Characterization of the Binding of Serum Amyloid P to Type IV Collagen*

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Serum amyloid P (SAP), a member of the evolutionarily conserved pentraxin family, is a normal component of a number of basement membranes, including glomerular and alveolar. In vitro SAP binds to a variety of proteins including fibronectin, proteoglycans, and the collagen-like region of the complement component C1q. In these studies, binding of SAP to type IV collagen, a major component of basement membrane, was examined. Purified SAP binds to human and mouse type IV collagen but not type I, II, or III collagens. Binding of SAP to type IV collagen is dependent on the presence of Ca$^{2+}$. This binding is saturable with a $K_d \sim 1.2 \times 10^{-7}$ M based on solid phase binding and $4 \times 10^{-5}$ M based on the IC$_{50}$ value from fluid phase binding data. Binding of SAP to type IV collagen was inhibited by both SAP and C-reactive protein (CRP). However, a 5-fold molar excess of CRP as compared with SAP was required to inhibit the SAP. Binding of SAP to typling IV collagen was inhibited by both collagen IV and C1q but not by phosphatidylethanolamine or bovine serum albumin. The inhibition data indicate that SAP may bind to the triple helical region of type IV collagen via a site distinct from its galactan binding site. The most likely site of SAP involvement in its interaction with type IV collagen may be the region spanning amino acid residues 108-120, which shows a great deal of sequence homology (60% strict identity) with the CRP region implicated in its binding to the collagen-like region of the C1q molecule.

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Serum amyloid P (SAP)$^1$ is a 10-subunit (two flat pentameric discs stacked face to face) M$_r \sim 230,000$ glycoprotein coded by a single gene on human and mouse chromosome 1 (1, 2). It is a member of the evolutionarily conserved pentraxin family (1). These are proteins that are made up of five identical non-covalently bound subunits arranged in a flat pentameric disc (1, 3). In vitro SAP binds to proteoglycans and fibronectin in a specific Ca$^{2+}$-dependent manner (4-6). Furthermore, SAP binds to the collagen-like region of complement component C1q (7). SAP is a normal component of a group of basement membranes including glomerular and alveolar basement membranes (8, 9). It comprises approximately 10% of the protein released from glomerular basement membrane after collagenase treatment (8). Association of SAP with glomerular basement membranes is completely disrupted or disturbed in a number of nephritides such as Alport’s syndrome (10), membranous glomerulonephritis, and membranoproliferative glomerulonephritis (11).

Basement membranes are multicomponent structures that perform a variety of functions. They are involved in maintenance of the differentiated state and basal and apical polarity of the cells as well as maintenance of the organ structure and filtration functions (12, 13). A number of factors affect the structure and function of basement membranes (e.g., the relative concentration of each component and the affinity of the interaction between components as well as their structure) (14-16). It is therefore possible that SAP, via its interaction with various components of the extracellular matrix, modifies the structure and function of the basement membranes with which it is associated. In the present study, the binding of SAP to type IV collagen was examined. The data indicate that SAP binding to type IV collagen is Ca$^{2+}$-dependent, specific and saturable with a $K_d \sim 1.2 \times 10^{-7}$ M for immobilized and a $K_d \sim 4 \times 10^{-8}$ M (based on the IC$_{50}$ value of the soluble phase binding assay) for soluble type IV collagen. Furthermore, SAP probably binds via its C1q binding region to the triple helical region of type IV collagen. The interaction of SAP with type IV collagen and other components of the basement membrane may affect the structure of the basement membranes, thereby affecting their function.

