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FIN EROSION DISEASE INDUCED IN THE LABORATORY

Several characteristics of the fin erosion disease in southern California Dover sole suggest that the causative agents are associated with the contaminated sediments on the Palos Verdes shelf. First, although the disease is present in populations in several areas off Los Angeles and Orange Counties, affected individuals are consistently most prevalent in the Palos Verdes area; second, the disease does not appear to be the result of an infectious process; and third, the fins that contact the bottom most often are the most frequently and severely affected.

The bottom sediments on the Palos Verdes shelf are characterized by high levels of chlorinated hydrocarbons and metals, small average grain size, and large amounts of organic material. Unidentified fibrous materials (glass, nylon, asbestos, etc.) may also be present in these sediments. Interstitial waters from some areas of the shelf are characterized by reduced pH, the presence of hydrogen sulfide, and the absence of oxygen. In addition, fish living on these sediments have been shown to be subject to bioaccumulation of chlorinated hydrocarbons but not of metallic elements.

To determine if the fins of healthy Dover sole are damaged by contact with contaminated bottom sediments, we exposed Dover sole from Santa Catalina Island and Dana Point that had been maintained in the laboratory for periods of up to 10 months to a sample of sediment taken from the most highly contaminated region of the Palos Verdes shelf. The experiment was started in February 1975. Signs of dorsal and anal fin erosion were noted in the test fish in March 1976.

METHODS

Test sediments (up to 10 cm deep) were maintained in recirculating 40-gallon aquaria at 12°C for 3 weeks prior to the addition of fish. The sediments contained high levels of chlorinated hydrocarbons, trace metals, volatile solids, and chemical oxygen demand. For the test, three Dover sole were added to each of four aquaria—two containing Palos Verdes sediments, and

two containing silica sand. The fish were fed Tetramin staple tablet food; levels of chlorinated hydrocarbons and trace metals in the food are shown in Table 1.

Following the addition of fish, water quality was measured weekly by Jean Wright; parameters checked were total ammonia, nitrite, nitrate, dissolved oxygen, temperature, pH, and specific gravity. Levels of chlorinated hydrocarbons and trace metals were monitored at the beginning and at the end of the test; these did not change significantly during the exposure period (Table 2). As the test sediments were disturbed by the fish during periods of activity, the characteristics of the interstitial waters (pH, oxygen, and hydrogen sulfide levels) in the upper layers of sediment varied (analyses of these parameters are still in progress).

After 13 months, specimens in one control and one test tank were sacrificed. Fin tissue from these specimens was examined by light microscopy by Raymond Bendele, Texas Veterinary Medical Diagnostic Laboratory, and tissue levels of metals and chlorinated hydrocarbons were measured in the Project's laboratories by Theadore Heesen, Tsu-Kai Jan, and Robert Johns on.

RESULTS

After 13 months, mortality in the test fish was similar to that in the controls—the small specimens in both of the test tanks and in one of the two control tanks had died. In general, the behavior of the exposed fish was similar to that of the controls. The fact that sediment particles were clinging to the blind side of the test fish when they left the bottom suggests that they were producing mucus.

The most striking difference between the two groups of fish was the presence of early signs of dorsal and anal fin erosion in the fish exposed to the contaminated sediments. The fin tips of these fish were missing, and the distal edge of the affected fin was covered with a border of clear epithelium punctuated by patches of dark color between the rays. This condition resembled the early signs of erosion seen in fish caught on the Palos Verdes shelf. Under the light microscope, the epithelium of the sacrificed fish did not appear either thickened or reduced in size, and no proliferation of fibrous tissue was visible. Three of the four test fish appeared to be affected by this condition, whereas no signs of fin erosion were evident in the control fish.

Levels of chlorinated hydrocarbons and trace metals in the tissues of the sacrificed fish are presented in Table 3. The concentrations of total DDT in the livers of the exposed fish were three orders of magnitude greater than the levels in the livers of the controls. Total DDT levels in the muscle and brain tissue of the exposed fish were also high. Total PCB values appeared to be elevated only in the livers of the exposed fish (approximately 20 times). No major differences between the trace metal levels in test and control specimens were apparent, with the exception of elevated cadmium in the liver of the one

exposed specimen. Liver weight to body weight ratios in the exposed fish were higher than in the control fish and approached the levels found in diseased specimens from the Palos Verdes shelf.

