Mink Serum Amyloid A Protein

EXPRESSION AND PRIMARY STRUCTURE BASED ON cDNA SEQUENCES

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The nucleotide sequences of two mink serum amyloid A (SAA) cDNA clones have been analyzed. One (SAA1) 776 base pairs long and the other (SAA2) 552 base pairs long. Significant differences were discovered when derived amino acid sequences were compared with data for apoSAA isolated from high density lipoprotein. Previous studies of mink protein SAA and amyloid protein A (AA) suggest that only one SAA isotype is amyloidogenic. The cDNA clone for SAA2 defines the "amyloid prone" isotype while SAA1 is found only in serum. Mink SAA1 has alanine in position 10, isoleucine in positions 24, 87, and 71, lysine in position 27, and proline in position 105. Residue 10 in mink SAA2 is valine while arginine and asparagine are at positions 24 and 27, respectively, all characteristics of protein AA isolated from mink amyloid fibrils. Mink SAA2 also has valine in position 67, phenylalanine in position 71, and amino acid 105 is serine. It remains unknown why these six amino acid substitutions render SAA2 more amyloidogenic than SAA1. Eighteen hours after lipopolysaccharide stimulation, mink SAA mRNA is abundant in liver with relatively minor accumulations in brain and lung. Genes encoding both SAA isotypes are expressed in all three organs while no SAA mRNA was detectable in amyloid prone organs, including spleen and intestine, indicating that deposition of AA from locally synthesized SAA is unlikely. A third mRNA species (2.2 kilobases) was identified and hybridizes with cDNA probes for mink SAA1 and SAA2. In addition to a major primary translation product (molecular mass 14,400 Da) an additional product with molecular mass 28,000 Da was immuno-precipitable.

Serum amyloid A (SAA) was first recognized in serum

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The nucleotide sequence(s) reported in this paper has been submitted to the GenBank™/EMBL Data Bank with accession number(s) J05445.

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The abbreviations used are: SAA, serum amyloid A protein; HDL, high density lipoprotein; AA, amyloid protein A; LPS, lipopolysaccharide; SDS, sodium dodecyl sulfate; T-1 fragment, first peptide fragment obtained after cleavage with trypsin; HNPN-1 fragment, first peptide fragment obtained after cleavage with 3-bromo-3-methyl-2-(2-nitrophenylthio)-H-iodole; kb, kilobase pair(s).

because of its cross-reactivity with antisera to the amyloid AA protein isolated from deposits of secondary amyloid (1, 2), and a precursor-product relationship between these two proteins has been established (3). SAA is a sensitive acute phase protein (4) found in circulation bound to HDL (apo-SAA) (5). Although the concentration during acute inflammation can reach 1 g/liter and the amount of SAA in the HDL fraction can reach 40% or more of total apoproteins (6), its function remains unclear. Interleukin-1, interleukin-6, and tumor necrosing factor released from activated macrophages during inflammation stimulate hepatic SAA production (7-9). Administration of LPS to mice produces a 2000-fold increase in the level of hepatic SAA mRNA (10) indicating that the regulation of SAA gene expression occurs at a pretranslational level. Recent studies indicate that SAA gene transcription increases during inflammation and is probably due to de novo initiation of RNA polymerase II activity rather than enhanced elongation of nascent SAA primary transcripts (10).

Heterogeneity of amino acid sequences in SAA proteins may confer properties which result in deposition of some SAA isotypes as AA protein in amyloidosis. Only SAA2 has been demonstrated in murine amyloidosis suggesting that various murine SAA isotypes may have different amyloidogenic potential, and the structure and expression of SAA genes have been most extensively studied in the mouse in which SAA is encoded by three non-allelic genes and a pseudogene (11, 12). The amino acid sequences derived from murine SAA1 and SAA2 genes show close homology to two murine SAA proteins (13). A polypeptide product corresponding to the open reading frame of the murine SAA3 gene has not been identified despite the fact that this gene contributes 30% of the SAA mRNA transcript pool (10).

In man, several SAA genes have been characterized by nucleotide sequence analysis (14-16), but, in contrast to murine models, no obvious amyloidogenic human SAA sequences have been detected (17, 18). However, in patients with amyloidosis and familial Mediterranean fever, the AA protein consists of 76 amino acids and has threonine at residue 69 (19), whereas all AA proteins characterized by sequence analysis have phenylalanine in this position. The latter is also the case with SAA from two single human individuals (18) as well as a pool of human sera (17). This phenylalanine/threonine substitution in familial Mediterranean fever amyloid arises from a complete codon substitution, as demonstrated by the structure of a corresponding human gene (16), and may be important in defining the amyloidogenicity of the altered SAA in familial Mediterranean fever. Of interest also is that the amino acid sequence deduced from the structure of one of the human SAA genes differs from any human AA protein so far studied (14). This could be another example of a non-amyloidogenic SAA gene.

