The cDNA Sequence of Mouse Pgp-1 and Homology to Human CD44
Cell Surface Antigen and Proteoglycan Core/Link Proteins*

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We describe the isolation and sequencing of a cDNA encoding mouse Pgp-1. An oligonucleotide probe corresponding to the NH2-terminal sequence of the purified protein was synthesized by the polymerase chain reaction and used to screen a mouse macrophage Agt11 library. A cDNA clone with an insert of 1.2 kilobases was selected and sequenced. In Northern blot analysis, only cells expressing Pgp-1 contained mRNA species that hybridized with this Pgp-1 cDNA. The nucleotide sequence of the cDNA has a single open reading frame that yields a protein-coding sequence of 1076 base pairs followed by a 132-base pair 3'-untranslated sequence that includes a putative polyadenylation signal but no poly(A) tail. The translated sequence comprises a 13-amino acid signal peptide followed by a polypeptide core of 345 residues corresponding to an Mr of 37,800. Portions of the deduced amino acid sequence were identical to those obtained by amino acid sequence analysis from the purified glycoprotein, confirming that the cDNA encodes Pgp-1. The predicted structure of Pgp-1 includes an NH2-terminal extracellular domain (residues 14-265), a transmembrane domain (residues 266-286), and a cytoplasmic tail (residues 287-358). Portions of the mouse Pgp-1 sequence are highly similar to that of the human CD44 cell surface glycoprotein implicated in cell adhesion. The protein also shows sequence similarity to the proteoglycan tandem repeat sequences found in cartilage link protein and cartilage proteoglycan core protein which are thought to be involved in binding to hyaluronic acid.

Pgp-1 is a plasma membrane glycoprotein of 85-95 kDa

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which was first characterized as an abundant cell surface antigen of NIH 3T3 cells (Hughes and August, 1981). The protein is expressed at a high density (approximately 106 molecules/cell) on macrophages, peripheral blood mononuclear cells, and granulocyte precursors in the bone marrow and at lower densities on spleen cells and in extracts of solid tissues (Hughes et al., 1983; Trowbridge et al., 1982). In the mouse, Pgp-1 is recognized as a differentiation alloantigen (Ly-24), and the gene controlling the expression of the molecule has been mapped to chromosome 2 by means of allelic determinants (Hughes et al., 1981; Colombatti et al., 1982; Lesley and Trowbridge, 1982). Early fetal thymocytes express the antigen, but this fraction of Pgp-1 cells declines at birth. In adult mice, Pgp-1 is found on only a minor population of thymic cells, predominantly CD4/CD8 progenitor thymocytes (Trowbridge et al., 1982, 1985; Lesley et al., 1985; Gause et al., 1987). Pgp-1 is expressed by a small fraction of peripheral T cells after antigenic or mitogenic stimulation and appears to define a subpopulation of mature T cells as primed or memory T cells (Budd et al., 1987; Lynch et al., 1987; Gause et al., 1988a, 1988b). Studies of the redistribution of Pgp-1 during cell movement and the lateral diffusion of the molecule (Jacobson et al., 1984a, 1984b) have suggested an association of the glycoprotein with the cell cytoskeleton.

