A Blood Group-related Polymorphism of CD44 Abolishes a Hyaluronan-binding Consensus Sequence without Preventing Hyaluronan Binding*  

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CD44 is a widely expressed integral membrane protein that acts as a receptor for hyaluronan (HA) and is proposed to be important to cell-extracellular matrix interaction. The Indian (In) blood group antigens reside on CD44, and most individuals express the Inb antigen. Homozygosity for the Ina allele occurs as a rare event and is associated with production of alloantibody to the common Inb antigen after transfusion or pregnancy.  

The present study demonstrates that a single point mutation (G252 → C) causes an Arg⁶⁶ → Pro substitution, which is responsible for the Inb/Ina polymorphism. Additional mutations were found in In(a+b-) cDNA but were not necessary to the antigenic phenotype as determined in site-directed mutagenesis studies. In studies using CD44 chimeric constructs, Arg⁶⁶ has previously been shown to be crucial for maintenance of HA-binding ability to a CD44 peptide. However, the present study demonstrates that the Arg⁶⁶ → Pro substitution does not reduce HA binding to the intact CD44 protein, which contains two proposed extracellular HA-binding motifs. Down-regulation of HA binding to In(a+b-) CD44 by anti-CD44 monoclonal antibody (mAb) ligands, however, was weakened, although all mAbs tested bound In(a+b-) and In(a-b+) CD44 equally well. Competitive inhibition studies using human anti-Inb also showed that some mAbs that inhibit HA binding to CD44 may do so by interacting with a domain separate from, but affecting the structure of, the Inb epitope.  

Investigation of the biochemical and molecular basis of human erythrocyte blood group antigens has begun to contribute to our understanding of structure/function relationships of some biologically active red cell membrane proteins (1). CD44, also previously identified as In(Lu)-related p80 (2, 3), the Hermes antigen (4, 5), Pgp-1 (6–8), and ECMRIII (9, 10), is a widely expressed integral membrane protein that bears Indian (In) blood group antigens (11, 12). CD44 has been shown capable of binding a number of extracellular matrix components, including hyaluronan (HA), fibronectin, heparan sulfate, and collagen types I and VI (10, 13). In addition, studies using monoclonal antibodies to this protein have suggested that CD44 plays a role in lymphocyte homing to mucosal lymph tissue (14), in leukocyte activation (15), and in tumor metastasis (16). CD44 also appears important to lymphopoiesis (17) and to progenitor cell-ECM interactions during erythropoiesis (18).  

A large number of CD44 isoforms have been described (16, 19–21). Nonactivated hematopoietic cells, including erythrocytes, express predominantly an 80–90-kDa “hematopoietic” isoform (CD44H) encoded by exons 1–5, 15–17, and 19 of the CD44 gene (22, 23). Exons 6–14 have been shown to be expressed in various combinations under a variety of circumstances, including leukocyte activation and malignant transformation. One of the most commonly identified CD44 isoforms, CD44E, contains exons 12, 13, and 14 (24). Exon 18 encodes an alternate, shortened cytoplasmic tail. Expression of some splice forms has been associated with increased metastatic potential or induction of an adhesive phenotype (23). CD44 isoforms can also vary in molecular weight due to differing glycosylation patterns. CD44H bears a number of consensus sites for covalent attachment of chondroitin sulfate, and a 180-kDa CD44 isoform has been shown to bear several chondroitin sulfate moieties (25).  

Several studies have attempted to identify the sites at which CD44H binds hyaluronan. Resting lymphocytes express CD44H but only bind hyaluronan poorly. Jurkat cells transduced with CD44H cDNA will bind HA when stimulated with phorbol 12-myristate 13-acetate (PMA) (26). However, PMA-treated cells transfected with CD44H cDNA did not bind HA unless first incubated with CD44 monoclonal antibodies known to enhance HA binding (26). Different monoclonal antibodies to CD44 may enhance, block, or not affect HA binding to CD44H (26, 27); the mechanisms that effect alteration of HA-binding ability have not been defined. The cytoplasmic domain of CD44H is also known to be necessary for activation of HA-binding ability (26).  

In the extracellular portion of CD44H, two proposed HA-binding domains containing positively charged amino acids (amino acids 29–46 and 150–162) have been shown to be important for maximal HA binding, using deletion and site-directed mutants (28, 29). However, the first domain is the only one that resides within the portion of CD44 homologous to cartilage link protein. Peach et al. (28) have shown that mutation of Arg⁴⁶ → Ala in a construct containing amino acids 1–220 of CD44H almost abolished HA binding altogether. Yang and colleagues (29), using different chimeric constructs containing only small CD44 peptides, showed that an Arg⁶⁶ → Gly substi-

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The study identified a specific motif, X7B, where B represents a basic amino acid (Arg or Lys), in the CD44 gene. The presence of this motif is associated with the ability of CD44 to bind hyaluronan (HA). The study used site-directed mutagenesis to create a plasmid called CD44W18, which expresses a CD44 protein with a Gly residue at position 187. This Gly residue abrogates the binding of HA.