EXPERIMENTAL PROCEDURES

Materials—Human SAP and CRP were purchased from Calbiochem. Each protein gave a single band of M$_r \sim 23,000$ and 25,000 when size fractionated on SDS-polyacrylamide gel (12% polyacrylamide) under reducing conditions. Mouse SAP was purified from acute phase mouse serum (provided by R. F. Mortensen, Ohio State University) by Ca$^{2+}$-dependent affinity chromatography on a column of phosphatidylethanolamine conjugated to agarose beads (Sigma) as described previously (17) followed by anion exchange chromatography on a Mono Q column (Pharmacia Biotech Inc.). The purified protein gave a single band of M$_r \sim 23,000$ upon size fractionation on SDS-polyacrylamide gel electrophoresis (12% polyacrylamide) under reducing conditions. Human type I, II, and III collagens were obtained from Life Technologies, Inc., and Sigma. Mouse type IV collagen was from Life Technologies, Inc. and was isolated from Engelbreth-Holm-Swarm tumor cells grown subcutaneously in letharyc mice. Size fractionation on SDS-polyacrylamide gel electrophoresis (7% polyacrylamide) gave two bands of M$_r \sim 170,000$ and 190,000 and minor high molecular weight bands (M$_r$ of intact type IV collagen was estimated at \sim550,000). Type IV collagen extracted from human plasma after pepsin digestion was purchased from Sigma, and anti-human SAP and CRP antibodies were from Dako Corp. Monoclonal mouse anti-human SAP and phosphatidylethanolamine were from Sigma, and anti-mouse SAP antibody and human C1q were from Calbiochem. 125I Labeling of SAP—Purified human SAP was iodinated by mixing 0.250 mCi of $^{125}$I-labeled sodium iodide (Amersham Corp.) with 500 µg of SAP in Tris-buffered saline (TBS, 20 mM Tris, 150 mM NaCl, 10 mM EDTA) in a glass tube coated with IODO-GEN reagent (Pierce). The
reaction was allowed to proceed for 7 min at 25 °C. Unincorporated radioactivity was separated by desalting on a Microcon 50 microconcentrator (Amicon, Inc.). The remaining protein was diluted in TBS containing 1 mM EDTA. Radioactivity of the final protein preparation was 95–98% precipitable by trichloroacetic acid. The specific activity of 125I-SAP was 0.1–0.3 μCi/μg. Iodinated SAP gave a single band of M̄ = 23,000 on SDS-polyacrylamide gel electrophoresis and retained its Ca2+–dependent binding to phosphatidylethanolamine.

ELISA Binding Assay—Type IV collagen or other proteins (1 μg/well) were coated overnight at 4 °C onto microtiter plates (Corning Inc.) using carbonate buffer (45.3 mM NaHCO3 and 18.2 mM Na2CO3, pH 9.6). The plates were washed with TBS washing buffer (20 mM Tris, 150 mM NaCl, 2 mM CaCl2, 0.05% Tween 20, pH 7.45) containing 10 mg/ml blocking reagent (Boehringer Mannheim) and blocked with TBS blocking buffer (20 mM Tris, 150 mM NaCl, 2 mM CaCl2, 0.05% Tween 20, and 5 mg/ml blocking reagent, pH 7.45) containing 10 mg/ml blocking reagent. Dilutions of SAP (100 μl) in TBS dilution buffer (20 mM Tris, 150 mM NaCl, 2 mM CaCl2, 0.05% Tween 20, and 5 mg/ml blocking reagent, pH 7.45) were added to triplicate wells, and binding was allowed to proceed for 3 h at 37 °C. Wells were washed 3 times with TBS washing buffer, and plates were incubated with the appropriate dilutions of primary antibody in TBS dilution buffer for 24 h at 4 °C. Wells were then washed 3 times in TBS washing buffer, and horseradish peroxidase-conjugated antibodies (Calbiochem) were diluted (1:2000) in TBS dilution buffer, added to each well, and allowed to bind for 90 min at 25 °C. Plates were washed 3 times with TBS washing buffer, the substrate solution (10 μg/ml O-phenylenediamine dihydrochloride in 50 mM citric acid, 100 mM NaH2PO4, 0.0003% H2O2, pH 5.0) was added to each well and the color reaction was allowed to develop at 25 °C and then stopped by the addition of 9.6% H2SO4. Absorption by each well at 402 nm was determined using an ELISA plate reader (Titertek). Inhibition of SAP binding to type IV collagen was determined by allowing 25 μg/ml SAP to bind to immobilized type IV collagen in the presence of soluble type IV collagen and soluble C1q for 4 h at 37 °C. Experiments determining the Ca2+ dependence of the SAP interaction with type IV collagen were performed using the same conditions; however, the Ca2+ levels for dilution and washing buffers were adjusted before the SAP binding assay.

