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The effects of adrenaline on heart rate and blood pressure in Salmo gairdneri at two temperatures

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The cardiovascular response of rainbow trout to adrenaline was studied at 14 °C and at 7 °C after a 36 h acclimatization period. Before adrenaline injection the heart rate was higher at 14 °C than at 7 °C ($Q_{10} = 2.5$). The pulse pressures and the mean aortic blood pressures were the same at both temperatures. Adrenaline had nearly no effect on the heart rate, but increased the pulse pressure drastically at both temperatures. Furthermore, the mean aortic blood pressures rose at both temperatures, indicating increased systemic vascular resistance after adrenaline injection. The pressure gradient across the gills decreased at both temperatures after adrenaline injection, indicating decreased branchial resistance.

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1. Introduction

The nervous and hormonal control of cardiovascular function in fish has been quite thoroughly studied (for reviews see Randall 1970, Johansen 1979). The effects of adrenaline on cardiovascular function in particular have been extensively documented. From the work on gill perfusates, head perfusates and whole fish (Rankin & Maetz 1971, Stevens et al. 1972, Wood 1974, Forster 1976, Girard & Payan 1976, Payan & Girard 1977, Holbert et al. 1979, Peyraud-Waitzenegger 1979, Booth 1979, Pettersson & Nilsson 1980, Wahlqvist & Nilsson 1980) it is clear that adrenaline causes a systemic vasoconstriction and a branchial vasodilation, usually after a short-lasting vasoconstriction. However, the effects of temperature on the cardiovascular function and on the adrenaline response are less well known. Laffont & Labat (1966) showed that at low temperatures (1—8 °C) an intravenous injection of adrenaline caused bradycardia but at high temperatures (9—20 °C) tachycardia in carp. Peyraud-Waizenegger et al. (1980) have found that the responses to adrenaline in summer (at 16 °C) were hyperventilation and tachycardia and in winter (at 8 °C) hypoventilation and bradycardia in eel. Heath & Hughes (1973) have shown that the heart frequency and ventral blood pressure increase considerably in acute heat stress from 15 °C upwards and drop markedly near the lethal limit around 25 °C in rainbow trout. Stevens et al. (1972) also found a marked increase in the heart rate of lingcod when the ambient temperature was increased, but the stroke volume was unaltered.

During late spring and early summer the water temperature in Finnish fish farms often fluctuates rapidly. To simulate the natural conditions, the temperature acclimatization time was kept short when investigating the adrenaline response of rainbow trout in relation to temperature change. The dorsal and the ventral aortic blood pressure, the heart frequency and the pulse pressure were determined at two temperatures before and after the injection of adrenaline into the blood stream of the fish.

2. Material and methods

The experiments were carried out at Laukaa Fish Culture Research Station in May—June 1980 on four 3-year old rainbow trout (*Salmo gairdneri* Richardson, 709 ±150 g, 38 ± 3 cm) obtained from a commercial fish farm (Savon Taimen Oy). Throughout the experiment lake water was used with a flow of 2 l/min/kg. The water quality was as follows: pH 7.0—7.1, $P_{CO_2} < 65$ Pa, oxygen saturation 100—110 % and specific conductivity at 20 °C 37—38 μS cm^{-1}.

The fish, taken from naturally fluctuating temperature conditions (10—14 °C), were cannulated via the dorsal and ventral aortae as described earlier by Soivio et al. (1975, 1981). After the operation they were allowed to recover for one week at the experimental temperature (14 ± 1 °C). The fish were fed Ewos pelleted trout food ad libitum daily until 48 h before the experiment, when the fish were enclosed in individual restrainers (Soivio et al. 1975).

The experiment was started at 14 °C by recording alternately the dorsal and ventral blood pressures of undisturbed fish with a Statham P23Db pressure transducer connected to a Honeywell Electronic 19 recorder. After a minimum of 10 min of blood pressure recordings, 1 ml adrenaline solution (concentration 0.5 μg/ml) was injected into the blood stream of the fish via the dorsal aortic cannula (Adrenalin, Medica, 1 mg/ml, Vnr. 450957, diluted with 0.9 % NaCl solution). New adrenaline solution was prepared each day. This injection of adrenaline gave a 0.12—0.15 μM concentration in the blood of the fish (4—5 nmol/kg fish). This concentration was selected because it is in the same range as that observed by Nakano & Tomlinson (1967) from the blood plasma of disturbed rainbow trouts. Before the experiments control injections were carried out by injecting 1 ml of 0.9 % NaCl via the dorsal aortic cannula. No effects on the blood pressure were observed after the injection. Immediately after the injection of adrenaline the dorsal and ventral aortic blood pressures were again alternately recorded for at least 10 min after the injection. After the blood pressure from all the fish had been recorded, their ambient temperature was lowered from 14 °C to 7 °C within 12 h, and the fish allowed to acclimatize to the new temperature for 36 h before the experiment was repeated at this lower temperature exactly as at 14 °C.