**Materials and Methods**

**Cells and Serum**

Erythrocytes were collected and washed in phosphate-buffered saline (PBS). For radioimmunoassays, a 5% (v/v) erythrocyte suspension in PBS was assayed according to previously published methods (33, 34).

**DNA Clones and Sequencing**

The wild-type CD44 (CD44WT) cDNA clone was provided in the pCDM8 vector as a gift of Dr. Brian Seed (Bostom, MA) (37). The insert was then cloned into the pDNA3 expression vector (Invitrogen, San Diego, CA). In(a+b−) Rh and CD44 WT cDNA clones were obtained by reverse transcription-polymerase chain reaction (RT-PCR) of mRNA obtained from EBV-transformed lymphocytes using the FastTrack mRNA isolation system (Invitrogen) or standard guanidinium isothiocyanate-osium chloride isolation techniques (38). Synthesis of first-strand cDNA was achieved by the GeneAmp RT-PCR kit (Perkin-Elmer), using oligonucleotide MT2 (Table I). Amplification conditions were as follows: denaturation for 1 min at 95°C, annealing for 2 min at 48°C, and extension for 3 min at 72°C (39). PCR of genomic DNA sequence from In(a+b−) propositus Bi was accomplished as described above, using the primers MT11 and MT12 (Table I), 1 μg of genomic DNA, and an annealing temperature of 55°C.

**Site-directed Mutagenesis—CD44W18 in ptZ18U was used as a template for synthesis of mutated cDNA fragments, using the mutagenizing primers listed in Table I in conjunction with either MT1 (for the noncoding primer Δ252n) or MT2 (for the coding-sense primer Δ252s), in order to synthesize overlapping mutated CD44 cDNA fragments.**

**Oligonucleotides used for RT-PCR and site-directed mutagenesis**

| Oligonucleotide (5’ to 3’) | Name   | Position
|---------------------------|--------|-----------
| CCA GCC TCT GCC AGG TTC GGT | MT1    | 1–21
| TCA GTA GCA CAT TGC ATG | MT2    | 1273–1292
| GAG TAT ACC CTT CGG ATT CTT | MT11   | 187–207
| ATG CCT AGA CTT TTC ATT CTC | MT12   | 310–330
| AGC ATG CCT CGG AGG GCC | Δ252s  | 242–262
| GCC TCT GGT CGG AGA GAT GCC | Δ252n  | 242–262

a Numbered according to Ref. 37.

b Oligonucleotide primers used for site-directed mutagenesis; the altered base is indicated by underlining.

**Tissue**

Abolished binding of HA to a chimeric protein containing CD44 amino acids 38–46 and proposed that HA binds to a minimal consensus motif BX,B, where B represents a basically charged amino acid (Arg or Lys) and X represents seven nonacidic residues. HA binding was enhanced if additional basic residues were clustered around the ends of the motif or if a basic residue occurred within the X domain (29). To date, no studies have shown the effect of mutations within the first HA-binding motif (amino acids 38–46) on HA binding by intact CD44H and the altered base is indicated by underlining.

**Effect of CD44 Polymorphism on Hyaluronan Binding**

**Table I**

| Oligonucleotide primers used for site-directed mutagenesis | Name   | Position |
|----------------------------------------------------------|--------|----------|
| CCA GCC TCT GCC AGG TTC GGT (5’) | MT1    | 1–21    |
| TCA GTA GCA CAT TGC ATG (3’)  | MT2    | 1273–1292 |
| GAG TAT ACC CTT CGG ATT CTT (5’) | MT11   | 187–207 |
| ATG CCT AGA CTT TTC ATT CTC (3’) | MT12   | 310–330 |
| AGC ATG CCT CGG AGG GCC (5’) | Δ252s  | 242–262 |
| GCC TCT GGT CGG AGA GAT GCC (3’) | Δ252n  | 242–262 |

a Numbered according to Ref. 37.

b Oligonucleotide primers used for site-directed mutagenesis; the altered base is indicated by underlining.