Solid Phase Binding Assay Using 125I-SAP—Immunol I Removawells (Dynatech Laboratories, Inc.) were coated with 60 μl of 50 μg/ml mouse type IV collagen in phosphate-buffered saline. The amount of type IV collagen bound to the wells for each experiment was determined by directly measuring the protein bound in each well of a 12-well strip coated with 60 μl of 50 μg/ml type IV collagen in phosphate-buffered saline against a known type IV collagen standard using the BCA protein quantitation assay (Pierce). Total bound collagen was determined to be approximately 780 ± 80 ng/well. These results agree with preliminary experiments that used trace amounts of 125I-collagen (20,000 cpm) to incubate with known type IV collagen standard using the BCA protein quantitation assay (Pierce). Total bound collagen was determined using an ELISA. Data represent the mean of two independent experiments.

RESULTS

Binding of SAP to Immobilized Type IV Collagen—Binding of SAP to immobilized type IV collagen was examined. Human SAP binds to mouse type IV collagen. In a non-quantitative ELISA, maximal SAP binding was observed at approximately 20 μg/ml SAP (data not shown). Binding of SAP (25 μg/ml) to immobilized type IV collagen was measured as a function of time (5 min to 15 h) at 37 and 4 °C. SAP binding to immobilized type IV collagen proceeded rapidly, reaching near maximum levels by approximately 3–4 h for samples incubated at 37 °C and by 8–9 h for samples incubated at 4 °C (Fig. 1).

To characterize the binding of SAP to type IV collagen, binding of 125I-SAP to type IV collagen was examined and subjected to Scatchard analysis. Serial dilutions of 125I-SAP (0.2–132.25 μg/ml or 0.1–57.5 pmol in 100 μl) were added to plates coated with approximately 1.3–1.5 pmol (720–830 ng/ml) of type IV collagen (Fig. 2A). Scatchard analysis of the binding data indicated a Kd = 1.2 × 10−11 M (Fig. 2B).

Binding of SAP to type IV collagen was further examined using a reversible solution phase binding assay. Binding of half-maximal saturation concentrations of SAP (17 μg/ml as determined by ELISA) was carried out for 3 h at 37 °C and by 8–9 h for samples incubated at 4 °C. Samples (100 μl) were then added to microtiter wells coated with type IV collagen. Specific binding of SAP to immobilized type IV collagen was determined, and the percent inhibition of SAP binding was calculated (Fig. 3). The IC50 of type IV collagen was shown to be approximately 20 μg/ml (4 × 10−10 M). Scatchard transformation of the data from these experiments was not linear. This may be due to the polymerization of soluble type IV collagen at neutral pH or the binding of soluble SAP-type IV collagen complexes to immobilized collagen IV since the SAP decamer possesses multiple binding sites for type IV collagen.

Since the binding experiments above used heterologous sources of protein (i.e. human SAP and mouse type IV collagen), binding of both mouse and human SAP with mouse type IV collagen was compared using ELISA binding assays. Both human and mouse SAP bind to type IV collagen (Fig. 4), and similar saturation concentrations were observed for both proteins (−20 μg/ml). Examination of the binding of human SAP to human and mouse type IV collagen (Fig. 5) as well as type I, II, and III collagens, mouse type IV collagen, and C1q (Fig. 6) indicated that SAP binds to human and mouse type IV collagen.
gens and C1q, but not type I, II, and III collagens. Furthermore, SAP does not bind to other proteins such as C1 inhibitor, BSA, and apoferritin (data not shown). The binding of SAP to human type IV collagen is lower than that of its binding to mouse type IV collagen. This may be due to proteolytic cleavage of human type IV collagen by pepsin during its extraction. The results indicate that binding of SAP to type IV collagen is saturable and of a relatively high affinity. Furthermore, these data indicate that this binding is specific for type IV collagen since SAP did not bind to other collagen molecules.