The human homolog of Pgp-1 has been identified as a serine phosphoglycoprotein (Isacke et al., 1986) in studies using a cross-reactive antibody (IM7) originally raised against the mouse glycoprotein (Trowbridge et al., 1982). Other studies (Omary et al., 1988) have demonstrated that human Pgp-1 corresponds antigenically to several independently described cell surface glycoproteins: the p85 antigen of chronic lymphocytic leukemia cells identified by monoclonal antibodies 50B4 and 50E0 (Letarte et al., 1986, Letarte et al., 1985); a T lymphocyte, monocyte, granulocyte, and brain antigen (mAb AlG3) (Haynes et al., 1983); and the Ina and Inb blood group antigen (mAb A3D8) regulated by the Lutheran inhibitor In (Lu) gene (Telen et al., 1984, 1986) and ECMRIII (Wayner et al., 1980) designated CD44 in the nomenclature of the Third International Workshop on Leukocyte Antigens (Cobbold, 1987); a medullary thymocyte antigen (mAb A1G3) (Haynes et al., 1983); and the Ina and Inb blood group antigen (mAb A3DB) regulated by the Lutheran inhibitor In (Lu) gene (Telen et al., 1983, 1984). Antibodies F10-44-2 and A1G3 have been used to map the gene for these antigens to the short arm of human chromosome 11 (Francke et al., 1983; Goodfellow et al., 1982). Recently, two other antigens implicated in cell adhesion phenomena, gp90 Hermes glycoprotein (Jalkanen et al., 1987) and ECMRIII (Wayner and Carter, 1987), have also been shown to be homologous to

1 The abbreviations used are: mAb, monoclonal antibody; PCR, polymerase chain reaction; SDS, sodium dodecyl sulfate; HPLC, high performance liquid chromatography; bp, base pair(s).
members of the CD44 (Fgp-1) group (Picker et al., 1989; Gallatin et al., 1989).

In this study, we have used amino acid sequence data obtained from purified Pgp-1 and an oligonucleotide probe synthesized by the polymerase chain reaction (PCR) to isolate CD44 corresponding to the mouse Pgp-1 glycoprotein. The deduced amino acid sequence of the mouse cDNA is highly similar to the deduced sequences of CD44 cDNA clones isolated from human cDNA libraries and also shows sequence similarity to proteoglycan core/link proteins (Stamenkovic et al., 1989; Goldstein et al., 1989). These studies confirm the relationship between CD44 and Pgp-1, which had been indicated previously by antigenic cross-reactivity studies, and elucidate the amino acid sequence relationships that characterize these proteins.

**EXPERIMENTAL PROCEDURES**

**Immunofluorometry Purification of Pgp-1—**Pgp-1 was purified by mAb affinity chromatography (Hughes et al., 1983) from the lymph nodes, spleens, and livers of MRL lpr/lpr mice. The published method was modified as follows. Tissue membranes were washed with 10 mM Tris-HCl (pH 7.5) containing 1 mM EDTA before detergent extraction with Triton X-100. Immediately prior to elution of the protein from the antibody column, the column was washed with 50 mM Tris (pH 7.6) containing 0.6% octyl glucoside (Calbi ochem). The protein was eluted with 100 mM diethyiamine (pH 11.5) containing 0.5% octyl glucoside into a neutralizing solution of 0.1 M sodium phosphate buffer containing 0.5% octyl glucoside.

**Amino Acid Sequence Analysis—**The NH2-terminal sequence of pure Pgp-1 (420 pmol) was determined in duplicate by use of an automated gas-phase sequenator (Applied Biosystems) (Hewick et al., 1981; Hunkapiller and Hood, 1983). Tryptic peptides of the affinity-purified glycoprotein were also sequenced. The peptides were prepared as follows. 50 wg of affinity-purified Pgp-1 was denatured with 350 AM NaCl and 0.5% octyl glucoside. The column was washed containing 0.5% octyl glucoside into a neutralizing solution of 0.1 M sodium phosphate buffer containing 0.5% octyl glucoside. The protein was eluted with 100 mM diethyiamine (pH 11.5) containing 0.5% octyl glucoside into a neutralizing solution of 0.1 M sodium phosphate buffer containing 0.5% octyl glucoside. The column was washed with an equal volume of equilibrating buffer. The run-through fractions and wash were pooled and concentrated to 0.33 mg/ml by evaporation. The reaction was run for 40 cycles; each cycle consisted of denaturation for 2 min at 94 °C, annealing for 2 min at 52 °C, and extension for 1 min at 72 °C. The amplified product was prepared with primers that were end labeled with [y-32P]dATP (New England BioLabs) and then used directly in the PCR as described. Single-stranded DNA was obtained from deletion subclones and sequenced using fluoresceinated M13 primers (Taq sequencing kit, Promega Biotech). These samples were electrophoresed and analyzed using the model 370A DNA sequencer (Applied Biosystems).

