

CHANGES IN BLOOD PRESSURE, HEART RATE AND BREATHING RATE DURING MODERATE SWIMMING ACTIVITY IN RAINBOW TROUT

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(Received 4 November 1966)

INTRODUCTION

There is a paucity of data concerning the changes in circulation occurring during swimming in fish. Data reported in the literature have been obtained from either restrained, anaesthetized or operated fish (Greene, 1904; Hart, 1945; Johansen, 1962; Robertson *et al.* 1966). Such procedures can have marked effects on fish (Fry, 1957). It is therefore surprising that little attention has been directed towards measuring parameters of circulation and respiration in unrestrained, unanaesthetized, intact fish (Randall, Smith & Brett, 1965). Recently, methods have been developed for measuring blood pressures using indwelling cannulae (Smith & Bell, 1964; Holeton & Randall, 1967). These techniques, combined with those of Brett (1964) which use varying water velocities to produce variations in swimming speeds in fish, permit measurement of various parameters of circulatory and respiratory systems in swimming fish.

The present study was undertaken to examine the effects of swimming on circulation and respiration using free swimming, unanaesthetized rainbow trout.

METHODS

The experiments were carried out on thirty-nine hatchery-raised rainbow trout (*Salmo gairdneri*) weighing between 200 and 600 g. The fish were fed three times weekly with a food containing beef liver, yeast and canned salmon with sulphamerazine added (1 g./10 lb. food) to reduce the incidence of furunculosis (Snieszko, Gutsell & Friddle, 1948).

A fish was anaesthetized by immersion in water containing 1:10,000 tricaine methanesulphonate (MS-222). The ventral aorta, dorsal aorta and opercular cavity were cannulated as described by Holeton & Randall (1967) and Smith & Bell (1964). The opercular cannulation permitted the measurement of the rate of respiration while the vascular cannulae allowed measurement of blood pressure in the dorsal and ventral aorta; that is, blood pressure afferent and efferent to the gills. The subintestinal vein was cannulated with a polyethylene T-cannula (Clay-Adams PE 50) to record venous blood pressure. In order to estimate venous blood flow the subintestinal vein was cannulated towards the tail and the vessel was tied off anteriorly. Flow was determined as the time taken to fill a 0.1 ml. heparinized pipette. All cannulae were 2 ft. long and filled with heparinized (1 unit/ml.) Courtland saline for fresh-water teleosts (Wolf, 1963).

When all cannulae were in place the fish was placed in a respirometer similar to that described by Brett (1964). Briefly, it consists of a Perspex tube through which water is recirculated. The fish swims against the current at a rate which is dependent on the rate of water flow through the tube, which in turn is determined by a variable speed pump. Fresh water was continuously added to the system (10 l./min.) to ensure an adequate oxygen supply.

The fish was allowed to recover from the operative procedures in the respirometer for at least 4 hr. The water was gently recirculated during this period, but the water velocity was so low that the fish could maintain its position in the tube without swimming.

After recording pre-exercise levels, the water velocity was increased stepwise once every minute for 5 min., maintained at a maximum level for 5 min. and then decreased stepwise once every minute for 5 min.

Ventral aortic, dorsal aortic and venous pressures were measured continuously throughout the experiment and for 30 min. after the cessation of swimming. Opercular pressures, also measured over the same time period, gave a measure of respiration rate. The heart rate was determined from the pulse rate on both the ventral aortic and dorsal aortic blood-pressure records. Pressures were recorded using Statham P 23AA and P 23BB pressure transducers which had been calibrated against a column of water and displayed on a Beckman type R Dynograph.

The temperature of the dechlorinated fresh water during the experiments was the same as that in the holding tanks. The temperature was 10–12° C., however, it never varied more than $\pm 0.5^\circ$ C. during any one experiment.

RESULTS

In general the fish swam continuously throughout the period of maximum water velocity. The tail-beat frequency during this period was about 160 beats/min.

Before exercise ventral aortic blood pressure was 40/32 mm. Hg, dorsal blood pressure was 29/25 mm. Hg and venous pressure was 9 mm. Hg (Fig. 1, Table 1).

During exercise ventral aortic blood pressure, both systolic and diastolic, increased by about 40%. The dorsal aortic systolic pressure increased by about 16% and the diastolic pressure by about 21%. During recovery from exercise both ventral aortic and dorsal aortic blood pressures decreased gradually to the pre-exercise level within 30 min. (Fig. 1).

