tion of the light microscope (*13*). Indeed, Tischendorf (*14*) has reported changes in the appearance of micrographs of bone at the lamellar level as a result of short-term loading; however, it is difficult to interpret these changes as lamellar slippage.

Composite-theoretical analyses have predicted upper and lower bounds for the elastic moduli of bone on the basis of the elastic properties of two phases, collagen and apatite (*15*). The compliant and viscous-like character of protein-polysaccharides from synovial fluid and from soft connective tissue has led to the hypothesis that polysaccharides in bone, particularly those at the cement lines, must be included as a third phase in any composite theoretical approach to modeling the elastic and anelastic properties of bone (*3, 16*). The results presented here indicate that it is the long-term anelastic behavior of bone in particular that results from viscous-like slip at the cement lines.

Bones adapt to stress in such a way as to become better able to support the stress (Wolff's law). The hypothesis presently accepted by many as the mechanism for this adaptive behavior is that bone cells respond to electric potentials generated by stress by means of a piezoelectric-like effect in the bone matrix (*17*). Cement line motion may constitute a secondary mechanism whereby bone responds to the static portion of the loads applied to it, as follows. Cement line motion over a period of months or years could play a role in the realignment of the osteons to the direction of time-averaged principal stress. The observed alignment of bone's material axes can deviate up to 10° from the axis of a long bone. If, as theoretical study suggests, active remodeling processes are insensitive to torsional stress about bone's material axes (*18*), then only a passive process, such as cement line slippage, could be expected to bring about this realignment of axes.

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8. A "shoulder" at least 7 mm in diameter was provided at the ends of the gauge section of the specimen to prevent it from screwing further into the threaded socket in the testing apparatus.
9. During the 5 to 15 minutes required to take the photomicrographs, the specimen recovered a portion of the residual strain. Since most of the creep deformation occurred over a long period (of the order of weeks), the portion of strain recovered in 15 minutes was small. See, for example, J. D. Ferry, *Viscoelastic Properties of Polymers* (Wiley, New York, ed. 2, 1970).
10. Creep refers to an experiment in which a step stress is applied and the strain is measured as a function of time. The creep compliance is defined as the strain divided by the (constant) stress.
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12. A bacteriostatic additive was placed in the Ringer solution to prevent decay of the bone specimen. Evidence for effects due to soaking alone on the mechanical properties of compact bone is equivocal at this point. For example, R. C. Tennyson, R. Ewert, and V. Niranjan [*Exp. Mech.* **12**, 502 (1972)] noted a 33 percent decrease in the compressional stiffness of bovine bone after immersion for 20 days in plain water and repeated impacts exceeding the yield point. Smaller decreases in stiffness were observed in human cancellous bone after immersion for 45 hours in distilled water, according to E. R. Fitzgerald [*Biorheology* **12**, 397 (1975)].
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Chicken Gizzard: Relation Between Calcium-Activated Phosphorylation and Contraction

Abstract. *Of the proteins in mechanically disrupted chicken gizzard fibers (no functional sarcolemma) only the 20,000-dalton light chains of myosin underwent large Ca^{2+}-and Sr^{2+}-dependent changes in phosphorylation. Phosphorylation closely corresponded with the Ca^{2+}- and Sr^{2+}-activated tensions. Adenosine 5'-O (3'-thiotriphosphate) only in the presence of Ca^{2+} induced irreversible Ca^{2+}-insensitive activation of tension and thiophosphorylation of the 20,000-dalton light chains, and blocked incorporation of ^{32}P from [γ-^{32}P]adenosine triphosphate into the myosin light chains.*

Since the discovery of a Ca^{2+}-sensitive myosin control system in the chicken gizzard (smooth muscle) (*1*), considerable effort has been devoted to elucidating the mechanism of activation of contraction. Further work has suggested that phosphorylation of the myosin (*2*) occurs over the same range of Ca^{2+} concentrations as the adenosinetriphosphatase activity of the actomyosin (*3*).