**RESULTS**

Pgp-1 was purified by antibody affinity chromatography (Hughes et al., 1983) from lymphoid tissue obtained from MRL lpr/lpr mice. These mice develop a massive lymphohyperplasia due to the expansion of an unusual T cell subset that expresses a characteristic phenotype including high levels of Pgp-1 (Davidson et al., 1986). The purified fraction contained a single protein corresponding to the 85-kDa Pgp-1 with >90% purity as assessed by silver staining or by autoradiography of the purified protein labeled with 125I. The NH2-terminal amino acid sequence of the purified protein extending to residue 26 (Fig. 1A) (corresponding to residues 14–39 of the predicted sequence (see Fig. 2)) was determined by automatic Edman degradation and analysis of the phenylthiohydantoin amino acid products. The unknown amino acid (X) at residue 2 (Fig. 1A) was later identified as cysteine from the nucleotide sequence of the isolated cDNA (residue 25 of the predicted sequence (Fig. 2)). Sequence analysis was also performed on six peptides obtained by reverse-phase HPLC of the products of trypsin digestion.

The strategy for cloning the mouse Pgp-1 gene utilized the PCR to synthesize a complementary oligonucleotide probe from a set of oligonucleotide primers corresponding to regions within the NH2-terminal amino acid sequence (Fig. 1B). An oligonucleotide for use as an internal probe was chemically synthesized from a region of sequence between those used for...
FIG. 1. Sequence of the PCR primers and the internal probe and their relationship to the NH2-terminal amino acid sequence of Pgp-1. A, the NH2-terminal sequence of murine Pgp-1 showing the regions of sequence from which sense and antisense PCR primers and the internal probe were synthesized. The NH2-terminal sequence corresponds to residues 14-39 of the predicted sequence (see Fig. 2). Residue 12 is shown by the predicted sequence to be cysteine (residue 25, see Fig. 2). B, sequences of the degenerate oligonucleotides synthesized for use as primers in PCR and as an internal probe are shown. Restriction endonuclease sites were incorporated into the primers as shown.

The primers. Each oligonucleotide contained the full complement of possible sequences corresponding to individual codons, except in the case of particular codons that were rare as judged by codon usage tables (Maruyama et al., 1987). The primer oligonucleotides also included a restriction endonuclease site, EcoRI, in the sense primer and PstI in the antisense primer. A murine macrophage cDNA library was used as the template for the reaction. On the basis of the 11-amino acid distance between the target amino acid sequences and the primer. A murine macrophage cDNA library was used as the template for the reaction. On the basis of the 11-amino acid sequence (residues 266-286); and 5) a hydrophilic cytoplasmic region (residues 14-147); 3) a predominantly hydrophobic in nature but is otherwise unremarkable. The signal sequence (CATAAA, 1194-1200). The deduced polypeptide product of 345 amino acids has a calculated M, of 37,800. The predicted primary structure of cDNA of Mouse Pgp-1...
The NH2-terminal portion of the protein contains all five of the potential sites of NH2-terminal glycosylation (Asn-X-Ser/Thr) at residues 22, 54, 97, 108, and 118 and all 6 of the extracellular cysteine residues. A portion of the extracellular Thr) at residues 22, 54, 97, 108, and 118 and all 6 of the extracellular cysteine residues. A portion of the extracellular region (residues 14-147), the GRand Average hydropathy (GRAVY) score = 4.1. A schematic diagram of the protein drawn below the plot shows the position of the signal peptide (+), transmembrane region (W), the putative sites of N-linked glycosylation (*), and the approximate positions of the two extracellular domains: the distal hydrophobic NH2-terminal region (residues 14-147), and the proximal hydrophilic region (residues 148-265, ■). The hydrophilic cytoplasmic tail (residues 287-358) is shown. The limits of the transmembrane region were confirmed using the method of Rao and Argos (1986).