Mink are susceptible to the development of AA amyloidosis,
and the observation that only valine occurs in amino acid position 10 of mink AA (20) while mink apoSAA contains isoleucine or valine at that position (21) has supported the postulate that certain SAA isotypes may be amyloidogenic. Several other amino acid positions in mink SAA demonstrate heterogeneity, but the amino acid sequence of the amyloidogenic isotype has not been known for this animal. As expression of SAA genes has been described in tissues outside the liver in other species (22, 23), it was also of importance to look for extrahepatic expression of amyloid prone SAA.

**EXPERIMENTAL PROCEDURES**

**Materials—**LPS was purchased from Difco. Molecular biological enzymes and Freund’s adjuvant were obtained from New England Biolabs and Boehringer Mannheim. The cell-free translation system was purchased from Promega Biotech. The cDNA synthesis kit was supplied by Bethesda Research Laboratories. λ ZAPII arms and Gigapack Gold packaging kits were purchased from Stratagene. Sequenase from United States Biochemical Corp. was used for nucleotide sequence reactions, and oligonucleotides were synthesized using an Applied Biosystems 360B synthesizer.

**Induction of SAA mRNA—**Two mink (Mustela vison) were injected subcutaneously with 1 mg/kg LPS (Escherichia coli 026:B6), 0.05 M sodium citrate (pH 7.0), 0.1 M β-mercaptoethanol, 0.5% Sarkosyl, and centrifugation over a 5.7 M CsCl cushion in 0.1 M EDTA (24). RNA pellets were washed in 70% ethanol, resuspended in water, and precipitated. The polyadenylated (poly(A+)) fraction was purified by precipitation. The polyadenylated (poly(A+)) fraction was purified by agarose-formaldehyde electrophoresis and transferred to nylon membranes (26). The resulting RNA blots were hybridized with either mink SAA cDNA inserts or sequence-specific oligonucleotides. 18mer oligonucleotides were 5’ end-labeled using [γ-32P]ATP and T4 polynucleotide kinase (27), and cDNA clones were radiolabeled by nick translation with [α-32P]dCTP or as single-stranded DNA probes using [α-32P]dCTP or as single-stranded RNA probes using T3 or T7 RNA polymerases and [α-32P]CTP. Hybridization with nick-translation labeled probes was performed at 42 °C overnight in 50% formamide, 5 X Denhardt’s solution (28), 0.05 M sodium phosphate (pH 6.5), 0.75 M sodium chloride, 5 mM EDTA, 250 μg/ml denatured salmon sperm DNA, and 500 μg/ml tRNA. Single-stranded RNA probes were hybridized in 50% formamide, 5 X SSC, 0.05 M Tris-HCl (pH 7.5), 0.1% sodium pyrophosphate, 1% SDS, 0.02% polyvinylpyrrolidone (M, 360,000), 0.2% Ficoll (M, 400,000), 5 mM EDTA, and 150 μg/ml salmon sperm at 65 °C overnight. Blots were washed at 65 °C in 0.2 X SSC and 0.1% SDS. Oligonucleotide probes were hybridized in 0.2% Denhardt’s solution, 0.05 M Tris-HCl (pH 7.5), 1 M NaCl, 0.1% sodium pyrophosphate, and 1% SDS. Blots were exposed to Kodak XAR-5 film at -70 °C.

