The nature of the protein components and their location in the sarcoplasmic reticulum membrane were studied using sarcoplasmic reticulum vesicles isolated from rat skeletal muscle and purified by a density gradient centrifugation system. On the basis of analysis by means of sodium dodecyl sulfate gel electrophoresis, the protein components appear to be similar if not identical with those reported by others for rabbit sarcoplasmic reticulum, and the relative amount of each component is also similar to that found with rabbit sarcoplasmic reticulum. Evidence is presented that radioiodine-labeled diazotized diiodosulfanilic acid is a nonpermeant labeling agent of the protein components of sarcoplasmic reticulum vesicles; this agent minimally disturbs the functional activities of these membranes. By means of this labeling agent and perturbing agents, it is concluded that the protein components with molecular weights greater than 120,000 and the (Ca$^{2+}$ + Mg$^{2+}$)-adenosine triphosphatase partially or totally reside on or at the external surface of the sarcoplasmic reticulum vesicles. In the case of the adenosine triphosphatase, highly controlled trypsin treatment cleaves the molecule into two products, a 65,000 molecular weight fragment and a 56,000 molecular weight fragment. The evidence indicates that the 65,000 molecular weight component of the (Ca$^{2+}$ + Mg$^{2+}$)-adenosine triphosphatase is located in a more exposed position on the external surface of the vesicle than others. The protein components designated by MacLennan (MacLennan, D. H. (1975) Can. J. Biochem. 53, 251-261) as "calsequestrin" and "high affinity Ca$^{2+}$ binding protein" are shown not to be on the external surface of the rat sarcoplasmic reticulum vesicle but rather to reside either within the core of the membrane or on the inside surface of the vesicle. The results of this study are in agreement with the model for the organization of the protein components of the sarcoplasmic reticulum membrane recently proposed by MacLennan (MacLennan, D. H. (1975) Can. J. Biochem. 53, 251-261).
was presented which indicated that $^{[25]}$DDISA is a useful nonpermeant probe for the localization of the protein components of isolated SR vesicles. The present report provides further information about this labeling probe, as well as data obtained by its use along with membrane perturbers in experiments aimed at further defining the functional localization of the protein components of SR membranes.

**EXPERIMENTAL PROCEDURE**

**Materials**  Phosphorylase a, peopin, and immobilized trypsin were purchased from Worthington Biochemical Corp. Lactoperoxidase and trypsin (A grade) were purchased from Calbiochem. Ovalubin was purchased from Fisher Scientific Co. Bovine serum albumin, $\alpha$-galactosidase, $\alpha$-chymotrypsinogen A, and myoglobin, and soybean trypsin inhibitor were purchased from Sigma Chemical Corp. Phospholipase C (Bacillus cereus) was purchased from Boehringer Mannheim Corp. Ultra pure sucrose (special enzyme grade) was obtained from Schwarz/Mann. The $^{[24]}$DDISA preparation used in our experiments was the generous gift of Dr. David A. Sears, Department of Medicine, University of Texas Health Science Center at San Antonio. All other reagents used were of analytical grade.

Fragmented SR membrane vesicles were prepared from rat skeletal muscle and purified by the sequential use of two sucrose density gradient systems by the method of Yu et al. (10).

**Analysis of SR Proteins by SDS-Polyacrylamide Gel Electrophoresis**—To dissociate the protein prior to electrophoretic analysis, the SR vesicles were dissolved in a solution of 0.1 M sodium phosphate (pH 7.4), 1% SDS, 1% mercaptoethanol, and 0.2% NaN$_3$. This solution containing approximately 1 mg of protein/ml was then heated for 3 hours at 37° to dissociate the protein.

Polyacrylamide gels (9.0 x 0.5 cm), usually of 5 or 7.5% gel concentration with 5% cross-linkage, were formed in a solution of 0.1 M sodium phosphate buffer (pH 7.1), 0.1% SDS, 0.2% NaN$_3$, 0.1% N,N,N',N'-tetramethylthelylenediamine, and 0.1% ammonium persulfate. The buffer system in the reservoir was 0.1 M sodium phosphate buffer (pH 7.10), 0.1% SDS, and 0.2% NaN$_3$. The dissociated protein (20 to 25 μg) was applied to each gel and run for 7 hours at 5 mA/tube. Bromphenol blue was used as a tracking dye. Gels were fixed and stained by the method of Fairbanks et al. (11), and electrolytically destained with a 5% acetic acid solution.