The hydropathy plot was generated using the Soap program (Kyte and Doolittle, 1982) and computed using an interval of 9 amino acids. The GRand Average hydropathy (GRAVY) score = 4.1. A schematic diagram of the protein drawn below the plot shows the position of the signal peptide (+), transmembrane region (W), the putative sites of N-linked glycosylation (*), and the approximate positions of the two extracellular domains: the distal hydrophobic NH2-terminal region (residues 14-147), and the proximal hydrophilic region (residues 148-265, ■). The hydrophilic cytoplasmic tail (residues 287-358) is also shown. The limits of the transmembrane region were confirmed using the method of Rao and Argos (1986).

The NH2-terminal portion of the protein contains all five of the potential sites of NH2-terminal glycosylation (Asn-X-Ser/Thr) at residues 22, 54, 97, 108, and 118 and all 6 of the extracellular cysteine residues. A portion of the extracellular domain (residues 150-265) is more hydrophilic than the NH2 region (residues 14-149) and contains possible sites of O-glycan, which are characteristically found in hydrophilic domains of low helix structure (Bourdon et al., 1987) (residues 178, 249, and 255). A hydrophobic stretch of 21 residues (266-286) comprises the only transmembrane region predicted by the method of Rao and Argos (1986). This region contains 1 cysteine residue (283). A hydrophilic tail of 72 residues presumably extends into the cytoplasm. It contains 6 serine residues, of which 2 (residues 288 and 313) may be phosphorylated. The 8th and 9th cysteine residues predicted by the cDNA sequence are found in the intracellular domain at residues 292 and 340.

As shown in Fig. 5, the mouse Pgp-1 molecule is highly similar to the recently described human CD44/Hermes protein (Stamenkovic et al., 1989; Goldstein et al., 1989) except that the human sequences lack the NH2-terminal sequence NH2-His-Pro-His-Gln. The human and mouse sequences (using the longer human sequence reported by Stamenkovic et al. (1989)) are otherwise identical over 72% of their residues. Eight cysteine residues found in the human sequence are conserved in the mouse sequence. Five of the six potential sites of N-linked glycosylation and three of the four possible chondroitin sulfate addition sites are also conserved. A region of reduced homology is the extracellular region between residues 155 and 265. The mouse sequence encodes an additional cysteine residue in the cytoplasmic domain which is not present in the human sequence.

The Pgp-1 sequence is also similar in the NH2-terminal region (residues 21-117) to proteoglycan core/link proteins. Comparisons with rat cartilage-specific proteoglycan core protein precursor (z = 6.7) and with rat proteoglycan link protein 2 (fragment) (z = 3.8), showed significant but not extensive (80%) homology. The z-score value (0.9) for Pgp-1 self-comparison suggested no significant internal duplication within the protein. A comparison of the sequences of Pgp-1 residues 21-117 with residues 154-254 of domain Ila of chicken link protein is shown in Fig. 6. Domain Ila is known to be involved in the binding of link protein to hyaluronic acid. It is predicted that a loop structure stabilized by two disulfide bonds between cysteines 182 and 254 and an cysteine 206 and 227 is critical to the function of the molecule (Perkins et al., 1989). The hyaluronic acid-binding regions have been defined as residues 207-226 and 242-251 using monoclonal antibodies and synthetic peptides (Goetinck et al., 1987). Furthermore, a number of studies have suggested that the arginine and lysine residues within these regions have an important function in link protein binding to hyaluronic acid, which appears to be a largely ionic interaction (Hardingham et al., 1976; Lyon, 1986; Goldstein et al., 1989). Although there is a similarity in the placement of a cysteine residue between Pgp-1 and the link protein, none of the arginine or lysine residues in the region.