There was no pulse pressure in the subintestinal vein, but during exercise there were irregular rapid increases in blood pressure (Fig. 3). The maximum venous blood pressure recorded during each period of the experiment is shown in Fig. 1 as the peak venous pressure. The minimum venous pressure did not change significantly during the exercise period. To demonstrate that the venous pressure changes recorded were not produced by bending of the cannula during swimming, dummy cannulae were placed in the coelom and outside the body wall. The pressure changes observed in the venous T-cannula did not occur in the dummy cannulae. Pressure changes in the dummy cannulae were associated with the body movements and were used to estimate tail-beat frequency.

The pre-exercise heart rate and breathing rate were 24 and 77/min. respectively.

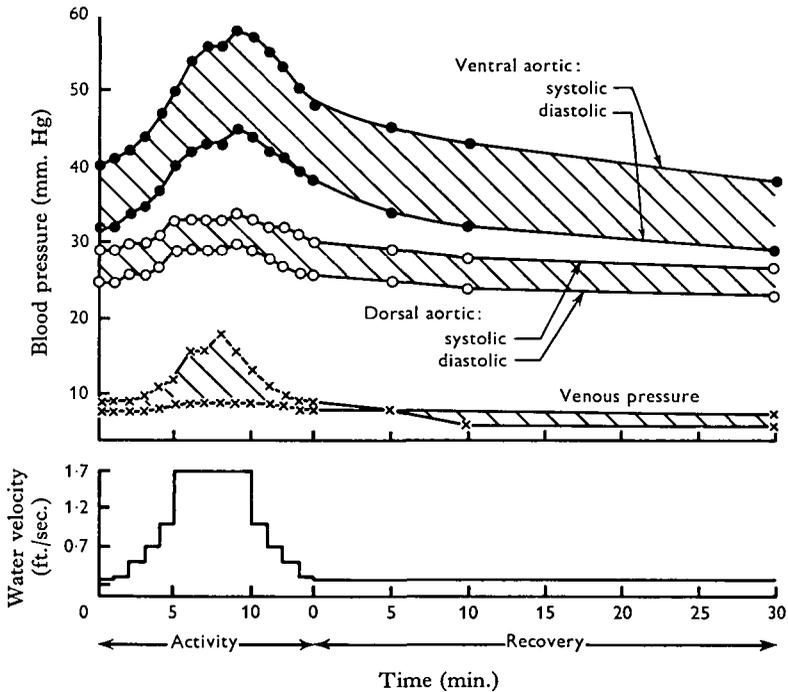


Fig. 1. Changes in blood pressure in the ventral aorta, dorsal aorta, and subintestinal vein during and after moderate swimming activity. Points are mean values from at least ten fish.

Table 1. Blood pressures, heart rates and respiratory rates during moderate exercise in rainbow trout. Values given are means \pm standard errors

