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EFFECT OF TAURINE ON A MUSCLE INTRACELLULAR MEMBRANE

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SUMMARY

Sarcoplasmic reticulum from rat skeletal muscle had rates of calcium oxalate uptake dependent upon the speed with which the muscle was removed and homogenized. Rapidly isolated sarcoplasmic reticulum (muscle removed and homogenized within 20 min) showed a 25% increase in rate of calcium oxalate uptake in the presence of 15 mM taurine, and the total sequestering capacity was also increased. The rate of calcium oxalate uptake by more slowly isolated sarcoplasmic reticulum (1 h between sacrifice of first animal and homogenization of the muscle) could be increased by 30% by performing the isolation with 10–15 mM taurine present in all media, compared with a paired isolation in the absence of taurine. Exposure of sarcoplasmic reticulum to taurine throughout isolation led to an increased yield of microsomes and sarcoplasmic reticulum. These effects of taurine on sarcoplasmic reticulum were not duplicated by phosphate, isethionate, KCl, cysteine or histidine, but aminopropanesulfonic acid showed similar effects. Taurine slowed the rate of loss of calcium transport and ATPase activities of sarcoplasmic reticulum caused by phospholipase C. It is suggested that taurine may function as a membrane stabilizer.

INTRODUCTION

Taurine is present as the free amino acid in mammalian organs in large amounts[1]. It is concentrated particularly in heart and muscle, and about 50% of the free amino acid pool in mammals can be taurine[1]. Despite its high concentration and ubiquity, it has no defined function. Salt water molluscs contain large amounts, and it may serve as an osmoregulatory substance in these species. Taurine is structurally related to 3-aminobutyric acid, and is also present in quantity in nerve tissue[2,3]. It has a depressant effect on neurons[4] and is enriched in nerve endings[2] and some workers have, therefore, suggested it to be a neurotransmitter[2,4,5], although the large quantities present in the brain seem to militate against this hypothesis. Others have suggested that it has a role in ion movements in the heart[6–10] and it has been reported that it reverses digoxin- and epinephrine-induced arrhythmias in the heart[8–10]. In addition, it has an additive effect with ouabain, being positively inotropic in guinea pig auricles

Abbreviation: PCMB, *p*-chloromercuribenzoate.

and negatively inotropic in the rat in both normal and low calcium medium[6]. Taurine can also protect against β-oleandrine poisoning[11]. These reports indicate an effect of taurine on ion movement, and we have, therefore, examined the effect of taurine on a membrane specialized for calcium transport, the sarcoplasmic reticulum. Calcium and digitalis have additive inotropic effects, and an increased efficiency of calcium transport by sarcoplasmic reticulum in the presence of taurine would possibly explain the reported favorable effects of taurine on the heart.

Interactions between taurine and ion transport in skeletal muscle do not appear to have been previously studied.

MATERIALS AND METHODS

Scintillation counting was with either a Beckman LSC 250 counter or a Nuclear Chicago Isocap 300. Calculations were performed with a Wang 700 advanced programming calculator. Phospholipase C (*Clostridium perfringens*) was obtained from Mann Research Lab., New York. Sucrose used was the special enzyme grade from Schwartz-Mann, Orangeburg, N. Y. ATP was obtained from P-L Biochemicals, Milwaukee, Wisconsin and ^{45}Ca from Amersham-Searle, Arlington Heights, Illinois. [γ-^{32}P]ATP was obtained from New England Nuclear, Boston, Mass.

Isolation of sarcoplasmic reticulum fraction

Female Sprague–Dawley rats (150–200 g) were decapitated and desired (white) muscles removed and trimmed of fat and connective tissue. The muscle was homogenized in a Waring blender (30 S) in 3 vol. of 0.32 M sucrose. The homogenate was centrifuged at $14700 \times g$ for 20 min in a Sorvall RC-2B centrifuge in rotor SS-34, the supernatant filtered through 4 layers of gauze, and centrifuged at $57250 \times g$ for 90 min. The precipitate was resuspended in 0.32 M sucrose and placed onto a 20–50% continuous sucrose gradient, (total volume 60 ml, one tube suitable for material from up to 25 g muscle) followed by centrifugation at $80000 \times g$ for 2 h in a Beckman 25.2 swinging bucket rotor. The sarcoplasmic reticulum appeared as a diffuse band midway up the tube. Fractions of 3 ml were collected and each assayed for $(Ca^{2+} + Mg^{2+})$-ATPase and calcium transport. The isolation was carried out at 4 °C. Appropriate fractions were combined and stored at −56 °C.