The cDNA sequence is found in the intracellular domain at residues 292 and 340. As shown in Fig. 5, the mouse Pgp-1 molecule is highly similar to the recently described human CD44/Hermes protein (Stamenkovic et al., 1989; Goldstein et al., 1989) except that the human sequences lack the NH2-terminal sequence NH2-His-Pro-His-Gln. The human and mouse sequences (using the longer human sequence reported by Stamenkovic et al. (1989)) are otherwise identical over 72% of their residues. Eight cysteine residues found in the human sequence are conserved in the mouse sequence. Five of the six potential sites of N-linked glycosylation and three of the four possible chondroitin sulfate addition sites are also conserved. A region of reduced homology is the extracellular region between residues 155 and 265. The mouse sequence encodes an additional cysteine residue in the cytoplasmic domain which is not present in the human sequence.

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residues in the reported binding region of domain II is conserved in Pgp-1, and Pgp-1 lacks the tandem repeat of this region characteristic of link and core protein characterized previously.

**DISCUSSION**

We report here the isolation and sequencing of a cDNA encoding mouse Pgp-1 from a macrophage cDNA library. The nucleotide sequence extends from within the putative signal peptide coding region to the 3'-untranslated sequence and has a single open reading frame encoding a polypeptide of 345 amino acids. The predicted amino acid sequence is identical to the sequences of six peptides obtained by tryptic digestion of the purified glycoprotein.

The predicted mass of the protein is 37,800 Da, whereas the apparent molecular mass of the mature Pgp-1 glycoprotein is about 85 kDa. Biochemical studies of the biosynthesis and processing of the protein have shown that the polypeptide precursor synthesized in the presence of tunicamycin, an inhibitor of N-linked glycosylation, has a mass of 50 kDa and that by 30 min is "chased" into a 65-kDa form, presumably as a result of the addition of O-linked sugars. In the absence of tunicamycin, a zero time precursor of 65 kDa was chased into the mature 85-kDa form by 30 min. This difference between the 50-kDa precursor in the presence of tunicamycin and the normal 65-kDa precursor can be attributed to cotranslational addition of the predicted five asparagine-linked high mannose oligosaccharides. Maturation to the 85-kDa form is attributed to the processing of the asparagine-linked oligosaccharide to complex-type structures and the addition of O-linked oligosaccharides. The discrepancy between the predicted 38-kDa polypeptide and the observed 50-kDa precursor synthesized in tunicamycin-treated cells implies that there is further modification of the protein core or anomalous migration of the polypeptide on SDS-polyacrylamide gel electrophoresis.

This study confirms a relationship between Pgp-1 and CD44 which had been suggested previously by homology in both proteins. The transmembrane regions and cytoplasmic tails of the two proteins are 96% homologous. Such high degrees of similarity suggest that these regions may be functionally important for these molecules. The murine Pgp-1 and CD44/Hermes/ECMIII glycoproteins thus appear to be species variants of the same gene product or members of a family of closely related glycoproteins.

The mouse amino acid sequence differs from the human sequence in two regions, the NH2 terminus and a region from residue 155 to 265. The deduced NH2-terminal sequence of Pgp-1 (residues 14–17), His-Pro-His-Gln, is absent from the predicted NH2 terminus of the human cDNA clones. A mouse cDNA sequence corresponding to CD44 obtained from D. E. Butcher (Stanford University) lacks the NH2-terminal His-Pro sequence but is otherwise homologous to the Pgp-1 sequence. The deduced Pgp-1 NH2 terminus is verified by its identity to the NH2 terminus of the purified Pgp-1 protein. In other studies, we have attempted to obtain NH2-terminal sequence data from human CD44 but have found that the NH2-terminal is blocked in this purified protein.