The best evidence that phosphorylation plays a role in the Ca^{2+}-activation of chicken gizzard (smooth muscle) has come from studies on the Ca^{2+}-dependent light-chain kinase isolated from chicken gizzard (*3–5*). This kinase phosphorylates the light chains of the myosin (*4*) and has a 105,000- and a 17,000-dalton component (*4*). The 17,000-dalton component appears to be the Ca^{2+} receptor site and to have molecular weight, amino acid composition, and biological activity similar to those of the modulator protein (*5*). In the presence of the light-chain kinase, phosphorylation of the 20,000-dalton light chain, the heavier of the two types of myosin light chains, and adenosinetriphosphatase activity are triggered in gizzard muscle actomyosin in the same range of Ca^{2+} concentrations. Other investigators studying smooth muscle present strong evidence that phosphorylation of the 20,000-dalton light chains of myosin is a requirement for actomyosin adenosinetriphosphatase (*6*). Support for the hypothesis that phosphorylation of the 20,000-dalton light chains alone could cause the activation of this enzyme comes from studies with platelet or myoblast myosin (*7*). All of these data are circumstantial, however, since they relate phosphorylation to enzyme activity, a biochemical measure of contraction, and not to the physiological measure, tension; therefore, we conducted the present study. Using functionally skinned chicken gizzard fibers, we identified the protein phosphorylation which was dependent on intracellular Ca^{2+} and determined the relationships between intracellular Ca^{2+}, phosphorylation, and activated tension.

Mechanically disrupted bundles of chicken gizzard muscle fibers were prepared by light homogenization as described by Kerrick and Krasner for mammalian skeletal muscle (*8*). These fiber bundles were freely permeable to ions physiologically and biochemically, as indicated by their graded tension response to changing concentrations of Ca^{2+} or Sr^{2+} (Fig. 1) in the presence of adenosine triphosphate (ATP). A rigor state, produced by the removal of ATP

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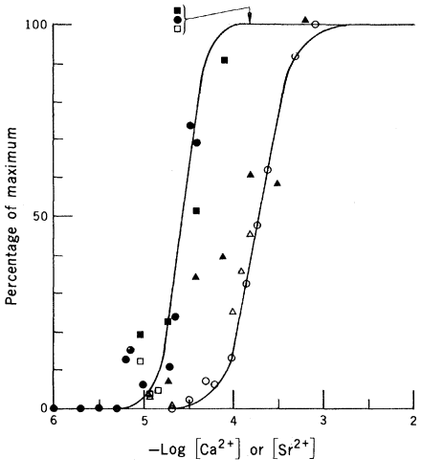

Fig. 1. Comparison of the percentage of maximum tension generation and phosphorylation of myosin light chains in mechanically disrupted chicken gizzard fibers in relation to $p\mathrm{Ca}^{2+}$ and $p\mathrm{Sr}^{2+}$ ($-\log [\mathrm{Ca}^{2+}]$ or $[\mathrm{Sr}^{2+}]$). The smooth curves were fitted to the tension data according to the Hill equation. Data for each set of measurements were normalized as percentages of the corresponding maxima. Symbols: ■, Ca^{2+}, phosphorylation (30 minutes); □, Ca^{2+}, phosphorylation (20 minutes); ▲, Sr^{2+}, phosphorylation (30 minutes); △, Sr^{2+}, phosphorylation (20 minutes); ●, Ca^{2+}, tension; and ○, Sr^{2+}, tension.

in the presence of Ca^{2+}, will prevent relaxation in the absence of Ca^{2+} or shortening in the presence of Ca^{2+}. The further addition of ATP reverses the rigor state and Ca^{2+} insensitivity. Biochemically, gizzard fibers also proved to be freely permeable to [γ-^{32}P]ATP (Fig. 2 and Table 1). Thus, the fiber bundles consisted of many functionally disrupted (no functional sarcolemma) muscle cells.

These bundles were mounted in a tension transducer similar to that described by Hellam and Podolsky (*9*) and isometric tension was monitored as they were immersed in solutions of different Ca^{2+} and Sr^{2+} concentrations (Fig. 3). All test solutions contained 70 m*M* K^+, 2 m*M* $MgATP^{2-}$, 1 m*M* Mg^{2+}, 7 m*M* [ethylenebis (oxyethylenenitrilo)] tetraacetic acid, 10^{-8} to $10^{-3.2}$*M* Ca^{2+}, 0 to $10^{-3.2}$*M* Sr^{2+}, propionate as the major anion, and an ATP regenerating system consisting of 15 m*M* creatine phosphate and 15 units of creatine kinase per milliliter. Ionic strength was adjusted to 0.15, and *p*H was maintained at 7.00 ± 0.02 with imidazole propionate. Temperature was maintained at 21° ± 1°C. Fiber bundles were also immersed in solutions similar to the tension solutions except that they contained [γ-^{32}P]ATP or ^{35}S-labeled adenosine 5′-*O*(3′-thiotriphosphate) (ATPγS), 85 m*M* K+, and no creatine phosphate or creatine kinase–ATP regenerating system. The fibers were incubated in [γ-^{32}P]ATP solutions for 20 or 30 minutes and then transferred to boiling sodium dodecyl sulfate (SDS). The fiber proteins were then electrophoresed on SDS-polyacrylamide gels (7.5 percent) and stained with Coomassie blue. Molecular weights were estimated from these gels with reference to known standards. The gels were scanned with a Gilford spectrophotometer and cut into 5-mm slices. The slices were dissolved in 30 percent hydrogen peroxide and counted in a scintillation counter. The amounts of phosphate or sulfur incorporated into the individual gel slices were determined and compared with the densitometry scans (Fig. 2) and, after normalization to the amount of protein present, with the Ca^{2+}- and Sr^{2+}-activated tensions (Fig. 1). Fluorographs (*10*) were used to observe incorporations of ^{32}P and ^{35}S into fibers under different experimental conditions (Fig. 4).