**Isolation and Analysis of mRNA—**Total cellular RNA from organs was prepared by extraction in 4 M guanidinium isothiocyanate, 5 mM sodium citrate, 0.1 M β-mercaptoethanol, 0.5% Sarkosyl, and centrifugation over a 5.7 M CsCl cushion in 0.1 M EDTA (24). RNA pellets were washed in 70% ethanol, resuspended in water, and precipitated. The polyadenylated (poly(A+)) fraction was purified by precipitation. The polyadenylated (poly(A+)) fraction was purified by agarose-formaldehyde electrophoresis and transferred to nylon membranes (26). The resulting RNA blots were hybridized with either mink SAA cDNA inserts or sequence-specific oligonucleotides. 18mer oligonucleotides were 5’ end-labeled using [γ-32P]ATP and T4 polynucleotide kinase (27), and cDNA clones were radiolabeled by nick translation with [α-32P]dCTP or as single-stranded RNA probes using T3 or T7 RNA polymerases and [α-32P]CTP. Hybridization with nick-translation labeled probes was performed at 42 °C overnight in 50% formamide, 5 X Denhardt’s solution (28), 0.05 M sodium phosphate (pH 6.5), 0.75 M sodium chloride, 5 mM EDTA, 250 μg/ml denatured salmon sperm DNA, and 500 μg/ml tRNA. Single-stranded RNA probes were hybridized in 50% formamide, 5 X SSC, 0.05 M Tris-HCl (pH 7.5), 0.1% sodium pyrophosphate, 1% SDS, 0.02% polyvinylpyrrolidone (M, 360,000), 0.2% Ficoll (M, 400,000), 5 mM EDTA, and 150 μg/ml salmon sperm at 65 °C overnight. Blots were washed at 65 °C in 0.2 X SSC and 0.1% SDS. Oligonucleotide probes were hybridized in 0.2% Denhardt’s solution, 0.05 M Tris-HCl (pH 7.5), 1 M NaCl, 0.1% sodium pyrophosphate, and 1% SDS. Blots were exposed to Kodak XAR-5 film at -70 °C.

**Antiserum to Mink SAA—**Protein SAA was isolated from mink serum after stimulation with LPS as described (29). Purified SAA (0.5 mg) in complete Freund’s adjuvant was injected intradermally in New Zealand White rabbits, and booster injections (0.25 mg of SAA (0.5 mg) in complete Freund’s adjuvant) were administered every 2 weeks. After phage elution, plaques (4.4 X 104) were screened with a Syrian hamster SAA cDNA insert, phSAA (23), and radiolabeled by nick translation as above. Positive hybridization signals were visualized by autoradiography. Selected clones were plaque-purified, and in vivo excision of PBS SK (-) phagemids was achieved by the addition of R408 helper phage. Plasmid DNA was isolated (34, 35) prior to analysis by restriction endonuclease cleavage and nucleotide sequencing.

**Analysis of Mink SAA Clones—**Nucleotide sequencing was performed using the dideoxy-chain termination method (36). DNA sequences were analyzed using the GeneWorks computer program (version 6.0). Prediction of the helical content of the derived amino acid sequences was done with Chou-Fasman analysis (37), and hydropathy was predicted by the method of Kyte and Doolittle (38).

**RESULTS**

**Identification and Characterization of Mink SAA-specific cDNA Clones—**When 4.4 X 105 recombinants from the mink hepatic cDNA library were screened with the cross-reacting hamster SAA cDNA probe, 152 positive clones were identified. After phage elution, 15 clones were selected for further analysis. Mapping of inserts with 10 restriction endonucleases indicated that the clones could be separated into two groups, each containing inserts of different sizes. The largest clone in each group, 776 base pairs (pmiSAA1) and 552 base pairs (pmiSAA2), respectively, were chosen for sequence analysis. 90% of pmiSAA1 and 100% of pmiSAA2 were determined from both strands. Sequence strategy and relevant restriction map data in the coding region are shown in Fig. 1. The nucleotide sequence and deduced amino acid sequences for mink SAA1 and SAA2 are shown in Fig. 2. The nomenclature for mink SAA1 and SAA2 was adopted according to the convention established in the murine system where SAA2 is amyloidogenic and SAA1 is not. For comparison, the published polypeptide sequence for mink SAA and AA (21, 20) is also included (Fig. 2). The longest clone, pmiSAA1, has a 5’-untranslated region of 28 nucleotides, a signal peptide region corresponding to 19 amino acids, a coding sequence for a...
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Expression of SAA in different tissues—Hybridization of mink hepatic RNA with the cross-reacting hamster SAA probe showed no detectable SAA mRNA in the sample from the control animal, while in both LPS-stimulated animals and apo-SAA is reported as glutamine (20, 21), the 10 cDNA clones studied in this region predict serine (codon TCT) in this position. The finding of isoleucine instead of phenylalanine at residue 6 could not be explained.

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FIG. 3. Northern blot of RNA from mink liver hybridized with a hamster SAA probe. Molecular weight standards are shown in lane a; lane b is 10 μg of RNA from unstimulated and lane c from LPS-stimulated mink liver.

