Nature of the Interaction of Dextran Sulfate with High and Low Density Lipoproteins in the Presence of Ca$^{2+}$

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The interactions of dextran sulfate with high density lipoproteins (HDL) and with low density lipoproteins (LDL) were studied to differentiate their mechanisms and to provide some information of the charge characteristics of these lipoproteins. The insoluble complex formation with high density lipoprotein 3 (HDL₃) progressively increased with increases of the Ca$^{2+}$ concentration; complete conversion occurred with 60 mM CaCl₂. Between the two major subclasses, HDL₄ was more effectively converted to insoluble complex at lower CaCl₂ concentrations than HDL₃. It appeared that primarily the phospholipid components of HDL were involved in the formation of the insoluble complex and that the protein components reduced the complex forming ability of HDL. However, both apolipoproteins A-I (apo-A-I) and A-II (apo-A-II) possessed a definite reactivity with dextran sulfate in the presence of divalent metal ions, and a reduction in the ionic strength progressively enhanced their complex formation. In addition, modification of the apo-A-I carboxyl groups with glycine methyl ester, after activation with carbodiimide, was found to greatly increase its complex forming ability. Although LDL strongly interacted with dextran sulfate, primarily involving the positive charges of the protein moiety, increases in the Ca$^{2+}$ concentration seemed to cause a progressive increase in the participation of the zwitterionic polar heads of the LDL phospholipids in complex formation. The study of the interaction of the preincubated apo-A-I and lyssolecithin mixtures with dextran sulfate in the presence of Ca$^{2+}$ revealed that these two components produced a molecular complex which interacted with dextran sulfate as an organized unit.

Sulfated polysaccharides and other polyanions interact with human plasma lipoproteins under a variety of conditions. LDL and very low density lipoproteins form insoluble complexes with sulfated polysaccharides even in the absence of divalent metal ions (1, 2). However, HDL requires high concentrations of both divalent metal ions and sulfated polysaccharides (2). It has recently been postulated on the basis of epidemiological studies that, while low density lipoproteins are atherogenic, high density lipoproteins exert antiatherogenic effects (3-5). Unlike LDL, HDL does not possess an affinity with aortic glycosaminoglycans (6, 7), and uptake by human arterial smooth muscle cells is rather limited (6, 9). These different behaviors may reflect in part differences in the charge characteristics and organization of the lipoprotein surfaces. Some basic information on the surface properties may be derived from a systematic study of the lipoprotein-polysaccharide interaction since the interaction is primarily governed by polar forces (1, 2).

We previously studied the interaction of LDL with dextran sulfate (10-13). In the absence of divalent metal ions, the interaction involved primarily the positively charged groups of the protein moiety (7, 19). However, in the presence of divalent metal ions, the charged groups of the phospholipids seemed to also contribute to the interaction (13, 14). To provide basic information on the nature of the involvement of lipoprotein phospholipids, we examined the interaction utilizing lecithin dispersions and lyssolecithin micelles (15).

The present communication describes the differences in the mode of interaction of dextran sulfate with HDL and LDL, and the charge characteristics of their constituents influencing the interaction.

MATERIALS AND METHODS

Buffer—Unless otherwise indicated, experiments were performed with Tris buffer, pH 7.4, ionic strength of 0.1.

Preparation of Dextran Sulfate—The sodium salt of dextran sulfate, with a sulfur content of 16.1% corresponding to 1.7 sulfate groups/hexose unit, was prepared from dextran with an average molecular weight of 150,000 according to the method of Ricketts (18) as previously described (10).

Preparation of LDL and HDL—Human plasma LDL of the Sf 0 to 10 class was isolated from fresh nonturbid human plasma and purified by ultracentrifugation at 4°C (10, 13). Total HDL (d < 1.006 to 1.210 g/cm³) was isolated and purified as described previously (19). To obtain HDL₂ and HDL₃, the total HDL fraction was dialyzed against a NaCl/NaBr solution of d = 1.125 g/cm³ and centrifuged at 105,000 x g for 36 h. The HDL₂ fraction was removed from the top of the tube. The bottom fraction containing HDL₃ was adjusted to a density of 1.210 g/cm³ with NaBr and then centrifuged at 105,000 x g for 36 h to float the HDL₃ to the top of the tube. Both HDL₂ and HDL₃ fractions were purified by recentrifugation after a 1:4 dilution with NaCl/NaBr solution of d = 1.125 and 1.210 g/cm³, respectively. All media used for centrifugation contained 0.025% EDTA (pH 7.4) and 2 mM NaNO₂. After purification, all lipoprotein fractions were dialyzed against a buffer containing 0.025% EDTA and 2 mM NaNO₂. After dialysis, all lipoprotein fractions were treated with 3 mM sodium carbonate and 1 mM sodium bicarbonate, pH 9.5, and then dialyzed against the buffer containing 0.025% EDTA and 2 mM NaNO₂.
54% protein and 46% lipids for HDLs. The methods used for the determination were previously described (19).