**Materials and Methods**

**Cells and Serum**

Erythrocytes were collected and washed in phosphate-buffered saline (PBS). For radioimmunoassays, a 5% (v/v) erythrocyte suspension in PBS was assayed according to previously published methods (33, 34). EBV-transformed B lymphocytes from the In(a+b-) donors Dh, Rh, and Bi were produced and propagated by standard methodology. Cell lines Ra and Bi were the generous gift of Drs. F. Spring and D. Anstee (Bristol, United Kingdom).

**All monoclonal antibodies (mAbs) used in this study have been previously described or have been evaluated in the Fifth International Workshop on Differentiation Markers in Human Hematolymphoid Tissues (2, 35). Human anti-Ina were identified using routine blood bank methodology (agglutination.**

**Anti-Inb** are from the patient previously described by Ferguson and Gaal (36). Anti-Ina-Rh was supplied by the International Blood Group Reference Laboratory (F. Springs, J. Smythe, and D. Anstee, Bristol, United Kingdom). The specificity of both antisera has been confirmed by several laboratories.

**CDNA Clones and Sequencing**

The wild-type CD44 (CD44WT) cDNA clone was provided in the pCDM8 vector as a gift of Dr. Brian Seed (Bostom, MA) (37). The insert was then cloned into the pDNA3 expression vector (Invitrogen, San Diego, CA). In(a+b-) Rh and CD44 WT cDNA clones were obtained by reverse transcription-polymerase chain reaction (RT-PCR) of mRNA obtained from EBV-transformed lymphocytes using the FastTrack mRNA isolation system (Invitrogen) or standard guanidinium isothiocyanate-osium chloride isolation techniques (38). Synthesis of first-strand cDNA was accomplished by the GeneAmp RT-PCR kit (Perkin-Elmer), using oligonucleotide MT2 (Table I). Amplification conditions were as follows: denaturation for 1 min at 95°C, annealing for 2 min at 48°C, and extension for 3 min at 72°C for 40 cycles, followed by a 10-min final extension period. The phosphorylated PCR product was denatured into calf intestinal alkaline phosphatase-treated pTZ18U (Bio-Rad) linearized with Smal. Sequencing was accomplished by standard dideoxy methodology using SequenaseTM 2.0 (U.S. Biochemical Corp).

**PCR of genomic DNA sequence from In(a+b-) propositus Bi was accomplished as described above, using the primers MT11 and MT12 (Table I), 1 μg of genomic DNA, and an annealing temperature of 55°C.**
pellets were analyzed in a scintillation counter (LKB/Wallac Inc., Gaithersburg, MD) to quantitate radiolabel bound to cells.

RESULTS

Identification of DNA Polymorphisms in Three Unrelated In(a<sup>1</sup>b<sup>2</sup>) Propositi—During the course of these and previous studies, two In(a<sup>1</sup>b<sup>2</sup>) individuals, Dh and Ra, were identified, and their B lymphocytes were transformed using EBV. Cloning and sequencing of RT-PCR products of propositus Dh mRNA led to identification of five mutations in comparison with the CD44H sequence published by Stamenkovic et al. (37) (Table II and Fig. 1). However, the mutations at nucleotides 322 and 370 were silent and were present in only 50% of all clones, suggesting that Dh was either heterozygous for these mutations or that these mutations were the result of errors during amplification. This latter possibility was made less likely by the fact that these silent mutations were present in multiple clones obtained from several independent PCR amplification reactions. We then performed RT-PCR on cDNA extracted from a second EBV-transformed cell line from an unrelated In(a<sup>1</sup>b<sup>2</sup>) propositus (Ra). Partial sequencing of cDNA clones obtained from this donor demonstrated the same mutations at nucleotides 252, 370, and 441 as did Dh. However, the silent mutation at nucleotide 322 was not found, and sequencing was not extended to nucleotide 831. Comparison of the Dh sequence with that published by Goldstein et al. (43) further showed that the nucleotides found at positions 441 and 831 (codons 109 and 239, respectively) in Dh matched both that sequence as well as the murine CD44 sequence. Thus, the G<sup>252</sup>C substitution...
changing amino acid 46 from Arg to Pro appeared the most likely basis of the Inb/Ina polymorphism.

During the course of later studies, a third unrelated In(a1b2) EBV-transformed lymphocyte cell line became available. PCR amplification and sequencing of genomic DNA from this cell line once again demonstrated a G252 → C substitution.

