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Localization of PGI (Biglycan, *BGN*)[1] and PGII (Decorin, *DCN*, PG-40)[1] Genes on Human Chromosomes Xq13–qter and 12q, Respectively

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The genes for PGI (biglycan, *BGN*) and PGII (decorin, *DCN*) have been assigned to human chromosomes X and 12 by Southern analysis of a panel of human–rodent somatic cell hybrid DNAs with cDNA probes for these related small proteoglycans. Regional localization of *BGN* to Xq13–qter and *DCN* to 12p12.1–qter was also obtained by examining hybrids containing spontaneous breaks or well-characterized translocations involving chromosomes X and 12. Biglycan (*BGN*) is a single-copy gene about 6 kb in length. Hybridization with subfragment cDNA probes suggests the presence of two copies of the decorin (*DCN*) gene, or related sequences, at the locus on chromosome 12, although there is no evidence for function of more than one *DCN* gene. Efforts to detect restriction fragment length polymorphisms with these probes were unsuccessful.

INTRODUCTION

PGI (biglycan or *BGN*) and PGII (decorin or *DCN*) are related but distinct small proteoglycans found in many connective tissues (Heinegard *et al.*, 1985; Hassell *et al.*, 1986). cDNAs encoding the core proteins (M_r 40,000) have been sequenced (Krusius and Ruoslahti, 1986; Day *et al.*, 1987; Fisher *et al.*, 1989) and indicate that the two proteins are composed predominantly of a series of 12 tandem repeats of a nominal 24-residue consensus sequence (Fisher *et al.*, 1989). The precise functions of the small proteoglycans *in vivo* are not known; however, *in vitro* studies using small proteoglycans from bovine skin demonstrated a specific, regular binding of the molecules to the "d" band of type I collagen fibrils (Scott and Haigh, 1985). This interaction is mediated by the core protein of the proteoglycans (Vogel *et al.*, 1987) and may be related to their ability to retard collagen fibrillogenesis *in vitro* (Scott *et al.*, 1981). The small dermatan sulfate proteoglycans also interact with fibronectin (Schmidt *et al.*, 1987), which in turn appears to inhibit the adhesion of fibroblasts to this substratum (Lewandowska *et al.*, 1987). Chinese hamster ovary cells stably transfected with human decorin cDNA formed more orderly monolayers and grew to lower saturation densities, implying some role for this protein in contact inhibition of cell proliferation (Yamaguchi and Ruoslahti, 1988). To determine the nature and location of the PGI (*BGN*) and PGII (*DCN*) genes in the human genome, we have used radiolabeled cDNA encoding the two human proteins and hybridized it to DNA isolated from human–rodent somatic cell hybrid clones. Here we report that PGI (biglycan) and PGII (decorin) are single-locus genes located on chromosomes Xq13–qter and 12q, respectively.

MATERIALS AND METHODS

Preparation of Probes

The nearly full-length human cDNAs encoding proteoglycans I (P16 and P6) and II (P2) have been described previously (Fisher *et al.*, 1989). P16 was a 1.7-kb PGI cDNA insert containing 120 bp of 5′-untranslated sequence, the entire 1104-bp coding sequence, and 460 bp of 3′-untranslated region. P6 was a 2.8-kb PGI insert containing an additional 1.1 kb of 3′-untranslated region, and it was identical to P16 in all other respects. P2 was a 1.6-kb PGII cDNA insert extending from codon 6 to the polyadenylation site (1.06 kb coding and 0.54 kb 3′-untranslated sequence). The P2 insert was also excised as 0.55-kb *Eco*RI–*Sac*I 5′- and 1.05-kb *Sac*I–*Eco*RI 3′-cDNA subfragments. Insert fragments and subfragments were purified by gel electrophoresis, electroeluted, and labeled with [α-^{32}P]dCTP by nick-translation.

Sequence data from this article have been deposited with the EMBL/GenBank Data Libraries under Accession No. JO4599.

[1] PGI (biglycan) and PGII (decorin) have been assigned the symbols *BGN* and *DCN*, respectively, by the Human Gene Nomenclature Committee.