Assays:

All incubations were done at 37 °C. Unless stated otherwise in the text, ATP was used as the Tris salt, prepared by passage of ATP through an AG 50 (H^+) ion-exchange column, followed by adjustment to the desired pH with Tris base. Protein was determined by the biuret[12] or Folin method[13] and phosphate by a method previously described[14]. Mg^{2+}-ATPase was measured in a solution containing 0.15 mg protein, 20 mM Tris, 3 mM $MgCl_2$, 0.2 mM EGTA and 4 mM ATP (pH 7.2). Total volume of incubation was 2 ml and length of incubation 10 min.

The calcium binding assay (calcium uptake in the absence of oxalate) was measured in a solution containing 16 mM histidine, 4 mM ATP, 5 mM $MgCl_2$, 100 mM KCl and 0.05 mM $^{45}CaCl_2$ at the appropriate pH. Reactions were terminated using the millipore filtration method of Martonosi and Feretos[15]. Phosphate and ^{45}Ca

were determined in the filtrate. Individual blanks were run for every reaction to correct for phosphate background and changes in ^{45}Ca counting efficiency. Calcium transport was measured in a solution containing 3.5 mM $MgCl_2$, 100 mM KCl, 20 mM NaCl, 4 mM ATP, 3 mM potassium oxalate and 0.1 mM $^{45}CaCl_2$ at the appropriate pH.

Binding and transport assays were run for 4 min, samples taken at 0.5, 1, 2 and 4 min and calcium bound and ATP hydrolyzed determined on these samples. Initial volume of incubation was 4 ml. For the binding assay, 0.2 ml (approx. 0.15 mg protein) of stock sarcoplasmic reticulum was used, and for the transport assay 0.05 ml (approx. 0.04 mg protein) was used.

Incubation of sarcoplasmic reticulum with phospholipase C (typical procedure)

1 ml of sarcoplasmic reticulum suspension (protein concentration 0.3 mg/ml) was diluted with either 0.5 ml water or 0.5 ml 45 mM taurine solution. 0.01 ml of a 15 mg/ml phospholipase solution was added, and the incubation stirred at room temperature. Samples were taken at 10-min intervals for calcium transport (0.05 ml), calcium binding (0.2 ml) or Mg^{2+}-ATPase (0.05 ml) assays. Assays were carried out in the presence and absence of 15 mM taurine. No washing procedure of the phospholipase-treated sarcoplasmic reticulum was carried out to avoid further damage to sarcoplasmic reticulum during manipulation.

Control experiments showed that after the phospholipase and sarcoplasmic reticulum sample had been diluted into the assay medium (4 ml) no further inactivation of the sarcoplasmic reticulum occurred. For maximum activity of phospholipase C calcium, or another cation, is required. As no exogenous calcium was present under the conditions used here, it was necessary to use a high ratio of phospholipase C to sarcoplasmic reticulum[16].

RESULTS

Effect of taurine in assay medium on sarcoplasmic reticulum functions

The stability and calcium binding and transporting activities of sarcoplasmic reticulum are correlated with the speed with which the muscle is removed from the animal and homogenized. With experience, this has been reduced to 20 min to sacrifice 3 animals, and remove, trim and homogenize the muscle.

With sarcoplasmic reticulum so isolated the presence of 15 mM taurine in the incubation medium increased the calcium transport activity as shown in Fig. 1. Taurine caused an increase in the rate of uptake of 25% in paired experiments from a single preparation, and this increase was highly significant ($P < 0.025$ at 0.5 min, $P < 0.005$ for other times). Fig. 1 also shows the effect of taurine on calcium binding at pH 6.5. The differences were not significant, although the P value increased monotonically with time. The presence of taurine did not affect the leaking of calcium from the membrane characteristically seen at pH levels below 7. ATPase activity was slightly but significantly increased. The ATPase rate in the presence and absence of taurine was almost linear over the 4-min total reaction period, is in marked contradistinction to the associated calcium uptake (Fig. 1A and Fig. 2). Mg^{2+}-ATPase rate was also linear. In this paper total ATPase activity has been used to calculate P/Ca ratio.