Condition	Water velocity (ft./sec.)	Blood pressure (mm. Hg)						Rate (no./min.)	
		Ventral aorta (n = 13)		Dorsal aorta (n = 16)		Subintestinal vein (n = 10)		Heart (n = 29)	Respiratory (n = 10)
		Systolic	Diastolic	Systolic	Diastolic	Peak	Minimum		
Pre-exercise	0.23	40 \pm 1.0	32 \pm 0.9	29 \pm 1.2	25 \pm 1.0	9 \pm 1.0	8 \pm 1.1	47 \pm 2.5	77 \pm 1.9
Exercise	0.31	41 \pm 1.2	32 \pm 1.1	29 \pm 1.2	25 \pm 1.1	9 \pm 1.1	8 \pm 1.2	47 \pm 2.4	76 \pm 1.5
	0.50	42 \pm 1.4	34 \pm 1.3	30 \pm 1.2	26 \pm 1.1	9 \pm 1.4	8 \pm 1.0	48 \pm 2.4	78 \pm 1.3
	0.71	44 \pm 1.8	35 \pm 1.5	30 \pm 1.1	26 \pm 1.0	10 \pm 1.3	8 \pm 1.1	50 \pm 2.3	78 \pm 2.2
	1.00	47 \pm 1.6	37 \pm 1.4	31 \pm 0.9	27 \pm 0.9	11 \pm 1.5	8 \pm 1.0	50 \pm 2.3	87 \pm 2.2
	1.70	50 \pm 1.6	40 \pm 1.6	33 \pm 0.9	29 \pm 0.9	12 \pm 1.8	9 \pm 1.2	52 \pm 2.2	94 \pm 2.1
	1.70	54 \pm 1.1	42 \pm 1.3	33 \pm 1.1	29 \pm 1.0	16 \pm 1.7	9 \pm 1.2	54 \pm 2.2	98 \pm 1.9
	1.70	56 \pm 1.6	43 \pm 1.8	33 \pm 1.1	29 \pm 1.1	16 \pm 2.7	9 \pm 1.0	53 \pm 2.2	100 \pm 2.1
	1.70	56 \pm 1.8	43 \pm 1.9	33 \pm 1.1	29 \pm 1.1	18 \pm 5.0	9 \pm 0.7	54 \pm 2.2	102 \pm 2.7
	1.70	58 \pm 1.8	45 \pm 1.9	34 \pm 1.0	30 \pm 1.4	16 \pm 3.0	9 \pm 0.8	54 \pm 2.3	99 \pm 2.3
	1.00	57 \pm 2.0	44 \pm 1.9	33 \pm 1.2	29 \pm 1.3	13 \pm 1.6	9 \pm 0.9	54 \pm 2.2	96 \pm 1.5
	0.71	55 \pm 1.9	42 \pm 2.0	32 \pm 1.2	28 \pm 1.2	11 \pm 1.5	9 \pm 1.0	52 \pm 2.3	87 \pm 2.2
0.50	53 \pm 2.1	41 \pm 2.2	32 \pm 1.1	27 \pm 1.2	10 \pm 1.0	9 \pm 1.0	51 \pm 2.4	84 \pm 2.3	
0.31	50 \pm 2.1	39 \pm 2.1	31 \pm 1.1	26 \pm 1.1	9 \pm 1.1	8 \pm 1.1	50 \pm 2.5	81 \pm 2.1	
Post-exercise									
0 min.	0.23	49 \pm 2.2	37 \pm 2.1	30 \pm 1.0	26 \pm 1.0	8 \pm 1.1	8 \pm 1.1	49 \pm 2.5	79 \pm 3.0
5 min.	0.23	45 \pm 2.1	34 \pm 1.9	29 \pm 1.0	25 \pm 1.0	8 \pm 1.1	8 \pm 1.1	48 \pm 2.7	78 \pm 2.2
10 min.	0.23	43 \pm 2.3	32 \pm 1.9	28 \pm 1.0	24 \pm 1.0	8 \pm 1.2	6 \pm 0.9	47 \pm 2.8	78 \pm 1.6
30 min.	0.23	38 \pm 1.5	29 \pm 1.0	27 \pm 1.0	23 \pm 0.9	7 \pm 1.3	6 \pm 1.0	46 \pm 2.8	78 \pm 1.3
60 min.	0.23	35 \pm 0.9	27 \pm 0.7	—	—	—	—	—	—

During exercise the heart rate increased by about 15% and the respiratory rate by about 30% (Fig. 2). Both rates returned to the pre-exercise levels within 30 min. after the exercise period.

Injections of atropine (0.5 ml. of 10^{-3} atropine in Courtland saline) into the pericardial cavity had no effect on the heart rate of the resting trout. The changes in heart rate occurring during swimming were identical both before and after injections of atropine into the pericardial cavity. The dose level of atropine, however, was sufficient to block the cholinergic fibres innervating the heart, since stopping water flow over the gills after administration of atropine did not provoke reflex bradycardia (Randall, 1966).