Effect of Ca$^{2+}$ on the Binding of SAP to Type IV Collagen—Binding of SAP to a variety of its ligands is dependent on the presence of Ca$^{2+}$ (4, 18, 19). In order to determine the effect of Ca$^{2+}$ concentration on the binding of SAP to immobilized type IV collagen, its binding in the presence of Ca$^{2+}$ (0.5–7 mM) and EDTA (1–10 mM) was examined in an ELISA binding assay (Fig. 7). Binding of SAP to type IV collagen was enhanced by 4–40-fold in the presence of Ca$^{2+}$ as compared with its binding to type IV collagen in the absence of Ca$^{2+}$. Enhanced binding was observed in the presence of 0.5 mM CaCl$_2$, whereas binding levels in the presence of higher calcium concentration (1–7 mM) remained at similar levels throughout the experiments. Binding levels of SAP at 5 mM Ca$^{2+}$ were more consistently lower in all experiments. However, this binding was not significantly different from other values obtained in the presence of Ca$^{2+}$ concentrations between 1 and 7 mM. As little as 1 mM EDTA diminished the binding of SAP to background levels. Binding of SAP to BSA was minimal. These data indicate that SAP binding to type IV collagen is dependent on the presence of Ca$^{2+}$ levels in the physiological range.

Inhibition of Binding of SAP to Immobilized Type IV Collagen by SAP and CRP—Human CRP is an acute phase reactant in which hepatic synthesis and plasma levels increase by up to 1000-fold in response to inflammation (20). It has a high degree of structural and amino acid sequence homology with SAP (1, 2, 21). Previous studies indicate that both SAP and CRP bind to C1q via its collagen-like region (7, 22). Binding of CRP to C1q is mediated via amino acid residues 108–120 (23). Comparison

![Graph A](image)

**Fig. 2.** Binding of 125I-SAP to immobilized type IV collagen (Co(IV)). A, plates coated with type IV collagen were washed 3 times in TBS washing buffer. Dilutions of 125I-SAP (0.1–57.5 pmol in 100 µl of TBS dilution buffer) were added to each well, and binding was allowed to proceed for 16 h at 4 °C. Samples were then removed, and wells were washed 3 times with TBS washing buffer, dried, and counted in a γ-counter. The specific binding of SAP was calculated based on the specific activity of 125I-SAP. B, Scatchard analysis of the binding data resulted in a Kd = 1.2 × 10$^{-7}$ M and an SAP to type IV collagen ratio of 1:8.8. Data represent the combined results from two independent experiments. Each data point is the mean value of duplicate samples.

![Graph B](image)

**Fig. 3.** Binding of 125I-SAP to type IV collagen (Co IV) in solution. Microtiter plates were coated with type IV collagen or BSA. 125I-SAP at 17 µg/ml was incubated with increasing amounts of type IV collagen (0–450 µg/ml) for 24 h at 4 °C. The samples were then transferred to the microtiter wells (100 µl/well), and binding of SAP to coated wells was quantitated. The percent inhibition was determined by using the following formula: (100 – (binding in the presence of an inhibitor/binding in the absence of any inhibitor)) × 100. The results represent the mean values of four independent experiments.

![Graph C](image)

**Fig. 4.** Binding of human and mouse SAP to type IV collagen. Various amounts of human (○) and mouse (●) SAP (100 µl/well) were added to wells coated with mouse type IV collagen. The extent of binding was determined using an ELISA binding assay. Data represent the mean of three independent experiments.
of CRP, mouse, and human SAP primary structures indicates that there is a high degree of amino acid sequence identity in this area (23); therefore, SAP may bind to C1q via a region similar to the CRP C1q binding region. The ability of SAP and CRP to inhibit the binding of SAP to type IV collagen was examined (Fig. 8). BSA, at concentrations of up to 750 μg/ml, could not significantly inhibit the binding of SAP to type IV collagen. Both SAP and CRP inhibited the binding of SAP to type IV collagen; however, an approximately 5-fold molar excess of CRP as compared with SAP was required for 50% inhibition of the binding of SAP to type IV collagen. The data indicate that SAP and CRP bind to the same region or to closely located regions of the type IV collagen molecule.