The amount of ^{32}P-labeled phosphate incorporated into the proteins of the gizzard fibers in the presence and absence of Ca^{2+} differed significantly only in the 20,000-dalton light chains; phosphorylation was much greater in the presence of Ca^{2+} than in its absence (Fig. 2). It is apparent that Ca^{2+} activates the kinase which phosphorylates the 20,000-dalton light chains of myosin.

We then investigated the relationships between intracellular concentrations of Ca^{2+} or Sr^{2+}, phosphorylation of the 20,000-dalton light chains, and isometric tension generation in these fibers. Figure 1 shows that for half-maximal activation of tension, the Sr^{2+} concentration must be approximately eight times higher than Ca^{2+} concentration. The sensitivity of phosphorylation of the 20,000-dalton myosin light chains to Ca^{2+} and Sr^{2+} concentrations is similar to that of activation of tension. These data show that in the chicken gizzard, tension (the physiological measure of contraction) is generated and phosphorylation of the myosin light chains is triggered over the same divalent cation concentration ranges of Ca^{2+} and Sr^{2+}. This phosphorylation is reversible in the absence of Ca^{2+} and presence of [γ-^{32}P]ATP (Fig. 4).

Gratecos and Fischer (*11*) reported that ATPγS can be used as a substrate for kinases, but that thiolabeled phosphate incorporated into proteins is resistant to phosphorylase phosphatase (E.C. 3.1.3.17). Sherry *et al.* (*12*) observed that gizzard light-chain kinase will use ATPγS to incorporate thiolabeled phosphate into the 20,000-dalton light chains of gizzard and that this phosphate is resistant to the action of phosphatases and consequently prolongs the activation of actomyosin adenosinetriphosphatase. In the present study, when functionally disrupted fiber bundles were incubated for 20 minutes in ATPγS (only in the presence of Ca^{2+}) and returned to a normal relaxing solution containing ATP and 10^{-8}*M* Ca^{2+}, the fibers contracted to a maximum and were essentially insensitive to changes in Ca^{2+} concentration (Fig. 3). These fibers were not in rigor; they relaxed in 2 m*M* (α,β-CH_2)-ATP or pyrophosphate (data not shown) and redeveloped tension in solutions containing ATP and low Ca^{2+} concentrations. (α,β-CH_2)-ATP dissociates actin and myosin but does not serve as a substrate for contraction (*13*).

Table 1. The incorporation of ^{32}P by the 20,000-dalton myosin light chains of fiber bundles that had been incubated in 2 m*M* $MgATP^{2-}$ or $MgATP\gamma S^{2-}$.

First incubation solution*	^{32}P incorporation from second incubation solution* ([α-^{32}P]$MgATP^{2-}$) (counts per 10 minutes)
$MgATP^{2-}$	68,100 ± 3,800
$MgATP\gamma S^{2-}$	8,600 ± 600

*pCa 3.8.