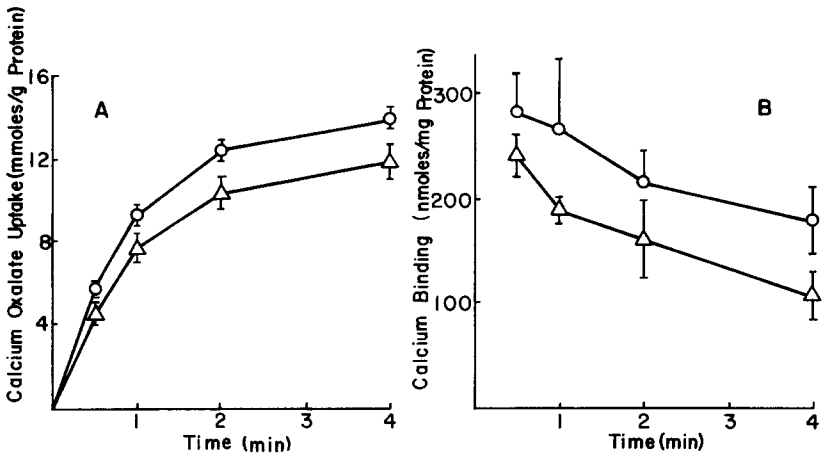

Fig. 1. A. Effect of 15 mM taurine in assay medium on calcium oxalate uptake at pH 7.2. ○—○, with taurine; △—△, without taurine. Values shown ± S.E. Uptake measured in medium containing 3.5 mM $MgCl_2$, 100 mM KCl, 20 mM NaCl, 16 mM histidine, 3 mM oxalate, 4 mM Tris–ATP, and 0.1 mM $^{45}CaCl_2$. Temp. 37 °C. No. of experiments 7. B. Effect of 15 mM taurine in assay medium on calcium binding at pH 6.5. ○—○, with taurine; △—△, without taurine. Binding measured in medium containing 5 mM $MgCl_2$, 16 mM histidine, 100 mM KCl, 4 mM Tris ATP and 0.05 mM $^{15}CaCl_2$. Temp. 37 °C. No. of experiments 7.

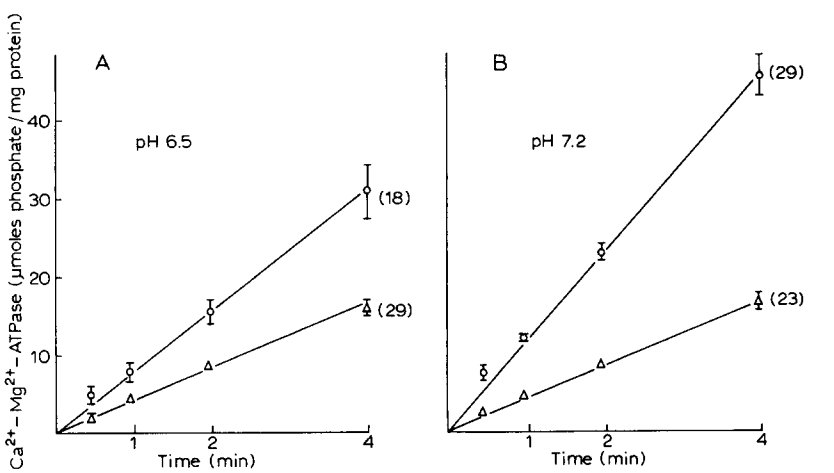

Fig. 2. (Ca^{2+}–Mg^{2+})-ATPase. ○—○, transport assay; △—△, binding assay. Points shown ± S.E. Where error bars are omitted, the S.E. lies within the size of the symbol. Number of preparations studied are shown in parentheses. Linear correlation coefficient for each line is 0.999. Mg^{2+}-ATPase activity at pH 7.2 is (6) 0.9 ± 0.04 μmole phosphate/min per mg protein.

