Geraci 1990). In this study, oil vapors may have irritated turtles, but did not appear to have reached levels capable of causing critical respiratory damage.

Hematology The most striking hematologic finding resulting from oil exposure was an elevation of white blood cell count, which may indicate a "stress" reaction related to oil exposure and/or toxicity. The response was oiling duration-dependent, characteristically increasing during oiling periods and returning to preoiling counts after exposure ceased. A similar "stress"-related leukocytosis has been described in mammals (Cole 1974) and in oiled herring gulls (Leighton 1986; 1990).

Red blood cell polychromasia occurred in peripheral blood smears from oiled turtles. In mammals, this diffuse red blood cell basophilia is associated with regenerative anemic conditions and represents an immature form of an erythrocyte. While these apparent regenerative states were temporary, the pathogenesis of such conditions remains speculative. Similar regenerative anemic conditions which eventually resolve with medical care and rehabilitation have been reported in marine birds exposed to oil spills (McNair 1980). Some parallels indicating the primary target of oil toxicity is the red blood cell have been found in seabirds that ingested crude oil (Hartung and Hunt 1966; Leighton 1986; Fry et al. 1985).

No significant differences were detected in most of loggerhead serum blood chemistries. If extrapolations can be made from mammals and birds to sea turtles, this data suggests that short-term exposure to oil has little effect on most liver, renal, pancreatic, or

metabolic electrolyte-related processes. This interpretation assumes that parameters in sea turtles are indicators of specific tissue or organ function as they are in mammals and some birds (Cole 1974).

In both control and oiled groups, ventilatory and hematologic fluctuations were especially pronounced in the first half of the experimental period, indicating that handling and laboratory stress persisted well beyond the 24 hr acclimatization period. This could not be avoided because a prolonged period of confinement in the laboratory, alone, could cause deleterious effects on the health of sea turtles. Since both groups were exposed to the same handling regimes, the more pronounced responses seen in oiled turtles suggest that oil effects were indeed evident.

While this study was underway, we incidentally sampled salt gland fluid and learned that it failed to function properly in oiled two turtles for a period of several days. The failure of the salt gland to produce fluids several days after exposure points to attenuated or delayed effects in salt transport. The delayed shutdown was unexpected, and unfortunately we did not study this response to oiling in detail since it was not part of the original experimental protocol. The salt glands had recovered when function was tested after two weeks of recovery. The nasal salt gland of sea birds orally dosed with crude oil also shows a loss of responsiveness, attributed to suppression of adrenocortical activity (Holmes et al. 1980; Leighton 1990). The primary site of action on both seabirds and turtles may be the adrenal glands (McEwan 1978). Prolonged interference with salt gland functioning could have serious consequences since it would interfere with both water balance and ion regulation.

Dermatopathology The changes present in the skin biopsies at day 2 to day 4 post oil exposure are consistent with an acute, primary contact or irritant dermatitis. During this period of time, dysplastic cellular changes in the epidermis were present and often marked. The epidermal dysplasia and most of the acute and chronic active inflammatory changes had resolved within a week following cessation of oil exposure, consistent with a chronic resolving inflammatory process. Dysplasia is generally a reversible condition, and, when the underlying inciting stimuli is removed, the dysplastic alterations generally revert back to a normal state. In some mammalian species, cancerous transformation supervenes. The integumentary changes in loggerhead sea turtles are in marked contrast to the lack of skin damage with oil exposure observed in dolphins (Geraci and St. Aubin 1982), where all structural and biochemical changes in epidermis were minor and reversible (Geraci and St. Aubin 1985).

Perhaps more worrisome than cancerous transformation is, that due to the nature of the acute dermatitis, a break could result in the integumentary barrier and act as a portal of entry for pathogenic organisms, especially if repair and protective processes could not keep ahead of the irritant properties of the oil.

Oil found in the feces of oiled loggerheads, ranging in concentration from 0.81-22.0 g L⁻¹ (Van Vleet and Pauly 1987), may have been absorbed from the gastro-intestinal tract, and could account for some of the observed hematologic abnormalities. If oil passed from the GI tract into the turtles' system it was not sufficient to cause intestinal bleeding and anemia, as reported in aquatic birds (Hartung and Hunt 1966). Tissue hydrocarbons of both control and oiled turtles were near lower detection limits. Thus it cannot be stated with certainty whether

uptake of hydrocarbons in the tissues occurred. In a dead green turtle found and necropsied during a Gulf of Iraq environmental damage survey, petroleum hydrocarbon levels of 4,050 and 310 mg kg⁻¹ were found in the liver and stomach, respectively (Greenpeace 1992). The impact of tissue oil uptake on the long term health and survival of sea turtles remains unknown.

Conclusions Experiments on the physiologic and clinicopathologic effects of oil showed that major body systems in marine turtles are adversely affected by short exposures to weathered oil. The laboratory oil slicks simulated conditions occurring during contact with weathered oil, but freshly spilled oil could prove to be considerably more harmful. Additionally, sea turtles pursue and swallow tar balls, and there is no firm evidence that they are able to detect and avoid oil (Odell and MacMurray 1986). Sea turtles are among the endangered or threatened marine species that may be most at risk in the event of a oil spill. For turtles such as the Kemp's ridley, which is barely holding onto survival, a serious encounter with oil could threaten survival of the species.

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Figure Legends

Fig. 1 (a) Percent time spent at the surface and (b) breathing frequency of loggerhead turtles, under control conditions (day 0), during four days of oil exposure (indicated by black horizontal bar), and during the eighteen day recovery period. Values are means $\pm$ 1 SEM.

(c) Venous oxygen partial pressure (pO_2) and carbon dioxide partial pressure (pCO_2) of oiled and control turtles. (d) Venous hydrogen ion concentration (pH) of oiled and control turtles.

Fig. 2 (a) Hemoglobin concentration and (b) hematocrit in control and oiled turtles. Values are means $\pm$ 1 SEM. (c) Red blood cell count and (d) white blood cell count in control and oiled turtles. Asterisk indicates significant difference.

Fig. 3 (a) Medium-power photomicrograph of a skin biopsy from the neck of a loggerhead sea turtle that was exposed to oil for 2 days. The outer epidermal keratinized layer (SC) is markedly thickened and irregular with occasional inflammatory cells and focal necrosis. The thickened epidermal strata (ES) shows increased number of cell layers. The superficial dermis is characterized by mild to moderate edema (ed). Hematoxylin and eosin stain, 100x.

(b) Medium-power photomicrograph of a skin biopsy from the neck of a loggerhead sea turtle, 3 days after oil exposure had ceased. The base of the epidermal keratinized layer is characterized by multifocal necrosis with infiltrates of primarily heterophils (large arrows) and small lymphoid cells (small arrows). Hematoxylin and eosin stain, 100x.