Figure 4A shows that the phosphorylation of the 20,000-dalton light chain is reversible at low Ca^{2+} concentrations (Fig. 4A, c′ and d′) corresponding to muscle relaxation. In contrast, Fig. 4B shows that thiophosphorylation of the 20,000-dalton light chains (which only occurs in the presence of Ca^{2+}) is not reversible at low Ca^{2+} concentrations (Fig. 4B, c′) and corresponds to a Ca^{2+}-insensitive fully activated state of contraction. Equally important, the only protein thiophosphorylated in the activated state is the 20,000-dalton light chain. Prior incubation of the fibers for 30 minutes in an ATPγS solution containing a high concentration of Ca^{2+} also blocked the incorporation of ^{32}P from [γ-^{32}P]ATP into the 20,000-dalton light chains (Table 1). The maximum change in incorporation of ^{32}P and ^{35}S was 0.2 and 0.8 mole per mole of light chain, respectively. This discrepancy in incorporation would in part be expected since phosphatases would be active against phosphorylated light chains but not thiophosphorylated light chains. This change of 0.2 mole of ^{32}P per mole of light chain is similar to that in frog myofibrils from experiments in vivo (*14*), but is less than that found in the trichloroacetic acid–insoluble residue of the same frog skeletal muscle (*14*) or in rabbit skeletal muscle (*15*). However, the incorporation of 0.8 mole of ^{35}S per mole of light chain is close to the expected value of 1.0 for fully phosphorylated light chains (*2*, *5*).

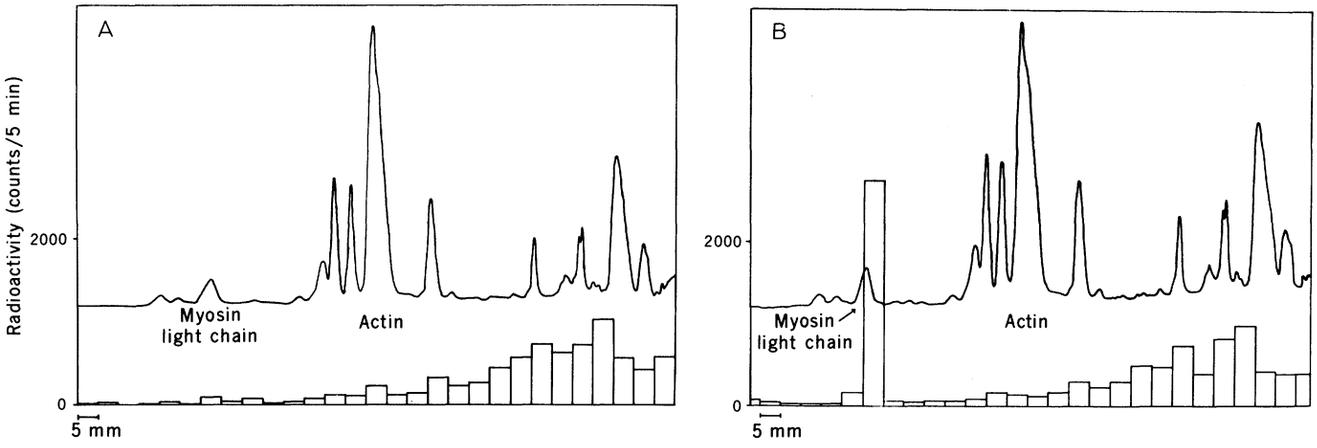

Fig. 2. Phosphorylation (^{32}P, counts per 5 minutes represented by bars) in 5-mm slices of SDS-polyacrylamide gels (7.5 percent) obtained from electrophoresis of proteins from mechanically disrupted chicken gizzard fibers incubated in solutions of (A) *p*Ca 8 (relaxation-conditions) and (B) *p*Ca 3.8 (contracting conditions). For reference to the corresponding proteins present, scans of the gels are also shown.

Our interpretation of these data in view of the known actions of kinases and phosphatases on ATPγS and thiophosphorylated proteins is that thiophosphate is incorporated into the 20,000-dalton myosin light chains and cannot be easily removed by the phosphatases. As a result, the chicken gizzard fibers are fully activated in the absence of Ca^{2+}. If this interpretation is correct, then phosphorylation of the 20,000-dalton light chains is the controlling requirement for activation of the contractile proteins rather than both high concentrations of Ca^{2+} and phosphorylation.

A similarity exists between the data on phosphorylation presented here for smooth muscle and those published for skeletal muscle. In frog (*14*) and rabbit (*15*) skeletal muscle, the 18,000-dalton light chains of myosin incorporate twice as much phosphate during tetanic stimulation as in the relaxed state of the muscle. Several investigators (*16*) have shown that a Ca^{2+}-sensitive light-chain kinase from the rabbit can phosphorylate the 18,000-dalton myosin light chains. This finding is consistent with the hypothesis that phosphorylation may play some general role in Ca^{2+} activation of muscle contraction, although this role seems unclear since the ATP activity in rabbit skeletal myosin does not depend upon the 18,000-dalton light-chain content (*17*). Myosin light-chain phosphorylation by itself or in combination with other calcium-binding regulatory proteins may be a common feature in the activation of muscle.

