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.

Table 1. Percentage of initial cardiac output by isolated hearts after 30 min of perfusion.

Additions to perfusate	sea raven	skate
none	84 ± 14.6 (N=8)	86 ± 8.6 (N=9)
IAA*	41 ± 12.1 (N=6)	12 ± 9.3 (N=6)
IAA + LACTATE (2 mM)	88 ± 5.2 (N=4)	72 ± 11.1 (N=6)
IAA + ACETOACETATE (0.5 mM)	76 ± 8.1 (N=8)	94 ± 3.6 (N=7)
PALMITATE (0.5 mM)	90 ± 8.9 (N=5)	76 ± 8.7 (N=6)
PALMITATE (1.0 mM)	99 ± 4.8 (N=5)	59 ± 10.5 (N=6)
IAA + PALMITATE (1.0 mM)	95 ± 5.2 (N=6)	–

* IAA - iodoacetic acid (0.5 mM)

All data are expressed as mean ± S.E.M.

The findings of this study are in keeping with the previously recognized enzyme distribution and catabolic activities of teleost and elasmobranch hearts in that the former can utilize a variety of metabolic fuels whereas the latter are best supported by ketone bodies. Research supported by operating grants from N.B. Heart Foundation and N.S.E.R.C. of Canada to William R. Driedzic.

TAURINE TRANSPORT BY FLOUNDER (Pseudopleuronectes americanus) RENAL BRUSH BORDER MEMBRANE VESICLES

Patricia King, Philip Newsholme, Rolf Kinne and Leon Goldstein, Division of Biology and Medicine, Brown University, Providence, R.I., and Departments of Physiology and Biophysics, Albert Einstein College of Medicine, Bronx, N.Y.

INTRODUCTION--Taurine (2-aminoethane sulfonic acid) is one of the major amino acids participating in the cell volume regulation of a variety of marine fish as well as marine invertebrates and other vertebrate species. In the marine fish, taurine is a relatively inert compound metabolically and its plasma levels appear to be regulated by excretion. In vivo investigations of the renal handling of taurine in the little skate, flounder, and dogfish revealed that there is a net secretion of this amino acid by the kidneys of these fish (Schrock et al, Am. J. Physiol. 242:R64-R69, 1982). Recently, we have been studying the mechanism of taurine secretion by the renal epithelium of the flounder. Previous studies indicate that taurine movement across the basolateral membrane into the cell is an uphill concentrating step dependent on the presence of Na^+ and Cl^-, and inhibited by ouabain, as well as other β-amino acids and GABA (King et al, J. Exp. Zool., 223(2):103-114, 1982). Taurine movement from the cell to the tubular lumen then occurs down its chemical concentration gradient (0.1 mM in plasma, 30 mM in cell, 0.5 mM in urine). The purpose of the present study was to investigate taurine transport across the apical membrane by assaying taurine uptake into flounder renal brush border membrane vesicles. In particular, we were interested in determining whether a carrier system facilitating efflux of taurine from the cell was present at the apical surface.

METHODS--Brush border membrane vesicles were prepared from freshly dissected flounder kidneys by methods previously described (Eveloff et al, Am. J. Physiol. 237(4):F291-F298, 1979). The final preparation of vesicles was suspended in vesicle buffer (mannitol 100 mM; $CaCl_2$ 2 mM; Tris-HEPES, 20 mM; pH 8.) at a protein concentration of approximately 10 mg/ml and stored at -80°C until use (up to 5 days). An aliquot of the vesicles as well as the whole renal homogenate and discarded fractions were assayed for Na^+-K^+-ATPase and alkaline phosphatase activities. Alkaline phosphatase activity in the brush border membrane vesicles averaged 10.5 ± 2.2 μmoles/mg protein-hr (mean ± S.E., n=8) and was enriched 10-fold compared to activity in the whole homogenate. Brush border Na^+-K^+-ATPase activity was 3.0 ± 1.3 μmoles/mg protein-hr (mean ± S.E., n=8); the ratio of vesicle/homogenate activity for