From the blood pressure recordings the mean dorsal (P_d) and ventral (P_v) aortic pressures according to Burton (1972): $(P_{syst} + 2P_{diast})/3$, the difference between the mean pressures, the ventral pulse pressures and the heart rates were calculated at the two temperatures before and after the injection of adrenaline.

The statistical analyses are based on the paired *t*-test.

3. Results

The heart rates at the two temperatures before and after the adrenaline injection are given in Table 1. The effect of temperature on the heart rate was pronounced; it was almost twice as much at 14 as at 7 °C. The Q_{10} value for heart rate was 2.5. Compared to the effects of temperature change, the effects of adrenaline on the heart rate were minimal. Although 3 out of 4 fish showed a slight bradycardia at 7 °C and 3 of the 4 fish showed slight tachycardia at 14 °C, the changes were negligible. Already within 5 minutes of the adrenaline injection the heart rate had returned to the pre-injection level.

The dorsal and ventral aortic mean blood pressures and the difference between these pressures are given in Fig. 1. Before the injection of adrenaline the dorsal and ventral pressures were the same at both temperatures. Within 30 s of the injection of adrenaline both the dorsal and ventral aortic pressures rose ($P < 0.05$) at both temperatures; at 14 °C, however, both pressures rose more and remained higher ($P < 0.05$) throughout the experiment than at 7 °C. The ventral aortic pressure reached a maximum at 28 ± 5 s (mean ±*SE*) at 14 °C and at 32 ± 8 s at 7 °C and was restored to the pre-injection level within 10 min at 7 °C, but remained higher ($P < 0.05$) at 14 °C still 10 min after the injection.

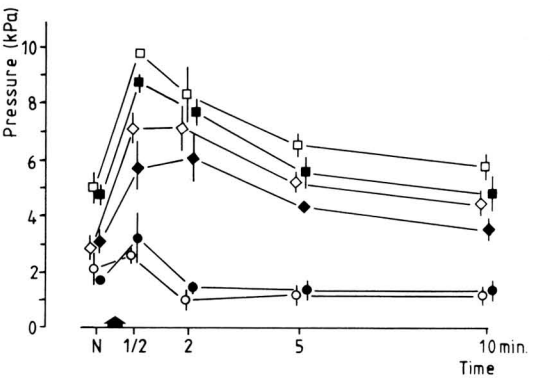

Fig. 1. Blood pressures prior to (N) and following the adrenaline injection (arrow). Open symbols: 14 °C, solid symbols: 7 °C; circles: ventral — dorsal pressure difference, diamonds: dorsal pressure, squares: ventral pressure. Means ±*SE* (vertical bars).

The dorsal pressure started to decrease first 2 min after the injection, and decreased regularly, again reaching the pre-injection level within 10 min at 7 °C, but remaining at a higher level ($P < 0.05$) at 14 °C.

The differences between the two temperatures in the mean pressure changes caused by adrenaline seem to originate only in the systemic side of

Table 1. The heart rates and ventral aortic pulse pressures (mean ±*SE*) of rainbow trout at 7 °C and 14 °C before and after injections of adrenaline into the dorsal aorta ($n = 4$).

	7 °C	14 °C	*P* 7/14 °C
Heart rate			
before	36.0±2.12	66.8±4.31	<0.001
after	33.8±1.89	71.3±1.89	
P	NS	NS	
Pulse pressure			
before	6.80±2.02	6.00±2.57	NS
after	15.44±5.73	15.44±5.73	
P	<0.05	<0.05	

the circulation, as the $P_V - P_D$ value was more or less the same at both temperatures. Immediately after the adrenaline injection the $P_V - P_D$ value rose slightly, due to the slower increase in the dorsal pressure than in the ventral pressure. However, since the ventral pressure started to decrease at 30 s after injection and the dorsal pressure 1 1/2 minutes later, the $P_V - P_D$ value decreased rapidly at this stage and was lower ($P < 0.05$, both temperatures combined) from 2 to 10 minutes after the injection than before it. The pulse pressures, given in Table 1., were similar at both temperatures, and increased markedly ($P < 0.05$) due to the injection of adrenaline. No differences between the temperatures were apparent.