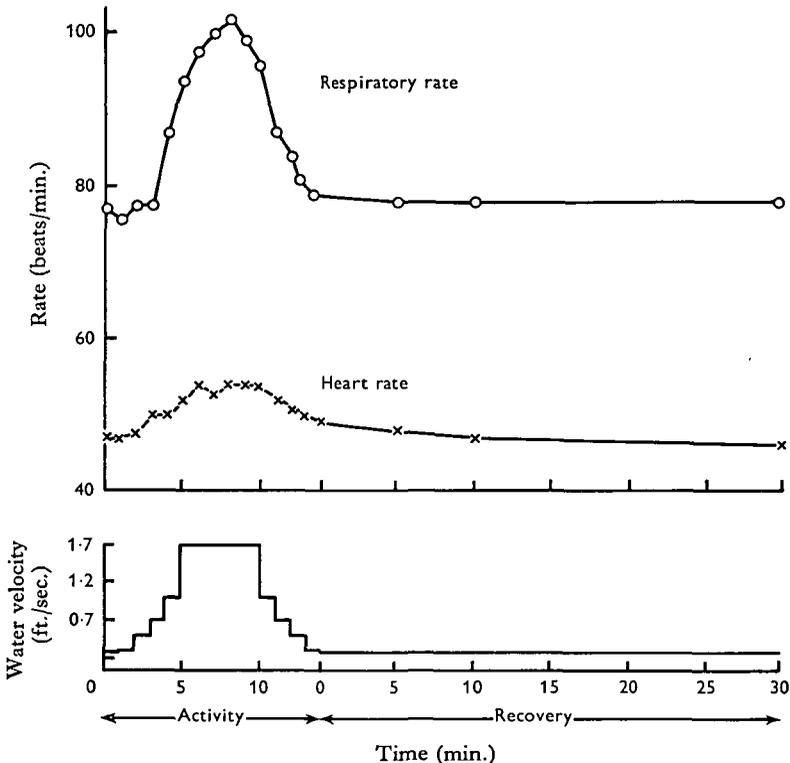


Fig. 2. Changes in heart rate and breathing rate during and after moderate swimming activity. Points are mean values from at least ten fish.

Blood flow in the subintestinal vein appeared to decrease during and after exercise (Table 2). The blood appeared to clot much more readily during and after exercise, so that an increase in viscosity may account for at least a part of the decreased flow measured from the subintestinal vein.

DISCUSSION

There have been very few measurements of blood pressure in unrestrained teleosts. (Randall *et al.* 1965; Holeyton & Randall, 1967). Previous workers have tended to use restrained fish (Greene, 1904; Hart, 1945; Johansen, 1962). In spring salmon Greene

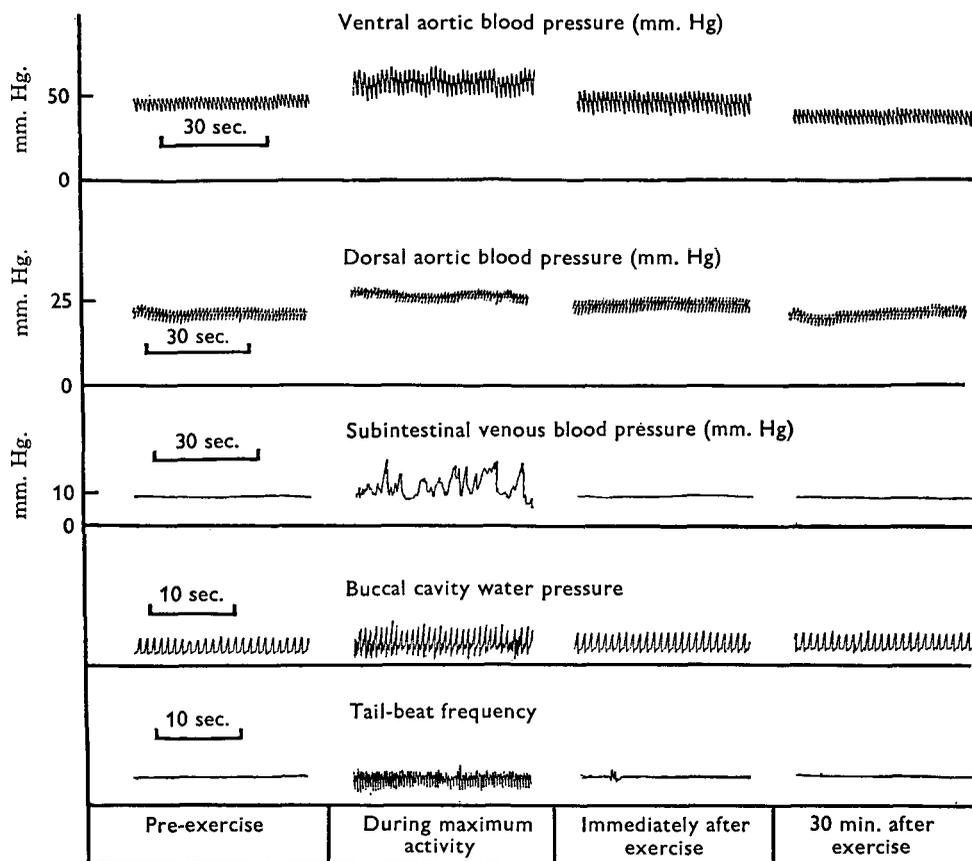


Fig. 3. Typical pressure records from the ventral aorta, dorsal aorta, subintestinal vein, the opercular cavity and a dummy cannula attached to the body wall.