Succinylation and Acetylation of HDLs—HDLs were succinylated in a manner similar to that described for LDL (13). The molar ratio of succinic anhydride/lysine residues was 14. Acetylation of HDLs was performed as previously described (10) using a molar ratio of acetic anhydride/lysine residues of 6. The extent of acetylation of the free amino groups was assayed by the trinitrobenzene sulfonic acid procedure (13). The succinylation and acetylation modified 96 and 97%, respectively, of the amino groups of HDLs.

Separation of Apo-A-I and Apo-A-II—The purified total HDL in a 0.1 M NaCl medium (d = 1.21 g/ml) was dialyzed against 0.025 M EDTA, pH 7.4, at 4°C, and extracted with ethanol/ether mixtures by the method of Shore and Shore (20). Apo-A-I and apo-A-II were fractionated by gel permeation chromatography on Sephadex G-150 and subsequent DEAE-cellulose column chromatography (21) at 4°C. The purified apo-A-I and apo-A-II exhibited well defined single bands on sodium dodecyl sulfate-polyacrylamide gel electrophoresis. The apolipoproteins were stored in 6 M urea at 4°C under nitrogen and were dialyzed against buffer prior to each use.

Modification of Apo-A-I Carboxyl Groups—The carboxyl groups of apo-A-I were modified by reaction with [1-14C]glycine methyl ester and 1-ethyl-3-(3-dimethylaminopropyl)carbodiimide HCl essentially according to the method of Hoare and Koshland (22). [1-14C]Glycine methyl ester was prepared according to the procedure of Wells et al. (23). Thin layer chromatography of the labeled preparation using 1-butanol/acetic acid/H2O (60:20:20, v/v) as the developing solvent showed a single yellow spot with ninhydrin with a Rf value higher than that of glycine. The spot completely superimposed with the radioactivity on the chromatogram. The modification of apo-A-I (4 mg/ml) was carried out at 25°C in 6 M urea at pH 4.75 using glycine methyl ester and the carbodiimide at the final concentrations of 1.0 and 0.4 M, respectively. The reaction was stopped by the addition of an equal volume of 4 M acetic acid buffer (pH 4.7). The mixtures were first dialyzed against 0.1 M Tris buffer (pH 8.0) containing 6 M urea and then against regular Tris buffer. The extent of carbonyl modification was determined by the specific activity of the modified apo-A-I. Preparations modified to the extents of 16 and 35% were used in the experiments.

Preparation of Lecithin Dispersions and Lysolecithin Micelles—Egg lecithin was purified by neutral alumina column chromatography according to the method of Wells and Hanahan (24) from crude egg lecithin prepared by the method of Faure (25). Lysolecithin (1-acyl-sn-glycero-3-phosphorylcholine) prepared from egg lecithin was purified by the method of Faure (25). Lysolecithin (1-acyl-sn-glycero-3-phosphorylcholine) prepared from egg lecithin was purified by the method of Faure (25). Lysolecithin (1-acyl-sn-glycero-3-phosphorylcholine) prepared from egg lecithin was purified by the method of Faure (25).