4. Discussion

The findings of this study can be divided into three categories: the effect of temperature as such on the cardiovascular parameters, the effect of adrenaline as such on these parameters, and the influence of temperature on the responses to adrenaline injections.

The effect of temperature is clear-cut; it increases the heart rate markedly, but has little effect on the other parameters studied. The Q_{10} value of 2.5 for the heart rate is in close agreement with the Q_{10} value of 2—3 for the lingcod (Stevens et al. 1972), but much higher than that reported by Heath & Hughes (1973) for rainbow trout in acute heat stress above 15 °C (Q_{10} value of 1.5—1.6). Probably the higher temperatures used in the latter study explain the differences, since in that study the other cardiovascular parameters changed differently from those in the present study; both the dorsal and the ventral blood pressures rose with increasing temperature, the dorsal pressure less than the ventral pressure — this also indicating increased branchial resistance. Moreover, Heath & Hughes were studying heat stress, whereas in the present study we confined ourselves to the temperature near the optimal growth range for rainbow trout (Wurtsbaugh & Davis 1977). The relatively high Q_{10} value for the heart rate suggests that, other factors being constant, the increase in cardiac output may contribute quite considerably to the increased oxygen demand due to the increasing temperature. Other cardiovascular factors in fact appear to stay constant within this temperature range. Stevens et al. (1972) found that in the lingcod the stroke volume was practically unaffected by temperature, and this study showed that in the rainbow trout both the pulse pressure and the dorsal and ventral aortic pressures remain constant at the temperatures studied. For this to happen, the vascular resistance must decrease; this conclusion was also reached by Stevens et al. (1972). One factor decreasing the vascular resistance is the decrease in the viscosity of blood due to the increased temperature. Furthermore, the circulatory patterns may change with increasing temperature (cf. Nikinmaa et al. 1981), possibly increasing the number of open capillaries which can then 'absorb' the increased cardiac output without a change in the blood pressure. This would also fit in with the difference between this study and that of Heath & Hughes (1973); when the temperature continuously rises, a point is reached when the decrease in the viscosity of blood and the opening and enlargement of the capillaries cannot "absorb" the increasing cardiac output. At that point the vascular resistance again increases, and the dorsal and ventral aortic blood pressures increase. One direct measurement of this is in the study of Nikinmaa et al. (1980), who reported a decrease in the proportion of capillary blood in the gills at a high acclimatization temperature (18 °C). Such a change could increase branchial resistance.

The effects of adrenaline on the cardiovascular function observed in this study confirm many earlier observations; the branchial resistance decreased after a rapid initial increase, and the resistance in the systemic circulation increased (for reviews see Randall 1970, Johansen 1979). However, it is notable that the increase in the systemic resistance due to adrenaline was greater at 14° than at 7 °C. This again adds to our knowledge of the different cardiovascular responses to adrenaline at different temperatures (see Introduction). In this study, although slightly expected, tachycardia and bradycardia were not present, and probably therefore the temperature increase did not influence the pulse pressure, in contrast to the findings of Peyraud-Waitzenegger et al. (1980) on eel.

It is notable that, according to Randall & Stevens (1967), small concentrations of adrenaline caused bradycardia and large concentrations tachycardia at 10—12 °C in *Onchorhyncus kisutch.* Thus the responses to adrenaline at low temperature resemble the low-dose response and at high temperature the high-dose response in eel (Peyraud-Waitzenegger et al. 1980) and carp (Laffont & Labat 1966), and slightly also in rainbow trout in the present study. Since this study

shows that the same concentration of adrenaline causes a greater pressure rise i.e. is a more potent modifier of vascular resistance at the higher temperature, it seems that at the low temperatures some of the receptors are less sensitive to adrenaline than at higher temperatures. Increase in temperature may lower the response threshold of these receptors, and elicit the "high-dose" response at lower concentrations than originally.

An even more likely explanation of the difference in the systemic pressure after adrenaline injection between the temperatures is probably that the response of the systemic vasculature to adrenaline is the same at both temperatures. However, at the higher temperature adrenaline causes an increase in the erythrocyte volume in post-branchial blood (Nikinmaa 1981) and this increase increases the systemic resistance on top of the actual vascular effect of adrenaline.

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