The molecular weight of each SR protein component was estimated by the method of Shapiro et al. (12) using the following of the many proteins studied by Weber and Osborne (13) as molecular weight standards: $\beta$-galactosidase (130,000), phosphorylase a (94,600), lactoperoxidase (84,000), bovine serum albumin (68,000), ovalubin (43,000), peopin (35,000), $\alpha$-chymotrypsinogen (26,000), and myoglobin (17,200). Relative mobility of each protein was calculated with respect to the migration distance of bromophenol blue.

**Labeling of SR Vesicles by Treatment with $^{[25]}$DDISA**—To label the SR protein prior to electrophoretic analysis, the SR vesicles were dissolved in a solution containing approximately 1 mg of protein/ml, 0.02 M Tris-maleate, 4 mM CaCl$_2$, 0.3 M sucrose plus an amount of immobilized trypsin (bound to Sepharose) equivalent to 0.06 mg of the enzyme/ml of medium; the pH of the system was 7.0. The reaction was terminated by the addition of a mass of soybean trypsin inhibitor twice that of the trypsin.

**Treatment of SR Vesicles with Phospholipase C**—SR vesicles were added to a 0.15 M NaCl and 0.1 M CaCl$_2$ solution to yield a suspension of 0.6 mg of SR protein/ml. The pH was adjusted to 7.0 with 0.1 M NaOH. Then phospholipase C (Bacillus cereus) was added to give a final concentration of 0.4 i.u. of enzyme/ml, and as the reaction proceeded the pH was maintained at 7.0 by automatic addition of 0.1 M NaOH delivered by a Radiometer Titrograph Apparatus. The enzymatic activity was terminated at the desired time by quick freezing in powdered dry ice.

**Analysis of Ca$^{2+}$ Transport, (Ca$^{2+}$ + Mg$^{2+}$)-ATPase Activity, and Protein and Phospholipid Content of SR Vesicles**—The Ca$^{2+}$ transport activity and the oxalate-promoted, ATP-dependent Ca$^{2+}$ transport activity of SR vesicles were determined by the methods of Yu et al. (10).

The phospholipid content of the SR vesicles was determined by the method of Sansone et al. (15).

The protein content of SR vesicles was measured by the modification of the method of Lowry et al. (16) described previously (10). The protein components, separated by gel electrophoresis, were estimated by densitometric scanning of the Coomassie blue-stained bands. When the amount of SR protein loaded on a gel was varied between 10 and 20 μg, the relative quantities of each of the protein components remained constant.

**RESULTS**

**Protein Components of SR Vesicles**—When dissociated SR vesicle proteins were separated by SDS-polyacrylamide gel electrophoresis on the traditionally used 7.5% gels, six well separated bands were obtained designated as Bands 1 through 6 on the basis of migration distance (Fig. 1a). Band 1 migrating the least and Band 6 the most. On the basis of the densitometric analysis of 15 SR preparations in which the amount applied was 20 to 25 μg, rather than the 80 μg used to obtain Fig. 1a, the percentage of the total SR protein found in each band is the following: Band 1, 15%; Band 2, 65%; Band 3, 8%; Band 4, 7%; Band 5, 3%; Band 6, 2%. This pattern of bands is similar to that reported by other investigators (1, 3, 17, 18) for rabbit SR.

However, when a 5% gel is used, as shown in Fig. 1b, it is clearly revealed that Bands 1, 2, 5, and 6 contain more than a single component. In the case of Band 2, which contains 65% of the protein, in addition to the major component, a minor component is usually evident by densitometric scanning and is occasionally observed by visual inspection, as is the case with the electrophoretogram shown in Fig. 1b. This minor component, which migrates slightly ahead of the major component is designated 2a.

The molecular weights of the proteins comprising these bands have been estimated by others (1, 3, 17, 18) with some variation in the values obtained. The estimates made in our laboratory (Table I) are in general agreement with those reported in the literature. However, it should be noted that most values reported in the literature were obtained using a 7.5% gel concentration for the measurement. Analysis of the data obtained in our laboratory from many individual measurements indicates that the relationship between the log molecular weight and the mobility of the protein relative to the tracking dye deviates from linearity in the molecular weight range above 80,000 with the 7.5% gel (Fig. 2); no marked deviation from linearity is apparent with a 5% gel. Hence, the molecular weight estimations for SR protein are best achieved using 5% gels, which are reported in Table I.
Analysis of SR vesicle protein components by SDS-polyacrylamide gel electrophoresis. a, 80 μg of SR protein was applied to a 7.5% gel; b, 20 μg of SR protein was applied to a 5% gel.