Formation and Analysis of Insoluble Complexes—The formation of insoluble complexes with dextran sulfate was studied as described previously (11, 13, 15). Mixtures containing a known amount of lipoprotein, apolipoprotein, or lipid dispersion, and various amounts of dextran sulfate and CaCl2 in 0.75 ml of Tris buffer were kept at room temperature (23–24°C) for 30 min. The insoluble complex formed was separated by centrifugation for 30 min at 2900 × g. The recovery of lipoproteins and apolipoproteins as insoluble complexes was routinely determined by protein analysis, and the recovery of lecithin and lysolecithin was determined by phosphorus analysis of the complex. The amounts of lipoproteins, apolipoproteins, or lipid dispersions used as well as any deviations from the standard procedure are given in the text. It must be noted that at dextran sulfate/lipoprotein weight ratios or Ca2+ concentrations which do not allow the complete precipitation of lipoproteins, the composition of precipitated lipoproteins may not necessarily be the same as that of lipoproteins remaining in supernatant solutions. This reflects the microheterogeneity of LDL and HDL with respect to their compositions and, hence, their reactivities with dextran sulfate. The lipoprotein particles with a higher proportion of the surface components reactive with dextran sulfate (e.g. LDL particles with a higher phospholipid content) are likely to be precipitated more effectively than those with a lower proportion of the reactive component.

Lipid and Protein Determinations—Lipid phosphorus was determined essentially according to the method of Bartlett (26) as modified by Parker and Peterson (27). The amount of phospholipid in the samples is given as the amount of phosphorus × 25. Protein was determined according to the method of Lowry et al. (28) with crystalline bovine serum albumin (Sigma Chemical Co.) dried over phosphorus pentoxide as a standard. Interference with the protein assay by Tris buffer was eliminated by incorporating the same amount of Tris buffer in the standards as described by Rej and Richards (29).

RESULTS

The interaction of HDLs with dextran sulfate was studied at various concentrations of CaCl2 in Tris buffer (Fig. 1). Although no insoluble complex was formed from HDLs in the absence of Ca2+, increasing amounts of complex were formed as the concentration of CaCl2 was increased. An equivalence dextran sulfate/HDL weight ratio was present for the formation of a maximal amount of insoluble complex and was shifted to higher values by increases in the CaCl2 concentration. Complete conversion of HDLs to the insoluble complex was achieved when the CaCl2 concentration was raised to 60 mM (Fig. 1, Curve 5).

In contrast to HDLs, LDL has a high reactivity with dextran sulfate and did not require the presence of Ca2+ for the formation of insoluble complex as described previously (10, 13). Complete conversion of LDL to the insoluble complex was attained at the dextran sulfate/LDL weight ratio of 0.04 in the absence of Ca2+ (Fig. 2, Curve 1). When CaCl2 was added to a concentration of 5 mM, the equivalence weight ratio for maximal complex formation was shifted to a value of approximately 0.07 (Fig. 2, Curve 2). Above CaCl2 concentrations of 10 mM, the complete conversion of LDL to insoluble complex was facilitated presumably by the increased reactivity of LDL with dextran sulfate as a result of Ca2+ addition.

![Fig. 1. The conversion of HDLs to insoluble dextran sulfate-HDL complex in the presence of 5 (Curve 1), 10 (Curve 2), 20 (Curve 3), 30 (curve 4), and 60 (Curve 5) mM CaCl2. Inosoluble complex formation was assayed with mixtures containing 320 μg of HDL and various amounts of dextran sulfate and CaCl2 in 1.5 ml of Tris buffer.](http://www.jbc.org/)

![Fig. 2. Effect of calcium ions on the formation of insoluble dextran sulfate-LDL complex. Curve 1, conversion of LDL to insoluble complex as a function of the dextran sulfate/LDL weight ratio in the absence of Ca2+. Curves 2, 3, 4, and 5, conversion in the presence of 5, 10, 20, and 30 mM CaCl2, respectively. Insoluble complex was formed with 0.8 mg of LDL, various amounts of dextran sulfate, and CaCl2 in 1.5 ml of Tris buffer.](http://www.jbc.org/)
The lower reactivity of HDL₃ with dextran sulfate in comparison to LDL might have been caused in part by the presence of a greater proportion of free negative charges on HDL₃. Thus, the complex forming ability of HDL₃ was expected to be further reduced by chemical modifications that increase the number of negative charges or the net negative charge of HDL₃. When approximately 96% of the HDL₃ amino groups were modified by acetylation and succinylation, the formation of insoluble complex was drastically reduced (Fig. 3, Curves 2 and 3) as compared to untreated HDL₃ (Fig. 3, Curve 1) in the presence of 60 mM CaCl₂. Succinylation effected a slightly larger reduction than did acetylation. When the concentration of CaCl₂ was reduced below 30 mM, neither succinylated nor acetylated HDL₃ produced a significant amount of insoluble complex.