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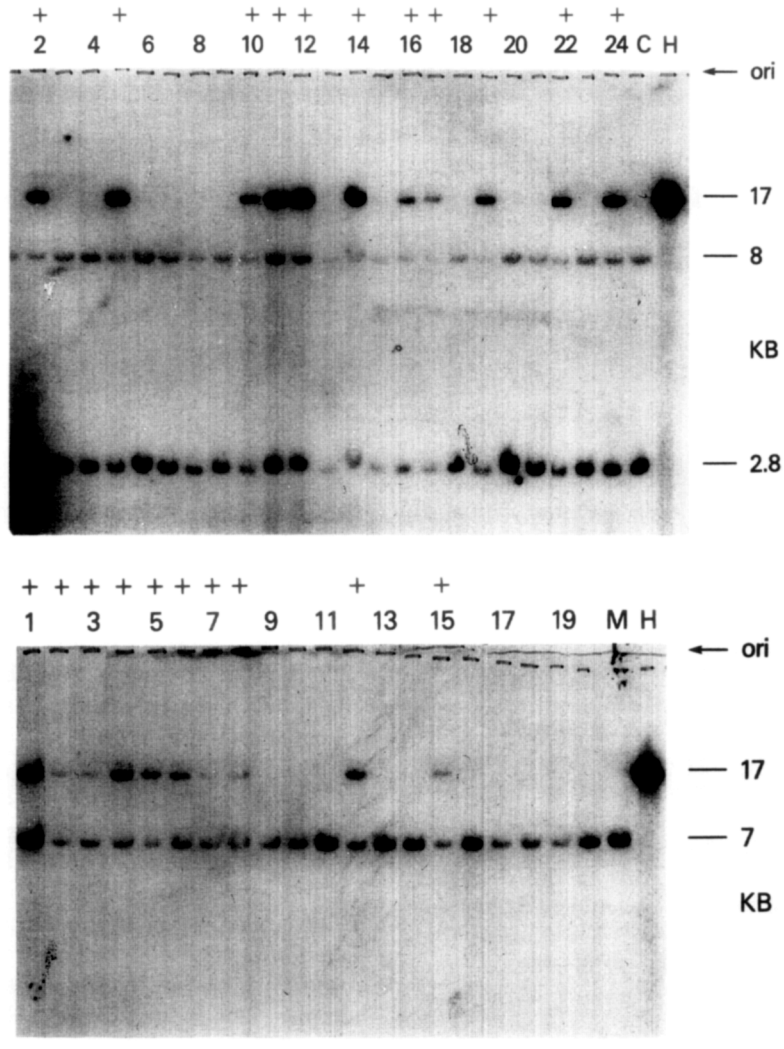

FIG. 1. Southern hybridization of representative *Eco*RI-digested human–hamster (**top**) and human–mouse (**bottom**) hybrid cell DNAs with 1.7-kb full-length human proteoglycan I cDNA probe. Each lane contains a different hybrid cell DNA. Parental Chinese hamster (**C**), mouse (**M**), and human placental (**H**) DNAs are also shown. Lanes containing the hybridizing 17-kb human band are identified (+). Some of the human–hamster hybrids (**top**) contained the human Xq13–qter translocation chromosome (lanes **2, 5, 11, 22,** and **24**), while others contained only the reciprocal Xpter–q13 translocation chromosome (lanes **9, 18,** and **20**). The presence of the PGI gene correlates with the presence of Xq13–qter.

Cell Hybrids

The human and rodent parental cells, fusion procedure, and isolation and characterization of hybrids have been described (McBride *et al.*, 1982a,b, 1983). In general, hybrid cells were analyzed for the presence of all human chromosomes except Y by standard isoenzyme analysis, as well as by Southern analysis with probes from previously localized genes, and frequently by cytogenetic analysis. The only human chromosomes present in the A9/HRBC2-A human–mouse cell hybrid (Di Cioccio *et al.*, 1975) were X and the distal portion of chromosome 2p (Olsen *et al.*, 1981), whereas only X_q was retained in 1W1-5 hybrid cells (Buck and Bodmer, 1976; Olsen *et al.*, 1980). The human–hamster hybrid AV/CHT-SC1 contained only human chromosomes X, 22, and 6pter–q13 (McBride *et al.*, 1983). Somatic cell hybrid lines containing well-characterized translocations or spontaneous breaks involving human chromosomes X and 12 were used for regional localizations of PGI and PGII genes. Nine hybrids isolated after fusion of human fibroblasts (GM0073) containing an X;14(q13;q32) reciprocal translocation with Chinese hamster fibroblasts (McBride *et al.*, 1982b) retained the Xq13–qter translocation chromosome and three hybrids contained the Xpter–q13 translocation chromosome; the normal X chromosome and the reciprocal translocation chromosome were absent in all of these hybrids. Human–mouse hybrids containing spontaneous breaks involving human chromosome 12 with loss of most of the short arm have been reported (McBride *et al.*, 1983). Mouse cell lines containing lim-