### Table I

Molecular weight estimation of SR membrane protein components with SDS-polyacrylamide gel electrophoresis

| Bands | Molecular weight |
|-------|-----------------|
| 1     | ~280,000        |
|       | ~220,000        |
|       | ~190,000        |
| 2     | ~119,000        |
| 2a    | ~110,000        |
| 3     | ~63,000         |
| 4     | ~50,000         |
| 5     | ~32,000         |
| 6     | ~20,000         |

*Average values from four experiments.

That the major component of Band 2 is most probably the (Ca^{2+} + Mg^{2+})-ATPase is indicated by the following data. Its molecular weight is similar to the values reported by others (1, 3, 17, 18); it comprises more than 50% of the protein content of the SR vesicle; and using the method of Meissner and Fleischer (17) it was found that the addition of [γ-32P]ATP to the SR vesicles causes this protein to be phosphorylated with 32P. On the basis of the molecular weight and extractability from the membrane with a deoxycholate, high ionic strength solution, Bands 3 and 4 probably represent "high affinity Ca^{2+} binding protein" and "calsequestrin" described by MacLennan (1). Proteins with the molecular weights of Bands 5 and 6 have been obtained from SR membranes by others (1, 3), but their functional role has not been defined. There is no evidence concerning the functions of the proteins comprising Band 1, nor indeed is it yet clear whether these proteins are components of the native SR.

### Use of [125I]DDISA to Label SR Vesicle Proteins—It is well established that [125I]DDISA is a useful nonpermeant labeling agent for the plasma membrane proteins of intact human erythrocytes (14). In a recent preliminary report from our laboratory (9), evidence was presented indicating that [125I]-DDISA also serves as a nonpermeant labeling agent for fragmented SR vesicles. The characteristics of the reaction of SR vesicles with [125I]DDISA have been further explored. The reaction of [125I]DDISA with the SR membrane is rapid with near maximal labeling occurring after 1 min incubation at 25°C. Moreover, the 125I cannot be significantly removed from the membrane by extensive washing. Of the [125I]DDISA taken up by SR vesicles, less than 4% of the label was associated with the membrane phospholipid components as isolated by the method of Sanslone et al. (15).

In Table II, data are reported on the effect that labeling with [125I]DDISA has on the oxalate-promoted, ATP-dependent Ca^{2+} transport and the (Ca^{2+} + Mg^{2+})-ATPase activities. Although the Ca^{2+} transport function was not affected, this labeling agent did cause some enhancement of the (Ca^{2+} + Mg^{2+})-ATPase activity.

### Localization of SR Membrane Proteins—Both this [125I]-
DDISA labeling procedure and the perturbation of the SR membranes by enzymatic and physical means were used to explore the location of the protein components of the SR membranes.

In Fig. 3 are plotted the densitometric tracing of the protein components after separation by 7.5% SDS-gel electrophoresis and the amount of radioactivity from $^{125}$I-DDISA in the protein components. Most of the $^{125}$I is associated with the proteins in Bands 1 and 2, which have the greatest amount of $^{125}$I/unit mass of protein. It should be noted that the protein components in Bands 3, 4, and 6 contain almost no radioactivity. Although the $^{125}$I specific activity of the protein in Band 5 is not as negligible as that of the proteins in Bands 3, 4, and 6, it is less than that observed for the protein components in Bands 1 and 2.

Phospholipase C causes a loss of SR vesicle phospholipid and $(Ca^{2+} + Mg^{2+})$-ATPase activity (19). In the present study, the effect of treating the SR vesicles with phospholipase C on the phospholipid:protein mass ratio, the $(Ca^{2+} + Mg^{2+})$-ATPase activity, and the labeling with $^{125}$I-DDISA is shown in Fig. 4. In addition to its known effects on SR phospholipid content and ATPase activity, the phospholipase C treatment markedly increases the labeling of the membrane protein with $^{125}$I. The changes in the $^{125}$I specific activity after labeling the vesicle with $^{125}$I-DDISA was measured for each SR protein component (Fig. 5a). All components show an increase in specific activity after 60 min of phospholipase C treatment, but the increase is most marked in the case of the protein components in Bands 3 and 4.

The effect of sonicating the SR vesicles on the labeling of these membranes by $^{125}$I-DDISA was also studied. This treatment also increases the $^{125}$I-labeling of the vesicles. The change in specific activity of the various protein components is shown in Fig. 5b; the protein components in Bands 3 and 4 show a marked increase in specific activity.