To obtain some information concerning the contribution of the protein components of HDL₃ to the interaction, the most abundant HDL apoproteins, apo-A-I and apo-A-II, were used in the interaction study. It was observed that apo-A-I and apo-A-II produce insoluble complexes with dextran sulfate in the presence of Ca²⁺ (Fig. 4), although the insoluble complex formation was less pronounced than with HDL₃.

Zwitterionic phospholipids, such as lecithin and lysolecithin, can also be converted into insoluble complexes by dextran sulfate in the presence of relatively low concentrations of Ca²⁺ (15). The effectiveness of the conversion of LDL, lecithin, lysolecithin, HDL, HDL₁, HDL₃, apo-A-I, and apo-A-II to insoluble complex at the equivalence ratios as a function of CaCl₂ concentration. Experimental conditions for the conversion of HDL, HDL₁, and HDL₃ were the same as those described for Fig. 1; the conditions for the conversion of LDL and of apo-A-I or apo-A-II were described for Figs. 2 and 4, respectively. For the conversion of lecithin or lysolecithin to insoluble complex, the mixtures containing 200 µg of lecithin or 133 µg of lysolecithin and various amounts of dextran sulfate and CaCl₂ in 0.75 ml of Tris buffer were used.
observed, while maximal conversion of apo-A-II was approximately 89%. When NaCl concentration was increased from 0.05 to 0.2 M, a sharp decrease in complex formation occurred with both apo-A-I and apo-A-II. In order to study the effect of the negative charges of apo-A-I on the formation of insoluble complex, the carboxyl groups of the dicarboxylic amino acid residues were modified with glycine methyl ester and 1-ethyl-3-(3-dimethylaminopropyl)carbodiimide HCl to the extent of 16 and 35%. These preparations were found to produce insoluble complex even in the absence of Ca²⁺. Approximately 46% conversion to insoluble complex was observed at the equivalence weight ratio of 0.05 when 16% of the carboxyl groups were modified (Fig. 7, Curve 1). With modification of 35% of the carboxyl groups, the equivalence ratio was shifted to approximately 0.2 and at this ratio complete conversion to insoluble complex was effected (Fig. 7, Curve 1). Upon addition of CaCl₂ to apo-A-I with 16% modification, the formation of insoluble complex was enhanced and the equivalence weight ratio shifted to higher values, these effects being less pronounced at 10 mM CaCl₂ (Fig. 7, Curve 2) than at 20 mM CaCl₂ (Fig. 7, Curve 3). Since the apo-A-I modified to 35% was completely converted even in the absence of CaCl₂, the main effect of the addition of CaCl₂ (Fig. 7, Curves 4 and 6) was the formation of an extended plateau region as observed with LDL.

![Image](http://www.jbc.org/)

**Fig. 7.** Effect of the modification of apo-A-I carboxyl groups on the conversion of apo-A-I to insoluble complex. Curves 1, 2, and 3, precipitation curves for the apo-A-I with 16% of the carboxyl groups modified in the presence of 0, 10, and 20 mM CaCl₂, respectively. Curves 4, 5, and 6, precipitation curves obtained with the apo-A-I with 35% modification at the CaCl₂ concentrations of 0, 10, and 20 mM, respectively. Experimental conditions were the same as those described for Fig. 4.

![Image](http://www.jbc.org/)

**Fig. 8.** Interaction of dextran sulfate with the lyssolecithin/apo-A-I mixture in the presence of 20 mM CaCl₂. The conversion of lyssolecithin and apo-A-I alone to insoluble complex is shown by Curves 1 and 2, respectively. Curve 3, conversion of the lyssolecithin/apo-A-I mixture to the insoluble complex as determined by both phosphorus (○ - - ○) and protein (△ - - △) assays. The complex was formed from 80 μg of lyssolecithin or 80 μg of apo-A-I, or a mixture of 80 μg of lyssolecithin and 80 μg of apo-A-I in the presence of various amounts of dextran sulfate at a CaCl₂ concentration of 20 mM in 0.75 ml of Tris buffer.