SAP Binding to Type IV Collagen Is Inhibited by Soluble Type IV Collagen and C1q but Not by Phosphatidylethanolamine—SAP binds to a number of substrates including C4 binding protein and β-amyloid fibrils via its galactan binding site (24, 25). Binding of the SAP analog CRP to C1q is mediated via its amino acid residues 108–120, which is distinct from the phosphorylcholine binding site of CRP (23, 26). It is possible that similar residues in SAP (108–120 region) are involved in its interaction with C1q and type IV collagen. The primary sequence of CRP in this region exhibits a high degree of amino acid identity (54–67%) with human and mouse SAP (23), and the substitutions in both SAP molecules either do not affect the binding to C1q or enhance this binding in the context of the CRP molecule (23). In order to examine the role of the galactan binding site in type IV collagen binding, binding of SAP to type IV collagen in the presence of phosphatidylethanolamine, C1q, and type IV collagen was examined. Binding of SAP to type IV
sulfates bronectin and proteoglycans such as heparan and dermatan tis (11). Previous studies have shown that SAP binds to fi-
membrulonephritis, and membranoproliferativeglomerulonephri-
basement membrane is disrupted or altered in a number of in-
including alveolar, glomerular, and sweat gland basement

collagen is inhibited by C1q and type IV collagen but not by phosphatidylethanolamine or BSA (Fig. 9). Comparison of the IC_{50} of C1q (7.5 μg/ml) with type IV collagen (40 μg/ml) indicates that an 2-fold molar excess of C1q as compared with type IV collagen is required to inhibit the binding of SAP to type IV collagen by 50%. The data indicate that the galactan binding site of SAP is not involved in its binding to type IV collagen. Binding of the SAP analog CRP to the collagen-like region of C1q is mediated via amino acid residues 108–120. This region is conserved in a number of pentraxins (23). It is therefore possible that this region is important in the interaction of SAP with type IV collagen. Studies are under way to further characterize the collagen binding sites of the SAP molecule. Furthermore, SAP most probably binds to the triple helical region of type IV collagen; however, the triple helical conformation by itself is not sufficient for binding of SAP to type IV collagen.

**DISCUSSION**

SAP is a component of a number of basement membranes including alveolar, glomerular, and sweat gland basement membranes (8, 9). The disposition of SAP in the glomerular basement membrane is disrupted or altered in a number of nephritides such as Alport’s syndrome (10), membranous glomerulonephritis, and membranoproliferative glomerulonephritis (11). Previous studies have shown that SAP binds to fibronectin and proteoglycans such as heparan and dermatan sulfates in vitro (4–6). SAP also binds to C1q via its collagen-like region in vitro (7). Based on the in vivo association of SAP with specific basement membranes, its in vitro binding to proteoglycans, and its interaction with the collagen-like region of the C1q molecule, experiments were conducted to characterize the interaction of SAP with the basement membrane-derived type IV collagen. The data indicate that SAP binding to type IV collagen is Ca^{2+}-dependent and saturable with a K_{d} = 1.2 × 10^{-7} M in the solid phase and 4 × 10^{-8} M (based on the IC_{50} value obtained in reversible soluble phase binding assay) in the soluble phase. These differences in binding may be due to different conformations assumed by immobilized versus fluid phase collagen. They may also reflect complex molecular interactions in liquid and solid phase as indicated by non-linear Scatchard data. These interactions may include binding of SAP to immobilized collagen or to soluble type IV collagen molecules that may be followed by either binding of the SAP component of the SAP-type IV collagen complex to immobilized type IV col-

**FIG. 9. Inhibition of SAP binding to immobilized type IV collagen by C1q, type IV collagen, and phosphatidylethanolamine.**

Inhibition of SAP binding to immobilized type IV collagen by BSA (○), C1q (●), type IV collagen (■), and phosphatidylethanolamine (▲) was examined. Various concentrations of each inhibitor were mixed with 2.5 μg of SAP (100 μl final volume) and incubated for 30 min at 37°C. The mixture was added to immobilized type IV collagen. The percent inhibition was determined using the formula outlined in the legend for Fig. 3. Data represent mean values from three independent experiments.

