blocking agents inadequate for inhibition of the renin-angiotensin system. However, angiotensin II administered the same way did increase drinking rates in both groups. A third possibility is that drinking behavior in these animals is under redundant control, and that other mechanisms drive "thirst" if the renin-angiotensin system is blocked. Furthermore, if peripheral levels of Captopril or Saralasin compromised the maintenance of blood pressure after hemorrhage, these other stimuli of drinking might well be enhanced. Indeed, drinking in flounder after hemorrhage plus Captopril was greater than after hemorrhage alone, although the difference was not significant ($p=.10$). Blood pressure was not measured in these experiments.

In conclusion, the marine stenohaline flounder and sculpin have control drinking rates one-tenth that of the salt-water adapted killifish. Exogenous angiotensin II stimulates drinking in the flounder and sculpin, but no evidence was obtained implicating endogenous angiotensin II in the physiological control of drinking behavior either in the basal state or following hemorrhage. This work was supported by NSF grant PCM 77-16465.

THE INFLUENCE OF Ca^{2+} AND CONTRACTION FREQUENCY ON TENSION DEVELOPMENT ON TELEOST VENTRICULAR STRIPS

H. Gesser and W.R. Driedzic, Department of Zoophysiology, University of Aarhus, Aarhus C., Denmark, Biology Department, Mount Allison University, Sackville, N.B.,Canada

In common with other vertebrate hearts, teleost ventricular strips develop increased tension as $[Ca^{2+}]_o$ is increased (Gesser and Jørgensen, J. Exp. Biol. 96:405-412, 1982). This may have direct relevance to the teleost heart since blood $[Ca^{2+}]_o$ is known to increase in association with intense activity (Ruben and Bennet, Nature 291: 411-413, 1981). In situations with enhanced circulatory requirements calcium stimulated ionotropy is likely to be favorable; however, an enlargement of the calcium pool also increases demand on the relaxation system. As such, a particularly prolonged relaxation time could compromise the maximal rate of contraction. Due to these considerations isolated heart strips were examined as to their ability to maintain force at different stimulation frequencies at low and high $[Ca^{2+}]_o$.

Experiments were conducted with 3 individuals each of mackerel (Scomber scombrus), ocean pout (Macrozoarces americanus), sea raven (Hemitripterus americanus), lumpfish (Cyclopterus lumpus), and longhorn sculpin (Myoxocephalus octodecimspinosus). The hearts were rapidly excised and placed in a solution gassed with 99% O_2: 1% CO_2 and containing NaCl 150 mM, KCl 5.0 mM, $MgSO_4$ 2.0 mM, NaH_2PO_4 0.5 mM, $NaHCO_3$ 11mM, and $CaCl_2$ 1 mM. Strips of approximately 1 mm diameter were cut from the ventricle, mounted for isometric recording of force at 15°C (Gesser, J. Exp. Biol. 69:199-206, 1977), electrically stimulated at 12 min^{-1} and stretched until peak force did not increase any further. After stabilization of force development the stimulation rate was increased by steps of 12 min^{-1} to the rate of 108^{-1} or to the rate at which arrythmic responses were observed. Each rate was maintained for about 30 sec. After this, the rate of 12 min^{-1} was reintroduced and the heart strips after stabilization were subjected to increased levels of Ca^{2+} from 1 to 7 or 9 mM. At the upper level of $[Ca^{2+}]_o$ the protocol with increasing stimulation rates was again carried out.

For all of the hearts examined increases in $[Ca^{2+}]_o$ resulted in an increased twitch force. The largest response was observed with mackerel in which a change in $[Ca^{2+}]_o$ from 1 to 9 mM resulted in a 270% increase in peak tension development. Sculpin, ocean pout and lumpfish were far less responsive in that similar alterations in $[Ca^{2+}]_o$ increased peak force development by only about 50%.

The maximum rate at which strips could contract regularly in response to imposed electrical stimulation varied amongst species. Mackerel, lumpfish, and sea raven could sustain the highest rate tested which was 108 contractions per min, whereas ocean pout and sculpin achieved only 96 and 60 contractions per min, respectively. Increasing the level of $[Ca^{2+}]_o$ from 1 to 7 or 9 mM did not influence this parameter.