SUMMARY

This experiment shows that changes in fin condition can be induced in Dover sole in the laboratory by exposing apparently healthy individuals to contaminated Palos Verdes sediments. The changes resemble early stages of fin erosion seen in field specimens.

In general, levels of chlorinated hydrocarbons in the exposed fish fall between the levels reported for Palos Verdes specimens with no apparent fin erosion and those with moderate to severe fin erosion (see Page 143). Although not indicative of cause and effect, the results of this test, in conjunction with measurements of chlorinated hydrocarbons in field specimens, suggest that certain levels of one or more chlorinated hydrocarbons may be associated with the onset of the fin erosion disease symptoms. Levels of trace metals in both control and test fish are generally similar to the levels reported for field specimens (see Page 143), with the exception of the cadmium level in the liver of the exposed fish. Since elevated levels of cadmium were not found in the livers of Dover sole from Palos Verdes (de Goeij et al. 1974), this increase is probably not associated with the fin erosion disease.

The uptake of chlorinated hydrocarbons by the test fish suggests that chlorinated hydrocarbons can be accumulated directly from the sediments. Although the test sediments were not screened to remove infaunal organisms, it is unlikely that the small number of organisms present and likely to be ingested by the Dover sole, if eaten over the period of 1 year, would be sufficient to result in the measured levels of chlorinated hydrocarbons.

Future studies will include direct testing of the chlorinated hydrocarbons under control conditions and under conditions simulating those seen in the field.

REFERENCE

de Goeij, J.J.M., V.P. Guinn, D.R. Young, and A.J. Mearns. 1974. Neutron activation analysis trace-element studies of Dover sole liver and marine sediments. In Comparative studies of food and environmental contamination, pages 189-200. International Atomic Energy Agency, Vienna.

Table 1. Characteristics (mg/dry kg) of a sample of Tetramin Staple Tablet food fed to Dover sole exposed to control and Palos Verdes sediments.

Total DDT	0.013
Total PCB	0.077
Cadmium	5.8
Chromium	8.5
Copper	12
Nickel	0.99
Lead	—*
Zinc	69

*Below limit of detectability

Table 2. Characteristics (mg/dry kg) of two samples of Palos Verdes sediments used in laboratory exposure tests.

	26 Feb 1975	17 Mar 1976
Total DDT	117	127
Total PCB	6.01	4.32
Cadmium	64	60
Chromium	1,000	870
Copper	550	540
Nickel	84	99
Lead	380	400
Zinc	1,900	1,700

Table 3. Levels of chlorinated hydrocarbons (mg/wet kg) and trace metals (mg/dry kg) in Dover sole maintained on Palos Verdes and control sediments for 1 year.[a]

	Control		Test	
	Large	Medium	Large	Medium
Total DDT				
Muscle	0.288	0.010	1.76	1.38
Liver	0.082	0.109	102	70.4
Brain[b]	0.026		1.94	
Total PCB				
Muscle	0.146	0.073	0.084	0.097
Liver	0.495	0.282	9.60	5.88
Brain[b]	1.48		1.82	
Cadmium				
Muscle	–[c]	0.018	–[c]	0.029
Blind side skin	0.003	0.003	–[c]	0.062
Liver	NA[d]	0.487	16.1	NA[d]
Chromium				
Muscle	0.62	0.07	0.05	0.10
Blind side skin	0.47	0.44	0.40	0.12
Liver	NA[d]	0.22	0.07	NA[d]
Copper				
Muscle	3.61	1.13	2.11	0.600
Blind side skin	1.31	2.21	1.99	1.22
Liver	NA[d]	6.49	12.7	NA[d]
Nickel				
Muscle	0.919	0.966	1.77	0.459
Blind side skin	0.639	1.87	1.60	2.73
Liver	NA[d]	0.807	0.319	NA[d]
Lead				
Muscle	–[c]	0.111	0.366	0.023
Blind side skin	–[c]	–[c]	0.233	0.011
Liver	NA[d]	–[c]	0.399	NA[d]
Zinc				
Muscle	42.6	16.2	22.0	12.6
Blind side skin	120	52.4	76.7	68.0
Liver	NA[d]	48.9	65.9	NA[d]

a. Large control specimen was 194 mm, SL, and 78.6 g; small control specimen was 174 mm, SL, and 96.9 g. Large test specimen was 204 mm, SL, and 113.5 g; small test specimen was 171 mm, SL, and 67.2 g.
b. Composite sample.
c. Below the limit of detectability.
d. NA = tissue not analyzed.