Our data show that the only protein in the mechanically disrupted fibers of chicken gizzard undergoing a significant change in phosphorylation during the activation of tension is the 20,000-dalton light chain of myosin. The same ranges of concentration of Ca^{2+} or Sr^{2+} are re-

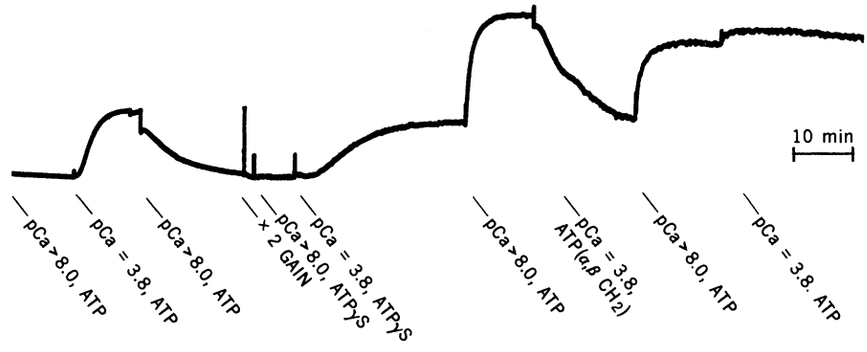

Fig. 3. The tension-time record of a fiber bundle. A comparison of contractions during and after exposure to 2 m*M* $MgATP\gamma S^{2-}$ solutions in the presence and absence of Ca^{2+} with similar control contractions in 2 m*M* $MgATP^{2-}$ solutions. Tension increases upward. Time of solution changes, Ca^{2+} concentration, and type of nucleotide used are indicated by short, solid line; *× 2 GAIN* indicates that the gain of the amplifier was increased by 2. The solution composition is described in the text.

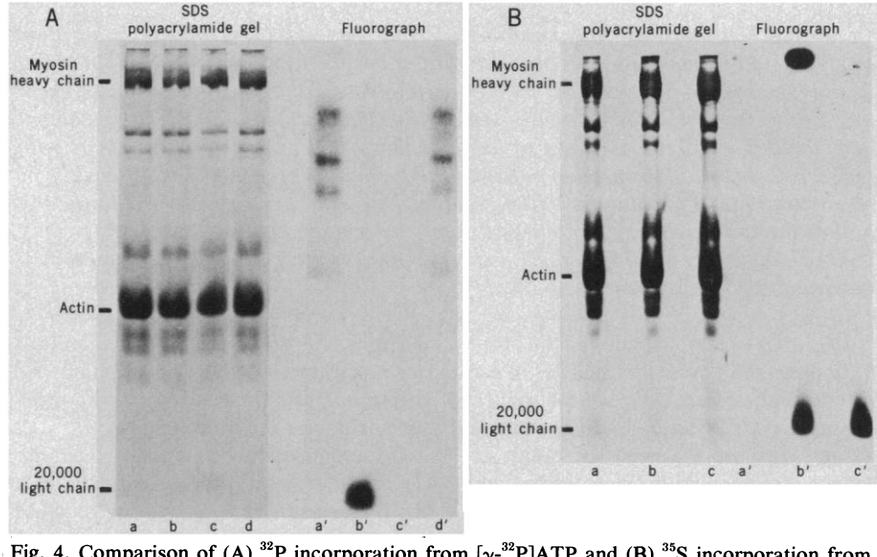

Fig. 4. Comparison of (A) ^{32}P incorporation from [γ-^{32}P]ATP and (B) ^{35}S incorporation from ATPγS into fiber proteins. Mechanically disrupted fibers were exposed to solutions containing (a, a') a low $Ca^{6>}$ concentration (*p*Ca 8) and 2m*M* labeled nucleotide; (b, b') a high Ca^{2+} concentration (*p*Ca 3.8) and 2m*M* labeled nucleotide; (c, c') a high Ca^{2+} concentration (*p*Ca 3.8) and 2 m*M* labeled nucleotide followed by a low Ca^{2+} concentration (*p*Ca 8) and 2 m*M* cold ATP; or (d, d') a high Ca^{2+} concentration (*p*Ca 3.8) and 2m*M* labeled nucleotide followed by a low Ca^{2+} concentration (*p*Ca 8) and 2 m*M* [γ-^{32}P]ATP. Fibers were exposed for 20 minutes to each solution. Solutions had the same composition as those used for the tension measurements shown in Fig. 3.

quired for phosphorylation and activation of tension. Furthermore, by the use of ATP analog, ATPγS, we have shown that although Ca^{2+} is required for the initial thiophosphorylation of the 20,000-dalton light chain, thereafter Ca^{2+} is not required for the maintenance of either thiophosphorylation of the light chains or tension. This indicates that it is the phosphorylation of the light chain rather than the presence of Ca^{2+} which is required for the activation of tension in smooth muscle fiber preparations. This same conclusion was reached by Hartshorne and co-workers (*12*) for the soluble actomyosin Ca^{2+}-sensitive adenosinetriphosphatase from chicken gizzard when they used ATPγS.