There was a decrease in peak tension development as stimulation frequency increased for all species. That is: a plot of peak tension versus frequency of contraction has a negative slope. The magnitude of the slope was not affected by $[Ca^{2+}]_o$. Although increases in $[Ca^{2+}]_o$ shifted the curve upwards.

The data of the present study clearly show that increased $[Ca^{2+}]_o$ does not compromise frequency of contraction driven by electrical stimuli $[Ca^{2+}]_o$ over the physiological range from 1 to 3 mM resulted in an increased force development of approximately 50% in all of the species studied. In a variety of teleosts blood total $[Ca^{2+}]$ increased from about 2.5 to 3.5 mM in association with exercise (Ruben and Bennet, 1981). It is possible that under these conditions Ca^{2+} could contribute to increased cardiac output.

Perhaps the most interesting finding is the decreased tension development at elevated rates of contraction. This is in marked contrast to the general finding with other vertebrate hearts. During activity in teleosts increased cardiac output is met primarily through increases in stroke volume as opposed to elevations in heart rate (Jones and Randall, Fish Physiology Vol. VII, 1978). It is possible that this is a strategy to avoid the consequences of negative ionotropy. Research supported by operating grants from N.S.E.R.C. of Canada and N.B. Heart Foundation awarded to William R. Driedzic and a N.A.T.O. Travel Award to Hans Gesser and William R. Driedzic.

RELATIONSHIP BETWEEN EXOGENOUS FUEL UTILIZATION AND PERFORMANCE BY PERFUSED ISOLATED TELEOST AND ELASMOBRANCH HEARTS

William R. Driedzic and Tom Hart, Biology Department, Mount Allison University, Sackville, N.B., Canada

The teleost heart apparently has the capability of utilizing exogenous lactate, fatty acids or ketone bodies as metabolic fuels. This contention is based upon activities of isolated enzymes (Driedzic and Stewart, J. Comp. Physiol. in press) and oxidation rates of ^{14}C-labelled substrates (Driedzic et al, MDIBL Bull. 20: 30-32, 1980). In contrast to the teleost heart, the elasmobranch heart lacks both the enzymes necessary to rapidly degrade fatty acids (Zammit and Newsholme, Biochem. J. 184:313-322, 1979), and plasma fatty acid binding proteins analagous to albumin (Fellows and Hird, Comp. Biochem. Physiol. 68B:83-87, 1981). Elasmobranch hearts though have high titres of enzymes required to oxidize ketone bodies. In light of these findings it was considered of interest to assess what substrates (and at what levels) could sustain fish heart function.

The problem was approached by monitoring the performance of perfused isolated sea raven (Hemitripterus americanus) and skate (Raja) hearts following a transition from standard teleost or elasmobranch Ringers (without glucose) to one containing additional compounds (see Turner and Driedzic, Can. J. Zool. 58:886-889, 1980 for details of preparation). Iodoacetic acid, a known glycolytic inhibitor, was included in the perfusion media in some experiments. The data are presented as the percentage of the initial cardiac output following 30 min of perfusion (Table 1).

In the absence of any additions to the Ringers,cardiac output by both sea raven and skate hearts was reduced by about 15% after 30 min of perfusion. The inclusion of iodoacetic acid in the media resulted in a much more rapid decrease in cardiac output for both hearts. Physiological levels of exogenous lactate, acetoacetate, and palmitate were able to restore cardiac output of the sea raven heart to the control level. In these experiments sea raven hearts were performing close to in vivo resting levels of cardiac work. Metabolic fuel dependence at high levels of cardiac demand is yet to be investigated. Exogenous lactate, acetoacetate and 0.5 mM palmitate could support performance of the skate heart but 1.0 mM palmitate was actually deleterious. It would be of interest to ascertain if this effect is due to the inability to metabolize fat which in turn would result in increased intracellular fatty acid derivatives as occurs in the ischemic mammalian myocardium (Whitmer et al, J. Biol. Chem. 25:4305-4309, 1978) or to generalized detergent actions of fatty acids.