Table 2. Blood flow from caudal musculature measured in subintestinal vein

	\bar{x}	\pm	S.E. (ml./min.)
Pre-exercise	0.114	\pm	0.0453
During exercise	0.094	\pm	0.0336
Post-exercise			
0 min.	0.084	\pm	0.0318
10 min.	0.036	\pm	0.0216
60 min.	0.086	\pm	0.0509

(1904) reported a ventral aortic blood pressure of 77 mm. Hg. He also observed periodic fluxes in blood pressure due to the heart missing every 4th or 5th beat. This has been observed in the eel (Mott, 1951) and in elasmobranchs (Lyon, 1926). It was only observed in the present study after exercise. Johansen (1962) reported a blood pressure of 30 mm. Hg in the bulbus arteriosus of the cod. The fish in his study were restrained and ventral side up, but not anaesthetized. Robertson *et al.* (1966) measured pressures in the ventral aorta of the spring salmon of 82/50 mm. Hg and dorsal aortic pressures of 44/37 mm. Hg. They stopped the water flow over the gills when recording

blood pressure, and this initiated a bradycardia reflex as indicated by the low heart rates. Hart (1945) recorded blood pressures from four species of fresh-water fish that were stunned, placed ventral side up and out of the water. Bulbus pressures recorded ranged from 21/5 mm. Hg in the bullhead to 65/62 mm. Hg in a gizzard shad. He showed that bulbus pressures tended to increase at higher heart rates.

Blood pressures reported here are lower than those recorded by Randall *et al.* (1965) and Holeyton & Randall (1967) from the same species of fish. The techniques are similar in all cases and the differences in blood pressure are probably related either to a seasonal effect (the present study was carried out in the winter whereas the other studies were carried out in summer), to a temperature effect, or to intraspecific differences between the various stocks of fish used. The study of Randall *et al.* (1965) was carried out in sea water whereas the other two studies were carried out in fresh water. A comparison of the results of Randall *et al.* (1965) with those of Holeyton & Randall (1967) would indicate that salinity has only a minor effect on blood pressure. Temperature changes affect heart rate (Grodzinski, 1955; Labat, Raynaud & Serfaty, 1961), an increase in temperature producing an increase in heart rate via an aneural mechanism (Laurent, 1962). Thus it would appear probable that the differences between blood pressures recorded here and those reported by Holeyton & Randall (1967) and Randall *et al.* (1965) are best explained in terms of differences in the temperature of the environment.

Satchell (1965) found that blood flow in the caudal vein increased when an isolated trunk preparation of an elasmobranch was stimulated electrically. He also demonstrated the presence of valves, which prevented the backflow of blood from the caudal veins into the segmental veins during activity. These valves do not appear to be present in the trout, since latex paint will pass into the segmental veins when injected into the caudal vein.

Activity in the trout results in intermittent increase in blood pressure in the subintestinal vein, but a decrease in blood flow through this vessel. There must, however, be an increase in venous return to the heart during swimming as there is an increase in cardiac output (Stevens & Randall, 1967). The increased pressure but decreased flow through the subintestinal vein indicates that venoconstriction is occurring, perhaps in the liver through which the blood from the subintestinal vein passes before entering the heart, and that the increased venous return to the heart during swimming is being shunted through some other venous pathway.

The results of the experiments using atropine injections indicate that the increases in heart rate occurring during swimming in the trout are aneural in origin. There are no sympathetic fibres innervating the fish heart (Cousteaux & Laurent, 1957), and all efferent nerves appear to be cholinergic (Randall, 1966) and can be blocked with atropine. A decrease in vagal tone during activity, however, may account for a part of the cardio-acceleration in some species of fish under certain conditions.