Effect of taurine in isolation media on sarcoplasmic reticulum functions

Where the isolation had taken 2–3 × more time, we found no effect of taurine on sarcoplasmic reticulum assays. The rat muscle used contained endogenous taurine levels of 14 μmoles/g tissue[17]. Nearly all of the taurine is found in the supernatant on homogenization. A batch of muscle was divided equally and from one half sarcoplas-

mic reticulum was isolated normally. Sarcoplasmic reticulum was isolated from the other half by the same method, except a constant level of between 9–15 mM taurine was maintained in all the solutions used. (In any one experiment taurine concentration was invariant throughout.) Both sarcoplasmic reticulum samples were characterized in the usual manner in non-taurine containing media. As Table I shows, there was a significant increase of 30% in the rate of calcium oxalate uptake by the sarcoplasmic reticulum. However, there was no increase in the amount of ATP hydrolyzed. In the transport assay, the ratio of total phosphate released from ATP to calcium taken up, or P/Ca ratio, therefore, fell by around 30%. Data for one series of experiments are shown in Table I. The same effect of taurine in the isolation medium on transport function was observed at pH 6.5. This effect was not due to an increased resolution of sarcoplasmic reticulum because the total mass of microsomes was increased in the presence of taurine. For the sarcoplasmic reticulum fraction in general, in paired isolations from equal weights of muscle from the same animal, taurine led to an increase in the amount of protein of between 10–30%.

There was a significant decrease in initial binding at pH 6.5 (taurine isolation, 157 ± 14 nmoles Ca/mg protein; control isolation 205 ± 32; $P < 0.05$ (number of preparations 6, analyzed by paired t test)) at 0.5 min, but no difference at 1.0 min (taurine isolation 174 ± 44; control isolation 181 ± 31; $P > 0.5$). We have found that whereas at pH 7 and above, the binding of calcium to the membrane remains constant, below pH 7 a slow leaking process is observed. The continuous exposure of sarcoplasmic reticulum to taurine throughout the isolation procedure appears to alter the rates of binding and leaking. No alteration was found in ATPase rates for binding between taurine and control isolations.

TABLE I

EFFECT OF TAURINE IN ISOLATION MEDIUM ON CALCIUM OXALATE UPTAKE AND P/Ca RATIO

Number of paired experiments 8. Assay conditions as given for Fig. 1A. The 4-min P/Ca ratios for fractions for combination off the sucrose gradient were taurine 3.6, normal 4.7 ($P < 0.005$).

Time (*min*)	*Uptake (mmole/g protein)*			*P/Ca Ratio*		
	Taurine	*Control*	*Increase (%)*	*Taurine*	*Control*	*Decrease (%)*
0.5	3.90 ± 0.65	2.98 ± 0.76	31 ($P < 0.005$)	2.5	3.4	27 ($P < 0.100$)
1	7.02 ± 0.85	5.50 ± 0.95	28 ($P < 0.001$)	1.9	2.8	32 ($P < 0.05$)
2	9.15 ± 1.20	7.26 ± 1.16	26 ($P < 0.001$)	2.4	3.5	31 ($P < 0.001$)
4	10.04 ± 1.33	8.26 ± 1.14	21 ($P < 0.005$)	4.2	5.3	21 ($P < 0.001$)

Leakage for calcium oxalate loaded vesicles

The P/Ca ratios discussed above are not the "true" ATP: calcium transport coupling ratios in that there is passive leakage of calcium from the sarcoplasmic reticulum vesicles at a rate dependent on the degree of filling. By passive leakage is meant outflow of calcium other than the reverse flow through the calcium pump which has been described[18–20]. Only the net calcium transport has been considered in calculating the P/Ca ratios. The rate of uptake of calcium is linear with time, as is ATP

hydrolysis, and the uptake curve (*e.g.* Fig. 1A) is the excess of uptake over leakage. Leakage was shown by the following:

Excess EGTA was added to the assay at various times during calcium transport[21–23]. The reappearance of radioactivity in solution measured the rate of leakage of calcium from the partially loaded sarcoplasmic reticulum. After 3 min of uptake at 37 °C under conditions such that only 50% of the calcium present in the original incubation had been sequestered the rate of leakage approached that of the initial rate of uptake (the vesicles contain 8 mmoles calcium/g protein at this stage). The same result was obtained if the sulfhydryl blocking agent *p*-chloromercuribenzoate (PCMB)[24] was used to block active transport. Salyrgan has been reported to have a similar effect[25,26].

At 15 °C, calcium transport is linear for 8 min. During this period, no leakage is detectable by the above techniques.

Vesicles partially loaded with ^{40}Ca oxalate were exposed to trace amounts of ^{45}Ca. This demonstrated that the bulk of intravesicular calcium oxalate was not available for exchange, in agreement with previous work[15,27].