The significance of the binding of SAP to type IV collagen and other components of the basement membrane is not yet understood. The ability of SAP to bind to type IV collagen as well as other ECM components suggests that it may facilitate the formation of the matrix or stabilize this matrix by bridging its components. Type IV collagen binds to a variety of other molecules via its triple helical region (30). Previous studies indicate that type IV collagen polymerizes to form a lattice via several interactions. These include head-to-head interactions of non-collagenous domains with one another (dimerization) as well as interaction of the amino-terminal 7 S domains (tetramerization) (27). Self-assembly of type IV collagen can also proceed laterally via its triple helical regions or via binding of the non-collagenous domain to the triple helical region of another molecule to form multimers (27, 29). Studies examining the interaction of laminin and heparin with type IV collagen demonstrated that both molecules show significant binding to the triple helical region of type IV collagen (28, 30). Since SAP binding to type IV collagen is most likely via type IV collagen’s triple helical region, it may modify the assembly of type IV collagen or its interaction with proteoglycans and laminin and alter the structure of the basement membrane and its function.

Crystallographic studies of the SAP molecule indicate that it is a very compact and highly structured protein (31, 32). Probably due to its highly ordered and compact structure, the SAP molecule is resistant to proteolysis (31, 32). Previous studies have shown that SAP binds to a number of proteins and protects them from proteolytic digestion (25). For example, SAP binds to Alzheimer’s β-amylloid peptide and amyloid A fibrils and protects them from proteolysis by enzymes such as trypsin, chymotrypsin, and Pronase (25). These observations suggest that SAP may act by binding to components of the ECM and protect them from digestion by extracellular matrix proteases and by altering the turnover rate and the structure of the basement membrane, thereby affecting its function.

Although the role of SAP in the structure and function of the basement membranes with which it is associated is not clear, it has been shown that in a variety of nephritides its association with the glomerular basement membrane is altered. It is possible that SAP via its interaction with type IV collagen and other ECM components may alter the assembly and function of the basement membrane. The absence of SAP or its altered disposition in the basement membrane, probably as a secondary defect to the initial injury, could lead to further changes in basement membrane assembly or turnover. This may partially account for basement membrane abnormalities observed in certain nephritides. The role of SAP in basement membrane structure and function is not clear; however, previous studies and the data presented here indicate that further studies of the effect of SAP on the assembly of ECM components and of the...
ability of SAP to protect ECM components against extracellular matrix proteases may be important and may help to clarify the biological significance of SAP and its potential role as a structural protein.

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Additions and Corrections

Vol. 271 (1996) 817–823

Interaction of cardiotonic thiadiazinone derivatives with cardiac troponin C.
Bo-Sheng Pan and Robert G. Johnson, Jr.

Page 818, Fig. 1: The chemical structure of EMD 57439, the (−)-enantiomer, was correct, while that of EMD 57033, the (+)-enantiomer, was in error. The correct structure is given below. We thank Dr. Norbert Beier for alerting us to the error.

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Page 14901, right-hand column, lines 14–16: This sentence should read: Furthermore, the triple helical region motif (Gly-aa-aa) is not the only requirement for this binding since SAP does not bind to collagens I, II, and III.

We suggest that subscribers photocopy these corrections and insert the photocopies at the appropriate places where the article to be corrected originally appeared. Authors are urged to introduce these corrections into any reprints they distribute. Secondary (abstract) services are urged to carry notice of these corrections as prominently as they carried the original abstracts.