FIG. 4. Northern blot of RNA from LPS-stimulated mink hybridized with a RNA probe transcribed from pmuSAA1. Lane a, 20 μg of liver RNA; lane b, 20 μg of brain RNA; lane c, 20 μg of lung RNA. Lane a, exposed for 15 min; lanes b and c, exposed for 24 h.

FIG. 5. SDS-polyacrylamide gel electrophoresis of primary translation products from mink liver mRNA immunoprecipitated with anti-mink SAA antibodies. Lane a is 100 μg and lane b is 20 μg of RNA from LPS-stimulated mink liver; lane c is 100 μg of RNA from unstimulated mink liver.
2) (21). We have studied 10 mink SAA cDNA clones from which information about amino acid residue 10, important for amyloidogenicity, could be derived. No codon predicting isoleucine at position 10 was identified, while four clones representing the "amyloid-prone" SAA2 (valine at position 10) were identified, and six clones encoded alanine at this position of SAA1. However, an isoleucine codon was found at amino acid residue 6 of all cDNA clones. Upon scrutiny of the amino acid composition of the BNPS-2 and the T-1 fragment isolated from apoSAA (21), the prediction from the cDNA clones of alanine and valine at position 10 and isoleucine at position 6 suggests a sequence which is in better agreement with the actual amino acid compositions of these peptide fragments. This is, however, not the case for protein AA, where only one isoleucine is found in the total sequence of AA1 and none in AA2 (20). The pmiSAA1 and pmiSAA2 nucleotide sequences both encode polypeptides eight amino acids longer than the published apoSAA, and the derived amino acid sequences of mink SAA correspond in that respect to SAA from horse (30) and sheep (40), and to protein AA from duck (41).

The derivation of isoleucine at positions 67 and 71 from one cDNA clone set and of valine and phenylalanine at these respective positions in the other group has defined SAA1 and the "amyloid-prone" SAA2 primary structure for mink. The differences between SAA1 and SAA2 for positions 24, 27, and 105 were not detected in apoSAA (Fig. 2) where no heterogeneity has been documented and may represent allelic variation. The six amino acid substitutions, including three in the NH2-terminal portion of the SAA molecule found in protein AA, indicate that SAA2 is the amyloidogenic isotype. Whether amyloidogenicity is related to altered binding to HDL, changed susceptibility to proteolysis, or different binding affinity to the tissue amyloid matrix is not clear. Prediction of secondary structure based upon the amino acid sequence of mink SAA1 and SAA2 did not reveal any major differences in helical content or hydrophobicity of these two polypeptides. Similar observations have been reported for murine SAA1 and SAA2, but circular dichroism studies indicate that significant structural dissimilarities between these two proteins occur when they are interacting with heparan sulfate in the presence of calcium (42). Although 18 h after LPS stimulation the hepatic mRNA species for mink SAA1 and SAA2 are equally abundant, SAA2 accounts for only 20% of apoSAA isolated from mink serum 36 h after LPS stimulation (21). This may be due to selective SAA2 removed from the circulation, as has been shown in the murine model (43), or may result from differential SAA isotype binding to HDL or differences in translation rate or post-translational modifications.

The discrepancies between mink SAA sequences obtained from protein analyses and those derived from cDNA sequences may reflect expression of a third SAA gene contributing to most of the SAA complexed to HDL 36 h after stimulation. Such a gene may direct biosynthesis of another "amyloid-prone" SAA isotype containing isoleucine at position 10 but was not evident in detailed studies of 10 cDNA clones. Anti-SAA antiserum immunoprecipitates a 28,000-Da primary translation product to SAA amyloidogenicity remains to be determined.

Our studies of expression of mink SAA1 and SAA2 18 h after stimulation by LPS show that liver is the site of most abundant SAA mRNA production. Minor amounts of SAA mRNA for both SAA1 and SAA2 were detected in lung and brain. The relative accumulation of SAA mRNA at extrahematopoietic sites compared with that in the liver was less in the mink than reported for the mouse (22) and hamster (23) although mink are equally susceptible to the development of amyloidosis. Mink SAA mRNA was not identified in amyloid-prone organs such as spleen and intestine. These data support the observation that amyloid deposition does not arise from locally produced and degraded amyloid-prone SAA isotypes but rather from heptatically derived SAA.

Amyloidosis is an important complication to chronic inflammation. These studies of SAA in mink have detailed the amino acid sequences of SAA isotypes which are differentially involved in amyloid deposition. In addition, these studies demonstrate a SAA-specific mRNA species which is considerably larger than those previously described. These observations suggest the importance of further studies of SAA and amyloidosis in mink.

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