![Image](http://www.jbc.org/)

**Fig. 9.** Effect of the concentration of NaCl on the stability of the insoluble complexes of dextran sulfate with apo-A-I alone (Curve 1), apo-A-I/lyssolecithin mixtures of the apo-A-I to lyssolecithin weight ratios of 1:1 (Curve 2), 1:2 (Curve 3), and 1:3 (Curve 4), and lyssolecithin alone (Curve 5). Insoluble complexes were formed at the equivalence ratios in the presence of 20 mM CaCl₂ in 10 mM Tris buffer of pH 7.4 in a similar manner as given for Fig. 8. The ionic strength of the media containing preformed insoluble complexes was increased by the addition of NaCl; the total volume was 0.75 ml. Prior to centrifugation, the mixtures were kept at room temperature for 30 min. The curves represent the recovery of insoluble complex as a function of NaCl concentration.

The effect of lipid binding to HDL apoproteins on the interaction with dextran sulfate was studied using apo-A-I and lyssolecithin as a model system. A lyssolecithin/apo-A-I mixture containing the components at a weight ratio of 1 gave a precipitation curve (Fig. 8, Curve 2) different from that of lyssolecithin (Fig. 8, Curve 1) or apo-A-I alone (Fig. 8, Curve 3). The recovery of apo-A-I from the insoluble complex was considerably enhanced by the presence of lyssolecithin. Furthermore, identical recoveries of apo-A-I and lyssolecithin at all weight ratios indicated that dextran sulfate interacted with organized lyssolecithin/apo-A-I units rather than interacting separately with lyssolecithin and apo-A-I. The equivalence ratio for the formation of insoluble complex from the lyssolecithin/apo-A-I mixture was higher than that for the complex formation from lyssolecithin or apo-A-I alone.

![Image](http://www.jbc.org/)

**DISCUSSION**

Although the major HDL apoproteins, apo-A-I and apo-A-II, possessed a definite reactivity with dextran sulfate in the presence of divalent metal ions, their presence seemed to reduce the complex forming ability of the phospholipids in the intact lipoproteins. The interfering effect of HDL proteins on the complex formation appears to be caused by the presence of a large number of free negative charges. The succinylation or acetylation of HDL aggravated the interfering effect by
increasing the number of negative charges or the net negative charge of the HDL proteins.

The differences in the complex forming abilities of LDL and HDL can be best explained by comparing the plots of insoluble complex formed from these lipoproteins and some of their major constituents as a function of Ca$^{2+}$ concentration (Fig. 5). The effectiveness of LDL in producing insoluble complex even in the absence of Ca$^{2+}$ appears to reflect the participation of the protein positively charged groups in the interaction despite the net negative charge present on the surface (13). The conversion of lecithin vesicles or lysolecithin micelles to insoluble complex required the presence of Ca$^{2+}$ and appeared to represent mutually enhancing interactions involving both positive and negative charges of the zwitterionic phospholipids (15). These are the direct electrostatic interaction between the phospholipid choline nitrogen and the sulfate groups of dextran sulfate, and the calcium cross-linking of the phosphate groups to the sulfate groups (Model A) or to the phosphate groups of neighboring phospholipids (Model B). It is possible that a large portion of the positive and negative charges on apo-A-I and apo-A-II may exist as ion pairs (16, 17) which could interact with dextran sulfate in a manner similar to that of the zwitterionic polar heads of lecithin or lysolecithin. The inefficient conversion of the apolipoproteins as compared to the zwitterionic phospholipids suggests that the interaction of the ion pairs may be reduced by the abundance of free negative charges present on apo-A-I and to a greater extent on apo-A-II. This explanation was substantiated by the formation of insoluble complex by apo-A-I with the carboxyl groups modified, even in the absence of Ca$^{2+}$. The decrease in the negative charges of apo-A-I by the modification and the consequent exposure of some positive charges which were involved in ion pairs apparently facilitated the interaction. Conversely, untreated apo-A-I and apo-A-II required high concentrations of Ca$^{2+}$ for the interaction of the ion pairs with the sulfate groups of dextran sulfate and for the charge destruction or neutralization of ionized carboxyl groups. We previously showed that an increase in the net negative charge present on HDLz (30) could have provided a favorable interaction. The higher equivalence weight ratio obtained for the mixture indicates a large increase in the participation of the sulfate groups in the interaction. This increased participation may be explained by the differences in the mode of the interaction of dextran sulfate with lysolecithin molecules in micelles and with those in lysolecithin-apo-A-I complex. When lysolecithin is present in the form of micelles, calcium cross-linking may occur between the phosphate groups of neighboring lysolecithin molecules due to their proximity (Model B) (15). However, the separation of individual lysolecithin molecules in the lysolecithin-apo-A-I complex may favor calcium cross-linking between the phosphate groups of lysolecithin and the sulfate groups of dextran sulfate (Model A), which requires twice the number of sulfate groups that are involved in Model B. These considerations support the contention that the lysolecithin-apo-A-I interaction occurs through penetration of monomeric lysolecithin molecules into apo-A-I (40, 42) rather than insertion of the apo-A-I into the lysolecithin micelles (41). Sufficient spacing out of the lysolecithin molecules between preformed or newly formed amphipathic helices (16, 17), or both, may be important in enhancing the flexibility of the peptide chain (42).