Exposing intact SR vesicles to a proteolytic agent would be expected to hydrolyze proteins exposed to the external environment. To obviate the possibility that the proteolytic agent can permeate the membrane, hydrolyzing the peptides within the membrane structure or within the vesicle, the proteolytic agent used could be attached to a clearly impermeant material. Hence, SR vesicles were treated with soluble and Sepharose-immobilized trypsin. Both cause similar changes in the pattern obtained with SDS-gel electrophoresis of the dissociated proteins (Fig. 6).

After short periods of treatment with immobilized trypsin, only the proteins in Bands 1 and 2 disappear; moreover, two new protein components appear, specifically Band 3α (molecular weight of 65,000) and Band 3β (molecular weight of 56,000). With the 5% gel shown in Fig. 6a, Band 3α is not clearly resolved relative to the native protein component in Band 3; However, by using an 8.5% gel (Fig. 7), allowing the electrophoresis to occur over a prolonged period of time, and using a brief digestion period, component 3α can be clearly separated from component 3. With more prolonged digestion with the immobilized enzyme, two more new protein components appear, specifically Band 5α (molecular weight of 38,000) and Band 5β (molecular weight of 28,000).

Also, soluble trypsin, which certainly cannot have been altered by being bound to Sepharose, destroys only the proteins comprising Bands 1 and 2 when used for short periods of time.

### Table II

| Length of time exposed to $^{125}$I-DDISA labeling procedure | Calcium uptake (μmol Ca$^{2+}$/mg SR protein) | $(Ca^{2+} + Mg^{2+})$-ATPase (μg P/min/mg SR protein) |
|---------------------------------------------------------------|---------------------------------------------|-------------------------------------------------------|
| 0 min                                                        | 1.99                                        | 104                                                  |
| 5 min                                                        | 1.98                                        | 131                                                  |
| 20 min                                                       | 1.00                                        | 155                                                  |

Fig. 4. Effect of treatment of SR vesicles with phospholipase C on phospholipid:protein weight ratio, $(Ca^{2+} + Mg^{2+})$-ATPase activity, and $^{125}$I content of the protein components obtained by labeling with $^{125}$I-DDISA. The data for the phospholipid:protein weight ratio (△) and for the ATPase activity (■) are average values from three experiments, and the data for the $^{125}$I labeling (○) are average values from two experiments.
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and rabbit SR. The other two components found in rabbit SR in appreciable abundance (1, 18, 20, 21) have been named the “high affinity Ca$^{2+}$ binding protein” and “calsequestrin” by MacLennan (1), and are also present in similar abundance in rat SR. MacLennan (1) reports in rabbit SR vesicles there are two minor protein components with estimated molecular weights of approximately 30,000 and 20,000, and in the present study, similar components were also found in the rat SR vesicles. Moreover, rat SR vesicles were found to contain appreciable amounts of proteins with molecular weights greater than 120,000 (Band 1, Fig. 1). Careful observation of the electrophoretograms reported for rabbit SR vesicle proteins reveals high molecular weight protein components to be present in these membrane preparations too (1, 3, 18, 22).

In an earlier report from our laboratory (23), it was reported that most of the membrane proteins from rat SR migrates on SDS-gel electrophoresis as a low molecular weight entity (molecular weight of ≈10,000) after dissociation by the method of Shapiro et al. (12) which involves an incubation followed by a long period of dialysis. In the present study, this dissociation procedure was modified by adding 0.2% NaN$_3$ and by omitting the dialysis procedure. With the inclusion of NaN$_3$ and the omission of dialysis, the low molecular protein is no longer observed suggesting that it resulted from peptidolysis. In this regard, MacLennan and Holland (24) point out that SDS methodologies are prone to peptidolysis because of the presence of either the endogenous peptidases of the isolated membranes or proteases from contaminating microorganisms. However, as summarized in a recent report (25) much data have also been obtained in our laboratory which are not consistent with this low molecular weight protein entity being a proteolytic artifact. Therefore, it is not yet possible to make a definitive statement regarding the status of the low molecular weight entity.

DDISA is selective in its labeling of the SR membrane protein components. The high molecular weight proteins in Band 1, Fig. 1a, are highly labeled, as is the (Ca$^{2+}$ + Mg$^{2+}$)-ATPase, whereas the proteins in Bands 3 and 4 (the high affinity Ca$^{2+}$ binding protein and calsequestrin) are almost unlabeled. It seems likely that this selectivity in labeling results from the nonpermeant character of [${}^{125}$I]DDISA relative to biological membranes (9, 14, 26). That this is the case with rat skeletal muscle SR vesicles is strongly supported by the findings of the present investigation, the most salient of which is the fact that calsequestrin and high affinity Ca$^{2+}$ binding protein are almost unlabeled in untreated SR vesicles, but are highly labeled when the vesicles are either treated with phospholipase C or sonicated prior to the labeling procedure.