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Selective Phospholipid Adsorption and Atherosclerosis

Abstract. *Disaturated (fully saturated) lecithins adsorb onto solid surfaces more readily than lecithins in which one or both fatty acids are unsaturated. If saturated lecithins adsorb to arterial walls as they do to glass and polystyrene surfaces, there may be increased probability of atherosclerosis when the disaturated lecithin content of plasma is elevated. Analyses of lecithins in plasma samples from patients with myocardial infarction, and from patients with premature atherosclerosis but with low concentrations of plasma cholesterol and triglycerides, are consistent with the hypothesis that a high concentration of disaturated lecithin in plasma may be a significant risk factor for atherosclerosis, independent of triglyceride and cholesterol concentrations.*

In order to supplement recent observations on the physical properties of lecithin and cholesterol (*1*), I measured the adsorption of phospholipids in aqueous dispersions on a variety of solid surfaces. Among synthetic lecithins containing different combinations of fatty acids a highly selective adsorption was found for those in which both fatty acids were saturated. These results suggested that if such phospholipids in plasma also adsorb on arterial walls, elevated plasma concentrations of disaturated (fully saturated) lecithins might be related to the presence of atherosclerosis. Assays of these lipids in plasma were therefore made. Results of the phospholipid adsorption studies and of the plasma measurements are reported here.

Dispersions of pure synthetic lecithins (*2*) in water were prepared by dialysis (*3*). As shown in Fig. 1, appreciable amounts of the disaturated dimyristoyl lecithin (DML) and dipalmitoyl lecithin (DPL) adsorbed on the surface of polystyrene beads (*4*). The amount adsorbed increased with increasing concentration of lipid in the dispersion and, at 20°C and high dispersion concentrations, reached more than twice the amount expected for condensed monolayer adsorption. The amount adsorbed decreased with increasing temperature. Under identical conditions more DPL was adsorbed than DML.

The data of Fig. 1 are equilibrium values, attained within 1 to 2 hours of mixing the beads with the dispersions, and were constant for at least 3 days. If the DPL system at 20°C was raised to a higher temperature, lipid desorbed from the beads until a new temperature equilibrium was reached. This desorption process at higher temperatures took several days instead of the 1 to 2 hours required to reach adsorption equilibrium.

In marked contrast to the behavior of the disaturated lecithins, DML and DPL, no adsorption to polystyrene beads occurred with dispersions of the unsaturated lecithins, dioleoyl lecithin (DOL) and 1-palmitoyl-2-palmitoleoyl-lecithin (PPoL). If either or both fatty acid moieties of the lecithin were unsaturated, adsorption was greatly reduced or abolished, even at temperatures as low as 5°C.

The intrinsic adsorption properties of DPL were not affected by the presence of large amounts of DOL in the dispersion. When DPL was codispersed with a fivefold excess of DOL at 50°C and the amount of DPL adsorbed plotted against the amount of DPL in the mixed dispersion, the points fell on the 50°C adsorption isotherm for pure DPL dispersions shown in Fig. 1.

The influence of the solid surface in the adsorption process was examined by using glass beads (*5*) and comparing the adsorption of DPL, DOL, and PPoL under conditions similar to those in the polystyrene bead experiment. As before, DPL was adsorbed while no adsorption occurred with DOL and PPoL. If the particle size of the two types of beads was taken into account, the amount of DPL adsorbed per unit surface area was approximately the same on both polystyrene and glass beads (Fig. 1). Thus both lecithin selectivity and extent of adsorption were independent of major differences in the chemical nature of the adsorptive surface, suggesting that the adsorption of disaturated lecithins can occur under a variety of conditions. A recent report (*6*) indicates that DML and DPL adsorb to membranes of Chinese hamster fibroblasts with temperature dependence similar to that in the polystyrene adsorption studies reported here (Fig. 1); in contrast, DOL, under similar conditions, is incorporated into the cell.

The unusual adsorption properties of the lecithin dispersions raised the possi-

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