Effect of other substances in isolation media on sarcoplasmic reticulum functions

Some other substances were tested in a similar way for their effect on sarcoplasmic reticulum in paired isolations to see whether the effects observed with taurine were specific to that compound or were more general phenomena. Results for oxalate and binding assays are shown in Table II. Sodium isethionate (2-hydroxyethane sul-

TABLE II

PERCENTAGE EFFECT OF OTHER SUBSTANCES ON CALCIUM UPTAKE AND ATPase RATE IN MATCHED ISOLATIONS

Values given as percentage of value for normal isolations. Number of paired isolations given in parentheses. As calcium leaking phenomenon is not seen at pH 7.2, the amount of calcium bound was averaged for all time intervals.

	Time (min)	*Sodium phosphate (9 mM)*	*Sodium isothionate (3 mM)*	*KCl (4.5 mM)*	*Cysteine (9 mM)*	*Histidine (9 mM)*	*Aminopropanesulfonic acid (9 mM)*
Transport assay							
pH 7.2	0.5	+17(2)	−36(2)	−36(1)	−74(1)	−50(2)	+86(1)
	1.0	+12	−38	−21	−84	−10	+113
	2.0	+15	−34	−20	−84	+13	+32
	4.0	+17	−29	−21	−63	+31	+33
$(Ca^{2+}+Mg^{2+})$-ATPase		+1	−12	+5	−14	+5	+40
Binding assay							
pH 6.5	0.5	−24	−11	+45	−88	−42	+51
	1.0	+3	+23	+103	−74	−36	−43
	2.0	+18	+32	+56	−78	−38	−14
	4.0	+176	−28	+24	−55	+2	−32
pH 7.2			−36		−78	+23	

fonate), potassium chloride, and cysteine all depressed the rate and extent of uptake in the oxalate assay. Sodium phosphate and aminopropanesulfonic acid improved the uptake. The latter substance is a homolog of taurine (aminoethanesulfonic acid). Histidine slowed the rate of uptake but increased the capacity. The increase in uptake observed with sodium phosphate may be due to the fact that phosphate, like oxalate, is a precipitating anion for calcium (solubility product approx. $4 \cdot 10^{-9}$ M^2). The binding assay showed for phosphate a continuous increase in calcium binding whereas in normal isolations the calcium leaks away from the membrane with time at pH 6.5. Aminopropanesulfonic acid increased calcium oxalate uptake, and lowered the P/Ca ratio, similarly to taurine. Also, like taurine, it showed decreased membrane binding at pH 6.5 (taurine causes a drop of 11%) with a delay in the time to maximum binding. At pH 6.5, aminopropanesulfonic acid also caused a similar increase in calcium oxalate transport (81% increase at 1.0 min). The difference in $(Ca^{2+} + Mg^{2+})$-ATPase activities associated with these experiments are shown in Table II, and it can be seen that there were substantial changes in the P/Ca ratios for isethionate, cysteine and KCl isolations. These other substances were studied to ascertain whether the effects observed with taurine were specific to that substance or were of general applicability to a range of compounds. For this reason, only one or two isolations were done with each substance. Detailed conclusions should not be drawn from the data in Table II, but in general they do bring out the specificity of taurine.

Treatment of sarcoplasmic reticulum with phospholipase C

As it appeared that taurine was protecting sarcoplasmic reticulum from damage during isolation, sarcoplasmic reticulum was deliberately damaged in the presence and absence of taurine to see if a protective effect could be demonstrated directly. Treatment with phospholipase C was used to cause damage. This enzyme cleaves the phosphorylated base (phosphoethanolamine, phosphocholine or phosphoserine) from a phosphoglyceride[28]. It has been reported that the phosphoryl group is needed for maintenance of the membrane structure which is necessary for calcium transport rather than serving directly in the transport process[29]. About 70% of the phospholipid is phosphatidylcholine[30]. Treatment with phospholipase C has been shown to inhibit calcium binding and ATPase activity of sarcoplasmic reticulum, but activity could be restored by treatment with a wide variety of lipophilic substances. Almost 60% of the phosphatidylcholine was cleaved before inhibition of calcium uptake was observed[31].

