Pituitary Activation by Bacterial Endotoxins in the Rainbow Trout (Salmo gairdneri)

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Endotoxins from Escherichia coli and Aeromonas salmonicida caused marked cortisol production in the rainbow trout (Salmo gairdneri).

Administering bacterial endotoxins to homeothermic vertebrates will regularly cause rapid increases in plasma cortisol, probably due to pituitary release of adrenocorticotropicin (ACTH) (1, 2, 5). Poikilotherms have not been studied, but Kimball et al. (1) concluded that pituitary activation is independent of the endotoxins' pyrogenic effect. Thus, it seemed reasonable that a similar effect would occur in fishes.

Groups of 10 yearling rainbow trout held at 10 C were injected intravascularly via the branchial artery with sufficient endotoxin in 0.2 ml of saline to yield a final dose of 25 mg/kg. Either Escherichia coli (Difco, 055-B 5) or Aeromonas salmonicida endotoxin (a fish pathogen) were used. The A. salmonicida endotoxin was prepared from log-phase cells by A. J. Ross of this laboratory [according to the method of Westphal et al. (4)]. Blood samples were taken at 0, 4, 8, and 18 hr and analyzed for cortisol by the fluorometric method of E. Smith and C. A. Muehlbaecher (Clin. Chem., in press). Control fish received saline. The mean cortisol response for the two endotoxins and control is shown in Fig. 1. At 4 hr postinjection, serum cortisol levels were somewhat elevated above the control values, but the differences were not statistically significant (α = 0.10). By 6 hr, both endotoxins had elicited a significantly increased cortisol response (α = 0.05), but after 8 hr only the fish injected with E. coli endotoxin still had significantly higher plasma cortisol levels than the controls. After 18 hr, neither endotoxin series was significantly different from the control fish (α = 0.05).

The small increase in serum cortisol in the control fish during the 18-hr period was due to handling stress.

These results are different from those reported for humans (1) in that the maximum response is delayed by several hours and a much higher (1000 ×) endotoxin dosage is required. The fact that the cortisol level in one endotoxin series eventually fell below that of the controls (Fig. 1) has also been noted in humans (1), although a statement of its statistical significance was not given. The delayed cortisol response in these fish may be explained by the considerably lower temperature (10 C). However, previous work has shown that their response to ACTH is immediate at similar temperatures (3).

Significantly, cortisol production is one of the few metabolic changes induced by endotoxin yet reported in fishes (3). Thus, the role of endotoxins in their gram-negative bacterial diseases remains obscure. However, since the present results were obtained with a poikilothermic vertebrate, the conclusion of Kimball et al. (1) that the febrile response itself is not the cause of pituitary stimulation by bacterial endotoxins is evidently valid.

LITERATURE CITED