Idiopathic Enteropathy in the Larval Pacific Herring, Clupea harengus pallasi (Valenciennes)

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The saturation of plumage and skin with sand, and the presence of hemorrhage on internal organs and around limbs, are conditions similar to those reported by Wooten (1954, op. cit.) for drowned ducks in Humboldt County.

To our knowledge, drowning has been reported only rarely in waterfowl as large as swans. However, considering all of the information, drowning appears to have been the most likely cause of mortality in the tundra swans.

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Idiopathic Enteropathy in the Larval Pacific Herring, *Clupea harengus pallasi* (Valenciennes)

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The Pacific herring constitutes an important commercial fishery around the North Pacific rim, as well as being an important forage species. Various factors which may affect early life stages and thus recruitment into the adult population have been studied. These include predation on eggs (Taylor, 1964, Fish. Res. Board Can. Bull. 143: 1–81) and larvae (Stevenson, 1962, J. Fish Res. Board Can. 19: 735–810; Fraser, 1969, J. Fish. Res. Board Can. 26: 1743–1762), high density of egg masses (Taylor, 1971, Rapp. Cons. Explor. Mer 160: 34–41), and temperature and salinity (e.g., Alderdice et al., 1971, J. Fish. Res. Board Can. 28: 1545–1562).

The purpose of this paper is to report preliminary observations of a severe necrotizing enteropathy in laboratory-reared Pacific herring hatchlings. Although several diseases have been reported in adult herring, diseases of larval herring, which may be significant determinants of a given year class success, are apparently unstudied and not reported in the literature. Difficulties in the collection, identification, and preparation of larval fishes from mixed wild populations have precluded the study of these life stages. Thus, this report is the first description of a disease of larval herring and, as well, demonstrates methods for the effective study of diseases of these animals.

Adult herring were obtained for laboratory conditioning and spawning from Puget Sound stocks in both 1982 and 1983. Conditioning, spawning and egg incubation were identical in each year and were effected using methods similar to those described by Alderdice et al. (1971, op. cit.). Animals examined in this study included those whose eggs were incubated under apparently optimal conditions as well as those used to examine the effects of environmental contaminants on egg development. Larval herring were collected for histological and ultrastructural studies of tissues between 18 and 30 hr posthatching. All animals were fixed whole in

