authors cannot give any satisfactory explanation of these phenomena. We propose here 3 mechanisms for the enhanced prostacyclin plasma concentration observed by Fletcher et al.

1. A cyclo-oxygenase stimulation, in the presence of a natural free radical scavenger, such as uric acid, resulting from the ability of lidocaine to accelerate lipoperoxidation[1]. 2. The inhibition, by lidocaine, of the 15-OH-prostaglan-deshydrogenase, described by Tai et al.[13], may contribute to enhancing the tissue and plasma prostanoid concentrations. 3. The impairment of phospholipasic activities by lidocaine[14,15] is also possible. However, it must be underlined that we employed exogenous arachidonate, so that this mechanism must be excluded in our experiments.

Conclusions. The pro-lipoperoxidant effect of lidocaine is associated with a stimulant action on cyclo-oxygenase activity, observable particularly in the presence of a radical scavenger, such as uric acid. This property is confirmed by in vitro and in vivo experiments. However, in in vivo conditions, lidocaine may enhance PGs biosynthesis by another pathway, such as phospholipase and PG 15-OH-deshydrogenase inhibition.

1 Deby, C., Deby-Dupont, G., Pincemail, J., and Neuray, J., Experientia *40* (1984), in press.
2 Deby-Dupont, G., Hans, P., Lochet, E., Neuray, J., and Deby, C., 5th International Conference Prostaglandins, Florence, May 1982, Abstract No. 181.
3 Hemler, M.E., Cook, H.W., and Lands, W.E.M., Archs. Biochem. biophys. *193* (1979) 340.
4 Egan, R.W., Paxton, J., and Kuehl, F.A., J. biol. Chem. *251* (1976) 7329.
5 Deby, C., Deby-Dupont, G., Noel, F.X., and Lavergne, L., Biochem. Pharmac. *30* (1981) 2243.
6 Deby, C., Descamps, M., Binon, F., and Bacq, Z.M., Biochem. Pharmac. *24* (1975) 1089.
7 Deby, C., Noel, F.X., Chapelle, J.C., Van Caneghem, P., Deby-Dupont, G., and Bacq, Z.M., Bull. clin. Sci. Acad. R. Belg. *64* (1978) 644.
8 Deby, C., Van Caneghem, P., and Bacq, Z.M., Biochem. Pharmac. *27* (1977) 613.
9 Deby, C., Deby-Dupont, G., Hans, P., Pincemail, J., and Lochet, E., Archs int. Physiol. Biochim. (1983) in press.
10 Bourgain, R.H., Deby, C., Deby-Dupont, G., and Andries, R., Biochem. Pharmac. *31* (1982) 3011.
11 Fletcher, J.R., Carey, L.C., and Ramwell, P.C., Prog. Lipid Res. *20* (1981) 869.
12 Armstrong, C., Fletcher, J.R., and Ramwell, P.W., Prostaglandins *19* (1980) 6.
13 Tai, H., and Hollander, C.S., Adv. Prostagl. Thromboxane Res. *1* (1976) 171.
14 Waite, M., and Sisson, P., Biochemistry *11* (1974) 3098.
15 Scherphof, G.L., Scarpa, A., and Van Toerenberg, A., Biochim. biophys. Acta *270* (1972) 226.

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Anaerobic metabolism during activity in the rainbow trout *(Salmo gairdneri)*

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Summary. Rainbow trout 27 g b.wt were trained to swim in a water tunnel at 1.1 body lengths · sec^{-1} (10 °C). Swimming speed was increased over 60 sec to either 2.3, 3.8, 5.3, 6.1 or 7.0 body lengths · sec^{-1} and fish were freeze clamped in liquid nitrogen. Other fish were sampled after a further 5 min steady swimming. Anaerobic energy production (mmoles · ATP · kg^{-1} min^{-1}) calculated from whole body lactate concentrations increased from 0.23 at 2.3 body lengths sec^{-1} to 1.6 at 7.0 body lengths sec^{-1}. Lactate concentrations decreased for periods of swimming greater than 20 min partly due to a catabolism of lactate.

The energy source for swimming is critically dependent on both the intensity and duration of effort[2]. For reptiles and amphibians whole body lactate analysis has been used to determine the importance of anaerobic metabolism during activity[3,4]. In rainbow trout, only the anaerobic contribution to initial activity may be calculated due to a significant catabolism of lactate under steady state conditions[5].