In a single experiment carried out on the sucker (*Catostomus macrocheilus*) injections of atropine into the pericardial cavity of the resting fish produced a large increase in heart rate (Table 3). During swimming there were increases in heart rate of up to 75% of the resting level. The heart rate of the fish during activity was similar before and after the injection of atropine into the pericardial cavity. Thus in the sucker there is vagal tone to the heart under normal conditions, which is released during

activity. Therefore, the magnitude of the increase in heart rate during swimming in fish is related to the extent of vagal tone. In the trout there is normally no vagal tone, and only a small increase in heart rate during swimming; in the sucker there is extensive vagal tone and a large increase in heart rate during activity. Thus there appears to be a neural and an aneural mechanism operating in controlling heart rate. The increases in heart rate during activity observed in the trout were aneural, whereas those in the sucker had a neural as well as an aneural component.

Increases in vagal activity during hypoxia result in bradycardia (Randall, 1966). Locomotory activity during hypoxia results in a large increase in heart rate in the trout, so that a fish in hypoxic conditions has the same heart rate during activity as a fish in an air-saturated environment. Thus it would seem that the larger increases in heart rate (but from a lower initial level), observed in a trout in a hypoxic environment are due in part to a release of vagal tone.

Table 3. Results of a single experiment on a sucker which show the effect of exercise and atropine on respiratory rate and heart rate

	Heart rate	Respiratory rate
Pre-exercise	38	37
During exercise	74	60
Post-exercise	35	18
Atropine into pericardial cavity	55	18
During exercise	67	64
Post-exercise	61	34

Johansen (1962) reported that activity in the cod was not associated with any changes in heart rate. He also demonstrated that experimental increases in venous return caused increases in stroke volume rather than changes in heart rate. Labat *et al.* (1961) reported that increasing venous return in the catfish caused an increase in heart rate that could be abolished with atropine. In the experiments reported here no correlation could be found between the increased heart rate observed and the intermittent increases in venous pressure recorded from the subintestinal vein.

Nakano & Thomlinson (personal communication) have measured increases in blood catecholamines during activity in rainbow trout. Intravenous injections of physiological doses of epinephrine (1.0–5.0 μg .) caused an increase in heart rate and blood pressure. Shepherd (1965) observed a cardio-acceleration in dogs with denervated hearts which was independent of the level of circulating catecholamines. Jensen (1963) have isolated a cardio-accelerator substance from the hagfish heart which is not a catecholamine. Breton *et al.* (1964) have observed an increased heart rate after electrical stimulation of an isolated teleost heart. Each of these observations indicates the possibility of an endogenous supply of a cardio-accelerator substance which is released by some aneural mechanism and which causes the observed increase in heart rate in the trout during swimming. At present, however, the nature of the mechanism producing the aneural cardio-acceleration of the trout heart during swimming is not understood.

Giaja & Markovic-Giaja (1957) reported that activity did not increase the rate of respiration in teleosts, but Saunders (1962) demonstrated a large increase in ventilation

volume during swimming in a number of teleosts. The increases in respiration rate recorded in this study could not be correlated with any changes in P_{O_2} or P_{CO_2} in the venous or arterial blood (Stevens & Randall, 1967). Because the changes in breathing rate occurred concomitantly with changes in muscular activity, the most logical explanation for the regulation of breathing rate during swimming in fish would appear to be a mechanism similar to the Harrison joint-tendon reflex of mammals.

SUMMARY

1. Changes in blood pressure in the dorsal aorta, ventral aorta and subintestinal vein, as well as changes in heart rate and breathing rate during moderate swimming activity in the rainbow trout are reported.
2. Blood pressures both afferent and efferent to the gills increased during swimming and then returned to normal levels within 30 min. after exercise.
3. Venous blood pressure was characterized by periodic increases during swimming. The pressure changes were not in phase with the body movements.
4. Although total venous return to the heart increased during swimming, a decreased blood flow was recorded in the subintestinal vein.
5. Heart rate and breathing rate increased during swimming and then decreased when swimming ceased.
6. Some possible mechanisms regulating heart and breathing rates are discussed.

This work was supported by grants from the National Research Council and the B.C. Heart Foundation. We wish to thank the Pacific Biological Station, Nanaimo, and particularly Dr J. R. Brett, for support and facilities made available for this investigation.

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