The higher equivalence ratio obtained for the apo-A-I-lysolecithin complex also indicates the primary involvement of hydrophobic interactions in the association of apo-A-I with lysolecithin. If the electrostatic interaction between the phospholipid polar heads and the protein ion pairs was substantially involved in the association, the number of lysolecithin polar heads and protein ion pairs available for interaction with dextran sulfate would be greatly reduced. Nevertheless, weak ionic interactions or hydrogen bonding could occur between the zwitterionic polar heads and the charged amino acid residues of apo-A-I since the comparison of the ionization behavior of apo-A-I and of its complex with dimyristoyl lecithin revealed some masking of the acidic and basic residues in the complex (43). Such weak interactions, however, can be effectively eliminated by the strong interaction of dextran sulfate with the lysolecithin-apo-A-I complex. The importance of the hydrophobic association was previously indicated by observations that the lysolecithin-apo-A-I interaction was not influenced appreciably by changes of pH, the presence of salts at high concentrations, or the sulfonation of apo-A-I (41). Valuable information on the surface organization of the lipoprotein molecules can be obtained by extending the present study with a variety of polymammon molecules differing in their charge characteristics.

Although the dextran sulfate-LDL interaction occurs in the absence of divalent metal ions through electrostatic interactions between the positive charges of the LDL basic amino

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**Dextran Sulfate-Lipoprotein Interaction**

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acid residues and the negative charges of dextran sulfate (13), the addition of Ca\(^{2+}\) seems to cause the participation of LDL phospholipids in the interaction. The extent of this participation appeared to become greater with increases in the Ca\(^{2+}\) concentration as evidenced by the upward shift and broadening of the equivalence weight ratios. These changes were indicative of the involvement of more sulfate groups of dextran sulfate in the interaction with the zwitterionic polar heads of the LDL phospholipids by the mechanism we previously proposed from the study of the interaction of dextran sulfate with lecithin vesicles and lyssolecithin micelles (15). We noted that, although the insoluble lecithin-calcium-dextran sulfate complex is easily dissociated upon the addition of EDTA, the LDL-calcium-dextran sulfate complex was not dissociated by EDTA alone; it was necessary to raise the pH of the medium above 10 or to increase the ionic strength by the addition of NaCl. Therefore, the dissociation apparently required not only the abolishment of the interaction between the LDL phospholipids and dextran sulfate, but also a reduction in the electrostatic interactions involving the positive charges of LDL either by deprotonation of a large portion of the ε-amino groups of the lysine residues or by an increase in the ionic strength of the medium.

It may be generalized that, in the presence of divalent metal ions, the interaction of all sulfated polysaccharides and glycosaminoglycans with LDL, VLDL, and the lipoproteins containing apo-E (44, 45) probably occurs by the joint participation of the lipoprotein phospholipids in the interaction. The joint participation of the protein and phospholipid moieties may be speculated to occur by a mechanism similar to that for the interaction, which may be cooperatively involved in the binding of the lipoproteins. Verification of the possible role of the phospholipid-receptor interaction would be important in the clarification of the binding mechanism.

REFERENCES

27. Parker, P., and Peterson, N. F. (1965) J. Lipid Res. 6, 455-460
Nature of the interaction of dextran sulfate with high and low density lipoproteins in the presence of Ca2+.
Y C Kim and T Nishida


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