Materials and methods. Rainbow trout (*Salmo gairdneri* Richardson), 13.4±0.2 cm length and 27.2±1.5 g b.wt were obtained from North East Fife Fish Farm, Scotland. They were held in fresh water and fed on proprietary trout pellets. Swimming experiments were carried out in an open-top flume (150 cm long × 25 cm diameter) as described by Johnston and Moon[6]. Temperature in both holding tanks and exercise chamber was maintained at 10±0.5 °C. Groups of 6–8 fish were conditioned to swimming in the chamber at 1.1 body lengths · sec^{-1} for at least 3 days prior to experiments. Following this training period, water flow was increased to speeds equivalent to 2.3, 3.8, 5.3, 6.1 or 7.0 body lengths · sec^{-1} over a period of 60 sec and half the fish were sampled. Other groups of fish were allowed to swim for a further 5 min prior to sampling. Only fish exhibiting normal swimming behavior were sampled. Fish that struggled or fell back against the restraining barrier (∼ 10%) were removed from the chamber and discarded. Fish were stunned and freeze-clamped in liquid nitrogen (−159 °C) as previously described[5]. Although a finite time is required to freeze the whole carcass, both initial and final samples were treated in a similar manner and are subject to the same errors. Lactate was determined in duplicate from perchloric acid extracts prepared from the whole carcass[5]. Preliminary experiments established that lactate concentrations continued to increase during the 1st 8–20 min swimming and thereafter declined as steady state conditions were obtained.

Results. Total body lactate concentration was 7.9±0.5 µmoles · g^{-1} in fish swimming at 1.1 body lengths · sec^{-1}. Figure 1 shows lactate concentrations after acceleration to either 2.3, 3.8, 5.3, 6.1 or 7.0 body lengths · sec^{-1} and following a further 5 min swimming. The rate of lactate production was much higher during acceleration than during steady swimming, varying from 3.8 mmoles kg^{-1} · min^{-1}

at 2.3 body lengths · sec^{-1} to 21.5 mmoles kg^{-1} · min^{-1} at 7.0 body lengths · sec^{-1}. The $\log_{10}$ net lactate production during the 1st 5 min swimming was found to be linearly related to swimming speed. The calculated ATP turnover rates from anaerobic glycolysis during this period are shown in figure 2.

Discussion. Experiments with small mammals and lizards have shown that 1-2 min are required for oxygen consumption to stabilize at a new level following an increase in running speed[7,8]. In salmon, this period is somewhat longer depending on temperature and the increment between increases in swimming speed[10,11]. The actual oxygen consumption (VO_2) of the fish under the pre-steady state conditions in our experiments is unknown. An estimate of $\dot{V}O_2$ under steady state conditions is available for rainbow trout of this size range[11]. Maximum $\dot{V}O_2$ for 27 g fish (corrected for differences in temperature assuming a Q_{10} for aerobic metabolism of 2.1[12]) is around 444 mg O_2 kg^{-1} · h^{-1} equivalent to an ATP turnover rate of 1.5 mmoles ATP kg^{-1} · min^{-1} (Roa[11]). Lactate production during the initial acceleration to 2.3 body lengths · sec^{-1} is equivalent to an ATP turnover rate of 2.7 mmoles · kg^{-1} · min^{-1} (assuming 0.016 mmoles ATP/mg lactate[3]). This falls to 0.2 mmoles ATP kg^{-1}min^{-1} after 5 min swimming. Anaerobic metabolism during this acceleration phase is greater than the predicted net energy expenditure from steady state levels of $\dot{V}O_2$ (1.4 mmoles · kg · $^{-1}$min^{-1}). It seems likely that the contribution of anaerobic metabolism to total energy requirements declines with time as oxygen uptake increases and the fish settle down to a more steady and economical mode of swimming. Estimates of the average anaerobic contribution during the 1st 5 min range from 17% of steady state $\dot{V}O_2$ at 2.3 body lengths · sec^{-1} to 52% at 7.0 body length · sec^{-1}.