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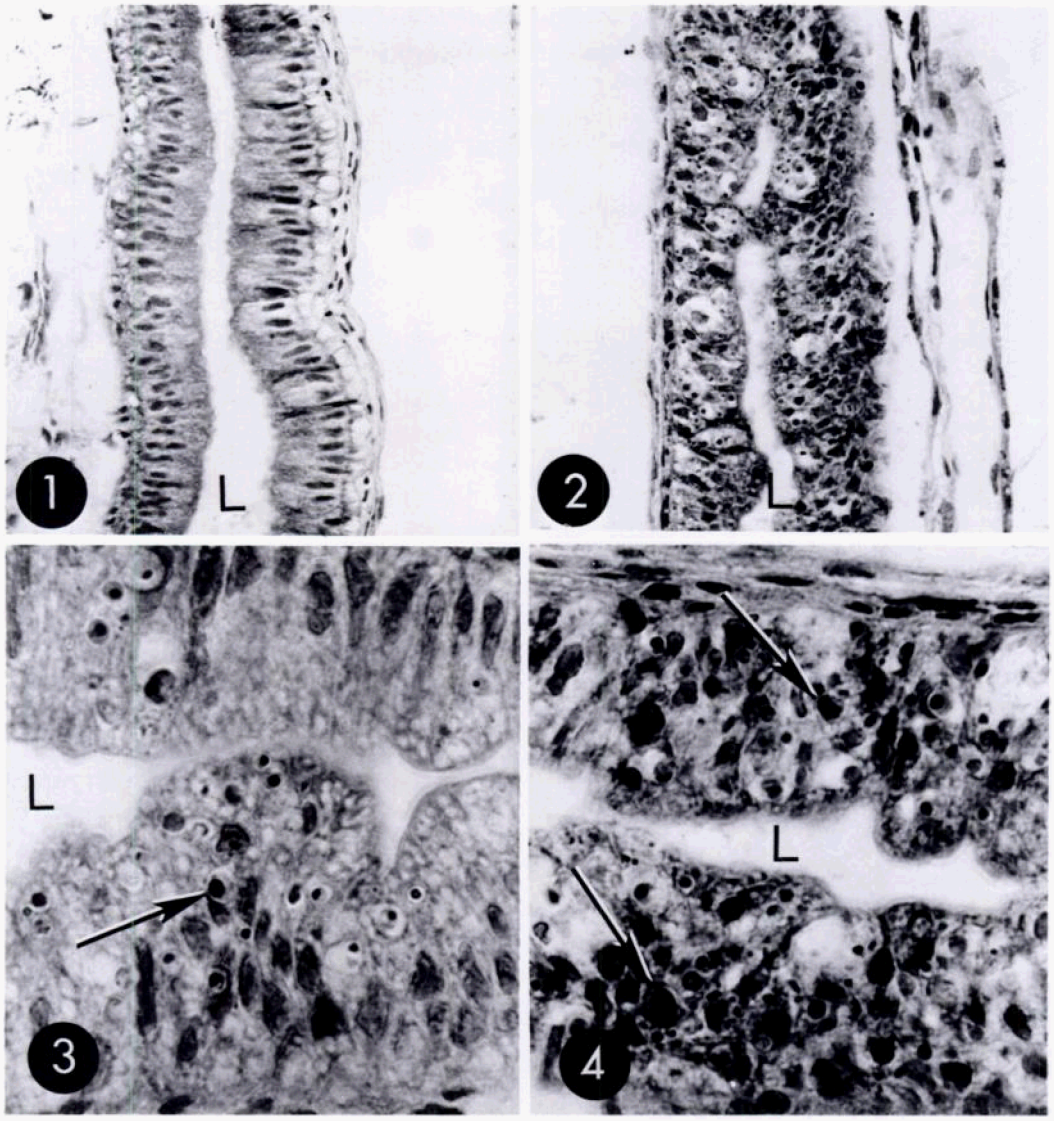

FIGURE 1. Methacrylate embedded section of normal intestinal epithelium of a larval Pacific herring. L, lumen. Hematoxylin and eosin H&E, ×210. FIGURE 2. Methacrylate embedded section of affected intestine of a larval Pacific herring. L, lumen. Note the numerous pyknotic nuclei and loss of normal columnar epithelial structure. H&E, ×210. FIGURE 3. Methacrylate embedded section of affected intestine of a larval Pacific herring. Note the characteristic pyknotic nuclei and halo surrounding the rounded cells (arrow). L, lumen. H&E, ×420. FIGURE 4. Methacrylate embedded section of more severely affected intestine of a larval Pacific herring. The two arrows show typical rounded, degenerating epithelial cells. L, lumen. H&E, ×420.

a phosphate buffered glutaraldehyde-paraformaldehyde fixative described by Elston (1980, Proc. Natl. Shellfish Assoc. 70: 65–93) and in 4% neutral buffered formalin. Whole larvae were embedded in a plastic histological medium, sectioned and stained with hematoxylin and eosin using methods described elsewhere (Elston et al., 1982, J. Fish Dis. 5: 265–284). Tissues were also embedded in a low viscosity epoxy