On being incubated with phospholipase C, sarcoplasmic reticulum were rapidly inactivated as regards ability to bind or transport calcium. (Mg^{2+})- and $(Ca^{2+} + Mg^{2+})$-ATPase activities were inactivated rather more slowly. Even after calcium uptake and binding had been totally abolished there was still appreciable ATPase activity. Fig. 3 shows a typical experiment on the effects of incubating sarcoplasmic reticulum with phospholipase C in the presence or absence of taurine. Fig. 3A demonstrates the effect on calcium transport. In this case incubation with phospholipase C was allowed to proceed long enough to abolish 82% of the original calcium uptake by the non-taurine treated sarcoplasmic reticulum. However, the taurine-treated sarcoplasmic reticulum still retained approx. 35% of its transport capability. Fig. 3B illustrates a similar experiment (with a different sarcoplasmic reticulum preparation) on calcium binding. Again, the presence of taurine led to a partial retention of binding ability. If incubation with phospholipase C was performed for

shorter periods such that sarcoplasmic reticulum activity was not completely abolished, the incubation without taurine was found to be damaged more rapidly. If incubation was allowed to proceed long enough, calcium binding and transport abilities were totally abolished in both cases. The presence of taurine also had a protective effect on Mg^{2+}-ATPase measured in the presence and absence of exogenous calcium, though this was not so marked. The protection was against long exposure to phospholipase, rather than brief exposure. Brief exposure led to taurine-treated and control sarcoplasmic reticulum losing ATPase activity at the same rate. Attempts were made to assay solubilized ATPase by filtering phopholipase C treated sarcoplasmic reticulum through 0.45 μm millipore filters and measuring ATPase activity in the filtrate. Even after 40% of the total ATPase activity had disappeared, however, only 2% of the original activity could be measured in the filtrate.

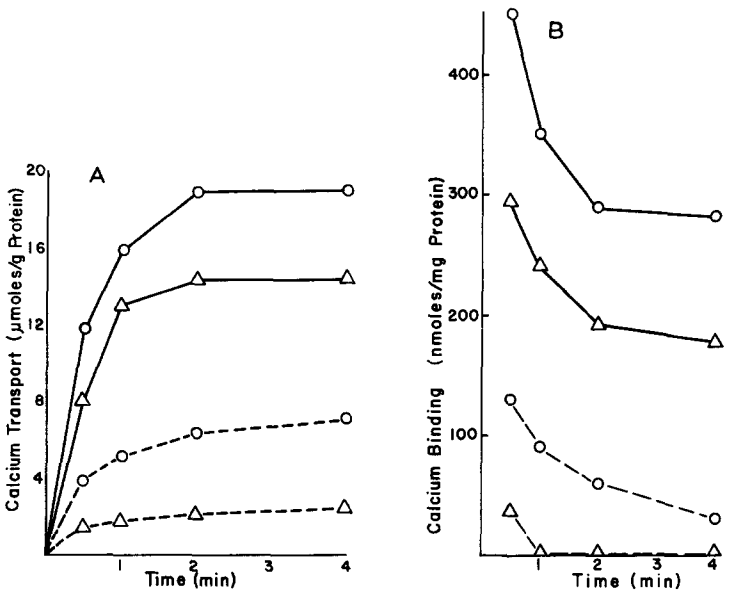

Fig. 3. A. Effect of phospholipase C treatment in presence and absence of taurine 15 mM on calcium oxalate uptake. Protein (0.25 mg/ml) incubated with phospholipase C (0.10 mg/ml) at room temperature for 10 min. Sample was then taken for calcium oxalate uptake assay at pH 7.2. B. Effect of phospholipase C treatment in presence and absence of 15 mM taurine on calcium binding at pH 6.5. Conditions as for A. ○—○, presence of taurine; △—△, absence of taurine. Full line is uptake before phospholipase C treatment and dashed line is uptake after treatment. These curves are typical of 4 experiments in which calcium uptake and binding were partially damaged.

p-Chloromercuribenzoate on sarcoplasmic reticulum

It has been reported that *p*-chloromercuribenzoate inhibited the ability of sarcoplasmic reticulum to bind and transport calcium[24]. We found that PCMB at a concentration of 0.1 mM totally abolished the binding and transport of calcium by sarcoplasmic reticulum as well as $(Ca^{2+} + Mg^{2+})$-ATPase in the presence and absence of taurine. Mg^{2+}-ATPase activity measured in the absence of exogenous calcium (*i.e.* no EGTA present), was reduced to 15–20% of initial activity on treatment with

PCMB, but taurine had a protective effect. In 4 experiments on sarcoplasmic reticulum preparations Mg^{2+}-ATPase activity was initially 2.47 μmoles/min per mg, and after PCMB treatment this was reduced to 0.41 μmole/min per mg in the absence of taurine and 1.66 μmoles/min per mg in the presence of taurine. It has been pointed out that the related compound Salyrgan affects sarcoplasmic reticulum in a way dependent on the order of addition of the reagents[32]. Our reactions were run by preincubating sarcoplasmic reticulum with PCMB for 10 min at 37 °C and starting reaction by the addition of ATP and calcium.