Expression of Inb Antigen by Cells Transfected with CD44 cDNA Constructs—In order to demonstrate which mutation(s) was responsible for the Inb/Ina alteration of phenotype, Jurkat cells were transfected with wild-type CD44 cDNA (CD44WT, as described by Stamenkovic et al. (37)), cDNA amplified from the In(a1b2) propositus Dh (CD44Dh), or a mutated cDNA (CD44(R46P)) that differed from CD44WT only at nucleotide 252 (Table II). Transiently transfected cells were tested for CD44 and Inb antigen expression using mAb A3D8 and two human anti-Inb sera, respectively (Fig. 2). All cells except nontransfected cells expressed easily detectable levels of CD44 migrating at approximately 80 kDa on SDS-polyacrylamide gel electrophoresis (Fig. 2, left panel). However, only CD44WT reacted with human anti-Inb sera, whereas CD44Dh and CD44(R46P) did not (Fig. 2, right panel). These results demonstrate that the Arg → Pro substitution at amino acid 46 is sufficient to effect the Inb/Ina antigenic polymorphism.

Effect of Inb → a-associated Alteration of the First Hyaluronan-binding Motif on Hyaluronan Binding—The first HA-binding motif identified by the sequence B7 is amino acids 38–46. The Arg46 → Pro substitution of In(a1b2) cells thus removes the basically charged amino acid at the C terminus of this motif. In previous studies, substitution of this amino acid by a glycine has been shown to abolish HA binding by this sequence expressed as part of a chimeric protein (29). Therefore, we examined the ability of the intact CD44 molecule with a mutation at this site to bind HA. Previous work has demonstrated that there is little HA binding to Jurkat cells expressing transfected CD44 cDNA, while incubation with PMA increases the ability of transfected CD44H to bind HA, and PMA-treated nontransfected Jurkat still does not bind HA (42).

We studied HA binding by cells transfected with CD44WT, CD44Dh (cDNA from the In(a1b2) propositus Dh), and CD44(R46P). As shown in Fig. 3, when compared with PMA-treated CD44WT-transfected cells, PMA-treated CD44Dh transfectants showed equal ability to bind HA, as measured both by percentage of positive cells as well as by degree of binding per cell, indicated by mean fluorescence channel. When the ability of CD44 WT to bind HA was measured, it also was found to bind HA as well (Fig. 4).

Therefore, despite alteration of the first B7 motif of CD44, intact In(b–) CD44Dh and CD44(R46P) maintained their ability to bind HA.

In experiments in which PMA-treated transfected Jurkat cells were pretreated with dithiothreitol, neither CD44WT nor CD44Dh bound HA (data not shown).

Effect of Alteration of the HA-binding Site of CD44WT on the Inb Epitope—We studied the ability of five antibodies that inhibited and one antibody each that enhanced or had no effect on HA binding to interfere with the ability of human anti-Inb to bind to its epitope on In(a1b1) erythrocytes. Reverse competitive inhibition experiments were also performed.

All antibodies were shown by Western blotting to bind equally well to In(a1b2) and In(a–b+) CD44 (data not shown). mAbs BRIC235, HP2/9, BU75, 5F12, and 3F12 had been previously shown to partially or completely inhibit HA binding to
CD44-transfected J urkat cells; mAb F10.44.2 had been shown to enhance HA binding 2-fold (42). mAbs BU75 and 3F12 had further been shown to inhibit 5F12 binding to red cells by 84 and 71%, respectively (27). Saturating dilutions of all CD44 monoclonal antibodies tested produced >10% inhibition of binding of subsaturating dilutions of human anti-Inb. However, only three antibodies (BRIC235, Bu75, and HP2/9) produced more than 25% competitive inhibition of anti-Inb binding (Fig. 5). In reverse experiments, human anti-Inb was unable to inhibit or enhance binding of subsaturating dilutions of anti-CD44 mAbs by greater than 25% (data not shown).

Comparison of Effect of Monoclonal Antibody Ligands on HA Binding to CD44WT, CD44Dh, and CD44(R46P)—Monoclonal antibody 5F12 has been previously described to inhibit markedly binding of HA to PMA-stimulated CD44-transfected J urkat cells (26, 42). As shown in Fig. 6, HA bound to 36.2% of CD44WT-transfected J urkat cells in the absence of 5F12 but to only 3.0% of cells in the presence of 5F12. However, HA binding to CD44Dh and CD44(R46P) transfecants was reduced by incubation with 5F12 from 45.1 to 30.6% and from 50.0 to 17.1%, respectively. In fact, four other monoclonal antibodies (BRIC235, HP2/9, BU75, and 3F12) that inhibited HA binding to CD44WT-transfected cells showed reduced inhibitory effect on HA binding to CD44Dh and CD44(R46P) (data not shown). In all cases, antibodies had greater inhibitory effect on CD44Dh than on CD44Dh, which may be attributable to the other amino acid changes encoded for by CD44Dh cDNA. However, approximately similar degrees of enhancement of HA binding were seen when F10.44.2 was used to augment HA binding to all three variants of CD44 (data not shown).