The extra-cellular domain of mouse Pgp-1 from approximately residues 155 to 265 (Pgp-1) is the other region of lowest homology (47%) when the mouse and human sequences are compared (Fig. 5). This is particularly noteworthy since this region of the human molecule is reported to contain the epitope of the Hermes 3 monoclonal antibody (Goldstein et al., 1989) which blocks binding of lymphocytes to mucosal high endothelial venules (Jalkanen et al., 1986). It is possible that an equivalent epitope is present on the mouse protein or that these sequence differences between the mouse macrophage and human lymphocyte proteins could reflect a functional difference between the proteins. The presence of Pgp-1 in a variety of cell types in addition to lymphocytes poses the question of how a functional specificity of this molecule could be controlled. It has been suggested that this control may take the form of different post-translational modifications of CD44 or of differential interactions of CD44 with "effector" molecules such as vascular addressins or other cell surface receptors (Goldstein et al., 1989; Jalkanen et al., 1987).

The Pgp-1 sequence also has significant sequence similarity to the NH2-terminal region of proteoglycan core and link proteins that form a ternary complex with hyaluronic acid (Goetinck et al., 1987). Although it is not yet evident that these sequences reflect functional properties of Pgp-1, the kinds of interactions mediated by these proteins are not inconsistent with the proposed role of the molecule in cell adhesion as a lymphocyte homing receptor (Jalkanen et al., 1986, 1987). collagen-binding receptor (Carter and Wayner, 1988), or in homotypic cell aggregation. Moreover, there is evidence that Pgp-1 acts as a proteoglycan core protein. We have observed that immunoprecipitates with anti-Pgp-1 mAb and extracts of cells labeled with H35SO4 contain a high

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2 D. E. Warder and J. T. August, unpublished observations.

3 E. C. Butcher, personal communication.

4 J. T. August, unpublished observations.

molecular mass glycoprotein (200 kDa) in addition to an 85-kDa glycoprotein. The 200-kDa molecule is strongly $^{35}S$O$_4$ labeled but contains only a small fraction of the protein (<1% of the protein present in the 85-kDa form). The properties of the 200-kDa molecule are those of a proteoglycan containing Pgp-1 as a core protein, and it is sensitive to chondroitinase ABC, suggesting that it is substituted with chondroitin sulfate. The deduced protein sequence of Pgp-1 contains three putative sites of chondroitin sulfate substitution present in the hydrophilic extracellular region.

Another putative post-translational modification of the glycoprotein is the phosphorylation of serine residues (288 and 313) in the cytoplasmic tail. Residue 288 lies within a consensus site for the action of protein kinase C (Woodgett et al., 1986; Kishimoto et al., 1985), and residue 313 is potentially a substrate for cAMP and cGMP-dependent protein kinases (Feramisco et al., 1982; Glass and Smith, 1983; Glass et al., 1986). These predictions are consistent with observations that Pgp-1 is phosphorylated at serine residues in the cytoplasmic region (Isaacs et al., 1986; Kalamiris and Bourguignon, 1988).

Another mouse cell surface glycoprotein implicated in lymphocyte homing is the MEL-14 antigen. It was reported that the MEL-14 antigen (Lasky et al., 1989) shows no homology with immunoprecipitated by MEL-14 was removed by preclearing a 90-kDa antigen of human peripheral blood lymphocytes. It was reported that homing is the MEL-14 antigen. It was reported that immunoprecipitated by MEL-14 was removed by preclearing a 90-kDa antigen of human peripheral blood lymphocytes.

Pgp-1 is phosphorylated at serine residues in the cytoplasmic region (Low, 1986; Davis et al., 1989). Another mouse cell surface glycoprotein implicated in lymphocyte homing is the MEL-14 antigen. It was reported that the MEL-14 antigen (Lasky et al., 1989) shows no homology with immunoprecipitated by MEL-14 was removed by preclearing a 90-kDa antigen of human peripheral blood lymphocytes. It was reported that homing is the MEL-14 antigen. It was reported that immunoprecipitated by MEL-14 was removed by preclearing a 90-kDa antigen of human peripheral blood lymphocytes.
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