DISCUSSION

If increase in P/Ca ratio (increase in leakage rate), based on total hydrolysis of ATP, is taken as an index of damage then sarcoplasmic reticulum are readily damaged. We have found the P/Ca ratio to be extremely sensitive to speed of isolation and it also increases somewhat during the interval between collecting fractions off the sucrose density gradient and measuring the functions of the combined fractions comprising the sarcoplasmic reticulum. An interpretation of the results obtained is that taurine has a protective effect on the sarcoplasmic reticulum membrane rather than an activating effect. Its presence during the isolation procedure helps preserve the integrity of the sarcoplasmic reticulum (as indicated by the higher protein yield obtained from taurine isolations) and protects it from damage. With slowly isolated, more damaged preparations, adding taurine to assay media could not reverse the damage already sustained. With rapidly isolated, more active preparations, the extent of membrane damage may be slight enough to allow taurine to reverse the damage to a certain extent during assay. Sarcoplasmic reticulum isolated under the described conditions contain endogenous calcium. Working with rabbit sarcoplasmic reticulum isolated by means of a continuous sucrose gradient others have found an endogenous calcium content of 13.8 ± 0.3 nmoles/mg protein[33]. The results obtained cannot be ascribable to differences in endogenous concentration as only 0.02–0.05 mg protein were used for transport studies in an assay medium containing 400 nmoles of exogenous calcium. Long enough treatment with phospholipase C abolished sarcoplasmic reticulum functions whether taurine was present or not, but taurine slowed the rate of inactivation. It had a protective effect on ATPase activities, and a stronger effect on preservation of calcium binding and transport. When phosphorylcholine is cleaved from a phosphoglyceride, a zwitterionic structure is converted to a non-ionic one. Phosphoglycerides are unlikely to have a highly specific function in calcium transport as about 60% have to be hydrolyzed before an appreciable fall in calcium transport occurs[31]. Also, by altering diet, it is possible to change the fatty acid composition of sarcoplasmic reticulum phospholipid without any apparent deleterious effects[29]. Their role in the membrane, therefore, is possibly a non-specific structural one in maintaining the conformation of the membrane. The presence of taurine in solution may help to maintain the conformation of the partially hydrolyzed membrane. Taurine is a stable, only slowly metabolized zwitterion present in cells in large amounts. Movement into and out of the cell is slow[1]. We suggest the possibility that one function of taurine is that of a membrane stabilizer.

Guidotti and Giotti point out that some of the effects of taurine on the heart seemed to be related to cellular calcium transport[34]. The results presented above

indicate that taurine, at a concentration endogenously present in rat muscle, has a stabilizing effect on the subcellular membrane responsible for the control of calcium transport during excitation–contraction coupling. Certain pathological states in both heart and muscle have accompanying defects in the transport and enzymatic functions of the sarcoplasmic reticulum. Genetically cardiomyopathic Syrian hamsters show marked decreases in calcium binding by the cardiac relaxing system or sarcoplasmic reticulum[35]. The same observation has been made in human heart failure[35]. Skeletal muscle samples from muscular dystrophy patients also show defects in the ability of sarcoplasmic reticulum to transport calcium[36,37]. Sarcoplasmic reticulum from dystrophic mice exhibits increased ATPase activity and decreased calcium binding and transport compared to non-dystrophic mice[38]. In the heart, at least, consumption of energy by the sarcoplasmic reticulum has been estimated to account for 15% of the total energy consumption of the organ[39]. A failing heart is less efficient, and exhibits a dislocation in energy utilization rather than production. Agents which can maintain tight coupling in the sarcoplasmic reticulum (low P/Ca ratios) thus have potential pharmacological importance in both heart and skeletal muscle.

Thus, there exists a body of evidence suggesting a link between taurine and ion transport. The data presented in this paper show that under the defined conditions the presence of intracellular levels of taurine results in a preparation of sarcoplasmic reticulum of greater calcium transporting activity and stability. The physiological and pathological consequences of altered intracellular taurine levels on myocardial and skeletal muscle function is not yet known, but the potential role of the aminosulfonic acid in membrane function is clearly worthy of investigation.

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