The [${}^{125}$I]DDISA labeling procedure appears to minimally perturb the native membrane as evidenced by the fact that the oxalate-promoted, ATP-dependent Ca$^{2+}$ transport activity is not altered. That [${}^{125}$I]DDISA labeling has a minor effect on vesicle function probably relates to the fact that the label reacts rapidly with the membrane protein at 2°C. This contrasts with the classically used lactoperoxidase labeling method (9, 27) which requires treatment at 25°C for about 30 min and the presence of H$_2$O$_2$.

If it is assumed as established that [${}^{125}$I]DDISA is a nonpermeant labeling agent, then the data presented in Fig. 3 provide strong evidence for the location on the external surface of the SR vesicle of much of the high molecular weight proteins.
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FIG. 6. Effect of treatment of SR vesicles with trypsin on the pattern of protein components observed on analysis by SDS-gel electrophoresis. The electrophoretograms in a relate to treatment with Sepharose-immobilized trypsin and those in b to treatment with free trypsin. The number of minutes treated in each case is from left to right 0, 5, 10, 30, and 60. The numbers either refer to the SR protein components as designated in Fig. 1 or to hydrolysis products as designated in the text.

Fig. 7. Analysis of proteins from trypsin-treated SR vesicles on 8.5% SDS-polyacrylamide gels. The number of seconds treated is from left to right 0, 20, 60. The numbers either refer to the SR protein components as designated in Fig. 1 or to hydrolysis products as designated in the text.

comprising electrophoretic Band 1 of Fig. 1 and of the (Ca^{2+} + Mg^{2+})-ATPase. These data also strongly indicate that neither the high affinity calcium binding protein nor the calsequestrin are on the external surface of the SR vesicle. That both phospholipase C treatment (Fig. 5a) and sonication (Fig. 5b) of the vesicles which perturb the native membrane enables [125I]DDISA to readily label calsequestrin and high affinity Ca^{2+} binding protein further supports the conclusion that these protein components are remote from the external surface of vesicles not perturbed by these means. These data are consistent with the model of organization of protein components in SR membranes proposed by MacLennan (1) but disagree with the conclusion of Ikemoto et al. (2) that the (Ca^{2+} + Mg^{2+})-ATPase does not project from the external surface of SR membranes and the conclusion of Thorley-Lawson and Green (3) that calsequestrin is on the external surface of isolated SR vesicles.

Our findings on the effect of brief treatment of rat SR vesicles with Sepharose-immobilized trypsin are similar to trypsin studies reported for rabbit SR vesicles by Inesi and...
Scales (18) and Louis et al. (22) in regard to the disappearance of the \((\text{Ca}^{2+} + \text{Mg}^{2+})\)-ATPase and the appearance of two fragments presumably derived from the ATPase.

On the basis of the great resistance of these trypsin-produced fragments to further trypsin digestion, Inesi and Scales (18) have suggested that a significant portion of this ATPase is imbedded within the SR membrane, and Louis et al. (22) have reached a similar conclusion. Similarly, our findings that treatment of SR vesicles labeled with \([^{14}\text{C}]\text{DDISA}\) with immobilized trypsin leads to the production of a fragment with a 65,000 molecular weight more highly labeled and a fragment with a 56,000 molecular weight less highly labeled than the parent ATPase indicate that part of the ATPase is buried within the membrane. These data lead to the tentative conclusion supported by the antigen-antibody studies of MacLennan (1) that much of the larger molecular weight fragment of the \((\text{Ca}^{2+} + \text{Mg}^{2+})\)-ATPase is located in an exposed fashion on the external surface of the SR vesicles while the smaller molecular weight fragment tends to be buried within the SR membrane.

Moreover, the fact that the specific activity of both fragments decreases as the time of treatment with trypsin increases suggests that some ATPase molecules are more exposed to the labeling agent than others. Further evidence on this point comes from the analysis of the remaining intact \((\text{Ca}^{2+} + \text{Mg}^{2+})\)-ATPase: e.g. after 15 min of immobilized trypsin treatment the remaining ATPase has a specific activity of 0.7 of that of the ATPase prior to the trypsin treatment.

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