In some early studies of lactate metabolism in salmonids elevated plasma lactate concentrations were found even at moderate swimming speeds (2-4 body lengths · sec^{-1})[7]. More recently we have shown that plasma lactate levels are actually reduced compared to tank-rested fish, in brook trout trained to swim continuously at speeds of up to 4.0 body lengths · sec^{-1} for a month[6]. Clearly, exercise training and prior handling have an important influence on activity metabolism in fish. Wokoma and Johnston[5] used a similar experimental design to that in the present study. They found that for 50 g rainbow trout trained to swim at 0.9 body length · sec^{-1} lactate concentrations increased steadily during the 1st 10 min swimming at 3.5 body lengths · sec^{-1} and thereafter declined. Following 24 h swimming, whole body lactate concentrations were not significantly different from 'rested' fish[5]. It would appear that lactate is an important substrate for oxidative decarboxylation in certain tissues with high mitochondrial volume densities such as red muscle and liver[14]. There is a net increase in lactate during the initial stages of activity since lactate has to diffuse from anaerobic tissues such as white skeletal muscle and be transported via the circulation to sites of catabolism. Hudson[15] obtained electromyographical evidence that both red and white muscles are recruited in rainbow trout at speeds in excess of 1.3 body lengths · sec^{-1}. An interesting possibility is that certain tissues (notably white muscle) may be operating largely anaerobically even at speeds at which the whole animals is in oxygen balance.

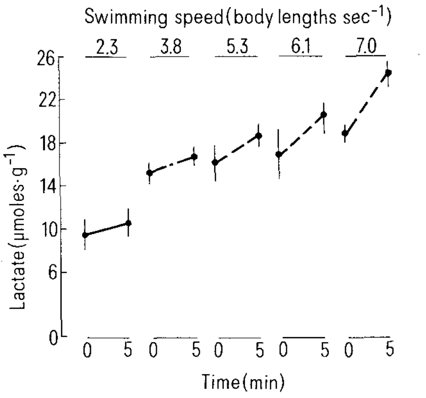

Figure 1. Whole body lactate concentrations (μmoles · g^{-1}) in rainbow trout exercise conditioned at 1.1 body lengths · sec^{-1}. Values represent concentrations (Mean ± SE 20 fish at each speed) following acceleration to either 2.3, 3.8, 5.3, 6.1 or 7.0 body lengths · sec^{-1} and after a further 5 min steady swimming.

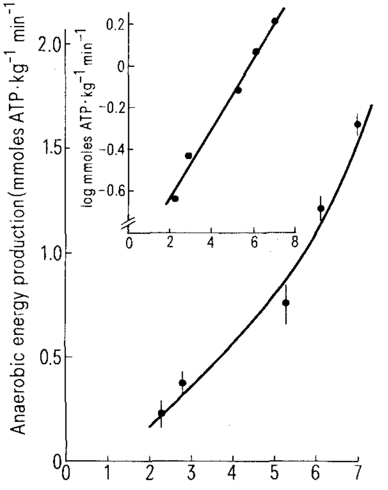

Figure 2. Average anaerobic energy production of rainbow trout during the 1st 5 min swimming at various speeds (see text for experimental details). Values of mmoles, ATP · kg^{-1}min^{-1} were calculated from the net accumulation of lactate in the whole carcass. Inset shows $\log_{10}$ ATP yield from anaerobic metabolism as a function of swimming speed.

1 To whom reprint requests should be addressed.
2 Johnston, I.A., Symp. Zool. Soc. Lond. *48* (1981) 71.
3 Bennet, A.F., and Licht, P., J. comp. Physiol. *81* (1972) 277.
4 Bennet, A.F., A. Rev. Physiol. *40* (1978) 447.
5 Wokoma, A., and Johnston, I.A., J. exp. Biol. *90* (1981) 361.
6 Johnston, I.A., and Moon, T.W., J. exp. Biol *87* (1980) 177.
7 Pasquis, P., Cacaisse, A., and Dejours, P., Respir. Physiol. *9* (1970) 298.
8 Bennet, A.F., Bioscience *30* (1980) 452.
9 Brett, J.R., Respir. Physiol. *14* (1972) 151.
10 Beamish, F.W.H., in: Fish Physiology, vol.7, p.101. Eds W.S. Hoar and D.J. Randall. Academic Press, New York 1978.
11 Roa, G.M.N., Can. J. Zool. *46* (1968) 781.
12 Prosser, C.L., in: Comparative Animal Physiology, p.362. Saunders, Philadelphia/Toronto 1973.
13 Black, E.C., Robertson, A.C., and Parker, R.R., in: Comparative Physiology of Carbohydrate metabolism in heterothermic animals, p.89. Ed. A.W. Martin. University of Washington, Seattle 1961.
14 Bilinski, E., and Jonas, R.E.E., J. Fish. Res. Bd Can. *29* (1972) 1467.
15 Hudson, R.C.L., J. exp. Biol. *55* (1973) 509.

0014-4754/83/121366-02\$1.50 + 0.20/0