Effect of Human Anti-Inb on HA Binding to CD44WT—Incubation of PMA-treated CD44WT transfecants with human anti-Inb had no effect on binding of HA-FITC (data not shown).

**DISCUSSION**

The CD44 protein (Fig. 7) is known to be a major receptor for HA, and it may also have physiologic importance as a receptor for fibronectin and laminin (18, 46). The N terminus of CD44 is homologous to cartilage link protein, but this domain only contains one of the two amino acid motifs (KNGRYSISR, residues 38–46) thought to be active in cellular binding to HA (29). The more C-terminal portion of the extracellular domain of CD44 encompasses two overlapping BX-B motifs (RDGTRVVQGKRYR, residues 150–162). Thus far, there has been insufficient evidence to indicate whether one of these two amino acid motifs is the more important in HA binding. Both motifs, expressed either as peptides or as part of chimeric molecules, have shown HA-binding activity (28, 29), and Liao et al. have suggested that the two motifs can come into proximity to one another and can cooperate in HA binding (44).

We have shown that a naturally occurring polymorphism of CD44 coding sequence is responsible for the expression of Inb/Ina blood group antigens. Evidence that the basis of the Ina/Inb polymorphism is an Arg46 → Pro mutation disrupted an HA consen-

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

**Fig. 5.** Inhibition of anti-Inb reactivity with human red blood cells by anti-CD44 mAbs with various effects on HA binding. mAbs BRIC235, 3F12, BU75, HP2/9, and 5F12 have been previously shown to inhibit HA binding to CD44, while mAb NIH44–1 had no effect on HA binding, and mAb F10.44.2 increased HA binding 2-fold (42). mAbs BRIC235 and HP2/9 showed marked but incomplete inhibition of anti-Inb binding, while mAb 5F12, which inhibits HA binding equally well, showed little inhibition. No antibody enhanced binding of anti-Inb.

**Fig. 6.** Inhibition of HA binding to various CD44 proteins by mAb 5F12. HA binding to PMA-treated J urkat cells transfected with CD44WT, CD44Dh, or CD44(R46P) was measured in the presence (shaded bars) and absence (black bars) of anti-CD44 mAb 5F12. The percentage of cells expressing CD44, as measured by monoclonal antibody A3D6, was equivalent for all three cell lines (89.9, 89.0, and 88.6%, respectively). CD44 copy number, as measured by linear mean fluorescence channel, varied slightly among cell lines (for CD44WT MFC = 205.4; for CD44Dh MFC = 219.6; and for CD44(R46P) MFC = 224.7). In both immunofluorescence and Western blot assays, mAb 5F12 as well as other mAbs bound equally well to all CD44 proteins expressed (data not shown). In the absence of 5F12, HA-FITC bound to CD44 transfecants in proportion to CD44 expression. However, saturating concentrations of 5F12 antibody inhibited HA binding to CD44WT by 92%, while HA binding to CD44Dh and CD44(R46P) was inhibited by only 32 and 66%, respectively.
HA. Their experimental system, however, did not utilize the intact protein; instead, amino acids 38–46 of CD44 were expressed as part of a chimeric construct, and mutation of Arg46 → Gly abolished binding of biotinylated HA to the CD44 peptide/RHAMM chimeric protein immobilized on nitrocellulose.

The work of Liao and colleagues (44) has shown that mutation of Arg154 → His in the second putative HA-binding motif also did not abrogate HA binding to recombinant intact CD44 expressed in J urkat cells, although other studies on the second HA-binding motif have shown that altering basic residues within this motif did reduce the ability of CD44 chimeric constructs to bind HA (28, 29). Clearly, conclusions from studies of HA binding to small peptides or chimeric constructs cannot necessarily be extrapolated to the full protein expressed by intact cells.

The cooperation of both extracellular BX, B motifs in the binding of HA has been proposed by Liao et al. (44). This hypothesis is further supported by the complete abrogation of HA binding to PMA-treated CD44-transfected J urkat cells pre-treated with dithiothreitol. As depicted in Fig. 7, the conformation of CD44 is likely to be highly dependent on disulfide bonds. Interaction of the BX, B motifs would thus be likely to be disrupted by reduction of these bonds and subsequent alteration of CD44 conformation. However, it should be noted that other studies concerning HA binding to proteins containing BX, B binding motifs have shown that the motif may not be required for the ligand to bind the molecule. Specifically, mutants of the cartilage link protein in which the exon containing this motif had been deleted nevertheless bound HA (47).

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