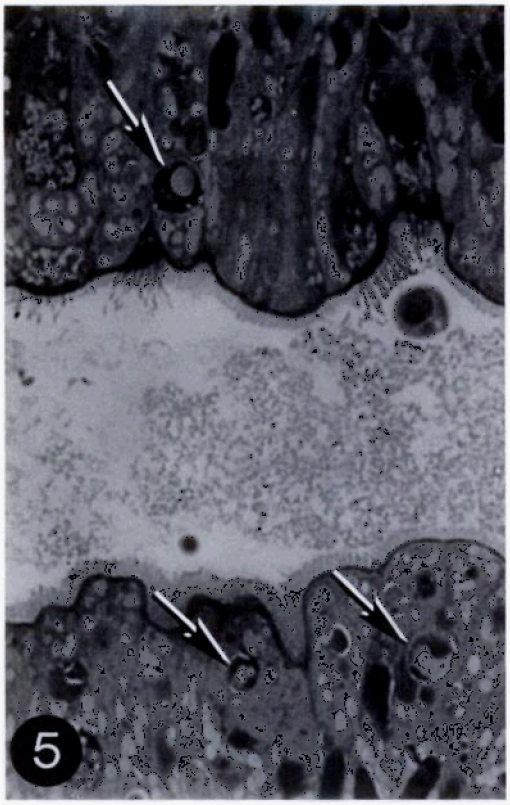

FIGURE 5. Epoxy embedded section of affected intestinal epithelium of a larval Pacific herring. Arrows show degenerating epithelial cells. Toluidine blue and basic fuchsin, ×780.

resin (Spurr, 1969, J. Ultrastruct. Res. 26: 31–43), stained with toluidine blue and basic fuchsin and examined histologically.

During the first year of study, 49 randomly selected individual animals were examined histologically. Of these, 28 (57%) exhibited enteric lesions. In contrast, 22 animals were examined in the second year of study, and no enteric lesions were observed. In the first year 78% of the larval herring reared under optimal conditions died within 2 days while only 22% of a similarly treated group died in year 2. Identical lesions occurred in approximately equal percentages in both untreated animals and in fish whose eggs and embryos had been exposed to experimental contaminants. Thus, the lesions occurred independently of any intentional toxicant exposures. There did not appear to be any relationship between the occurrence or severity of lesions and the number of hours post-hatching.

Normal intestinal epithelium is shown in Figure 1. Affected animals displayed a continuous series of pyknotic nuclei and rounded cells within the epithelium (Figs. 2–4). Progression of the disease and increasing intensity of the lesions were indicated by detachment of rounded epithelial cells into the intestinal lumen (Fig. 5) not seen in unaffected animals, and a terminal state in which virtually all epithelial cells were necrotic. The loss of normal epithelial cell structure in advanced cases (Fig. 4) suggests that the observed cells were pyknotic epithelial cells rather than other cells migrating through the epithelium. The disease appeared to progress from the anus in an anterior direction along the intestine. The posterior-most aspect of the intestine was always affected and a variable degree of anterior extension of the lesions was noted for each fish examined.

The digestive tract in these larval fish could be histologically differentiated into three general zones: (1) pharynx, anterior to the stomach, (2) stomach, displaying zymogenic columnar epithelium, and (3) straight tubular intestine lined with nonzymogenic columnar epithelium. The affected epithelial cells displaying the lesions described here were exclusively the columnar intestinal cells. Even in instances of advanced anterior extension of the necrotic epithelium, the disease never progressed into the zymogenic epithelium of the stomach. In the intestine, the affected cells appeared to be separated from intact epithelium by a thin halo or clear space (Figs. 3–5). Histological examination of tissues did not reveal any evidence of infectious agents associated with the disease process.

The severity of the lesions in advanced cases suggested that the disease virtually

interrupted normal function of the postgastric digestive tract. Therefore, continued development, normal nutrient absorption and fluid absorption and retention functions would not occur. Thus a significant effect on these 1-day-old hatchlings was suggested. The findings did not indicate whether or not recovery was possible. The high percentage of affected animals in the first year of study further suggested that occurrence of this disease in the wild population could have a significant impact on year class success. It is clear that the etiology of this disease cannot be determined from these preliminary observations. Nutritional, environmental and genetic factors as well as infectious etiologies are possibilities. However, this preliminary description of an enteropathy in larval herring is significant for several reasons. First, it has demonstrated that specific disease processes occur in larval herring and therefore could be important factors in determining recruitment into the adult population. Secondly, this report exemplifies the importance of monitoring marine laboratory animal health where animals are used in controlled experiments. Clearly, serious diseases in laboratory animals may increase experimental variability or contribute extraneous effects which confound results. Finally, this study, in addition to others cited in the introduction, demonstrates that the laboratory study of larval animals is an important tool for the examination of factors which may affect populations in nature.

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