TABLE 1

Segregation of Proteoglycan P1 (*BGN*) Gene with Human X-Chromosome Long Arm

Human chromosome	Gene/chromosome +/+	+/−	−/+	−/−	% Discordancy
1	23	28	11	24	45
2	17	34	9	26	50
3	25	26	8	27	40
4	37	14	17	18	36
5	19	32	5	30	43
6	32	19	15	20	40
7	22	29	15	20	51
8	25	26	11	24	43
9	21	30	10	25	47
10	15	36	4	31	47
11	23	28	6	29	40
12	25	26	8	27	40
13	18	33	13	22	53
14	32	19	19	16	44
15	25	26	18	17	51
16	25	26	10	25	42
17	31	20	21	14	48
18	26	25	16	19	48
19	19	32	10	25	49
20	28	23	12	23	41
21	37	14	17	18	36
22	21	30	6	29	42
X	51	0	0	35	0[a]

Note. The proteoglycan I (biglycan) gene was detected as a 17-kb band in *Eco*RI digests of human–rodent somatic cell hybrid DNAs after Southern hybridization with 1.7- or 2.8-kb full-length human proteoglycan P1 cDNA probes (Fig. 1). This band was easily resolved from cross-hybridizing 2.8- and 8-, or 7-kb bands in Chinese hamster or mouse DNAs, respectively. Detection of the human band is correlated with the presence or absence of each human chromosome in the group of somatic cell hybrids. Discordancy represents the presence of the gene in the absence of the chromosome (+/−) or the absence of the gene despite the presence of the chromosome (−/+), and the sum of these numbers divided by total hybrids examined (×100) represents percentage discordancy. The human–hamster hybrids contained 27 primary clones and 13 subclones (28 positive of 40 total) and the human–mouse hybrids represented 14 primary clones and 32 subclones (23 positive of 46 total).

[a] Examination of hybrids containing well-characterized translocations or spontaneous breaks allowed regional localization of the gene to Xq13–qter (see text).

ited portions of the human X chromosome following chromosome-mediated gene transfer have been described (Olsen *et al.*, 1981; McBride *et al.*, 1982c).

DNA Isolation and Filter Hybridization

DNA was isolated from hybrid cell lines, digested with *Eco*RI, and size-fractionated by (0.7%) agarose gel electrophoresis, and partially depurinated fragments were transferred to positively charged nylon membranes in 0.5 *M* NaOH (Reed and Mann, 1985). The membranes were hybridized at 42°C with ^{32}P-labeled probes in 50% formamide containing 5× SSPE (1× SSPE is 0.15 *M* NaCl, 0.01 *M* sodium phosphate, 0.001 *M* EDTA, pH 7.4), 5× Denhardt's solution (1× is 0.02% bovine serum albumin, 0.02% Ficoll, 0.02% polyvinylpyrrolidone), 10% dextran sulfate, 0.2% SDS, and sheared denatured herring sperm DNA at 200 μg/ml. Membranes were washed at 55°C in 0.1× SSC (1× is 0.15 *M* NaCl, 0.015 *M* sodium citrate, pH 7.0) containing 0.2% SDS. The membranes were used repeatedly after removal of the probe in 0.4 *M* NaOH, neutralization, and prehybridization with carrier DNA.

DNA Restriction Fragment Length Polymorphisms (RFLPs)

DNAs were isolated from the peripheral leukocytes of 10 unrelated normal individuals, digested with restriction endonucleases, size-fractionated by gel electrophoresis, transferred to nylon membranes, and hybridized with the probes.

RESULTS

The proteoglycan genes were chromosomally mapped by Southern analysis of a panel of human–rodent hybrid cell DNAs with ^{32}P-labeled probes prepared from PGI and PGII cDNAs.

PGI Mapping

A 1.7-kb cDNA insert (P16) containing the entire proteoglycan I coding region was used to probe genomic DNA from hybrid cell lines at high stringency. This probe identified a single 17-kb band in human *Eco*RI digests (Fig. 1) that was clearly resolved from cross-hybridizing sequences in hamster and mouse DNAs. Analysis of the entire group of hybrids (Table 1) allowed unambiguous localization of the proteoglycan I gene to the human X chromosome, and it segregated discordantly (≥36%) with all other human chromosomes. This localization was confirmed by detection of the gene in a human–mouse hybrid (A9/HRBC2-A) that contained only the human X chromosome and distal short arm of chromosome 2 (2p) and in a human–hamster hybrid (AV/CHT-SC1) that retained only human chromosomes X, 22, and the short arm of 6. Examination of hybrids isolated (McBride *et al.*, 1982b) after fusion of human fibroblasts containing an X;14(q13;q32) reciprocal translocation with hamster fibroblasts permitted regional localization of the PGI gene. Nine independent hybrids containing the Xq13–qter translocation chromosome in the absence of the normal X also retained the gene, whereas four hybrids containing the reciprocal translocation chromosome had lost the gene (Fig. 1, top). Two other hybrids with spontaneous X-chromosomal breaks and loss of Xq also lost the proteoglycan I gene. Finally, a hybrid (1W1-5) containing only the long arm of the human X chromosome also retained the gene. A series of cell lines

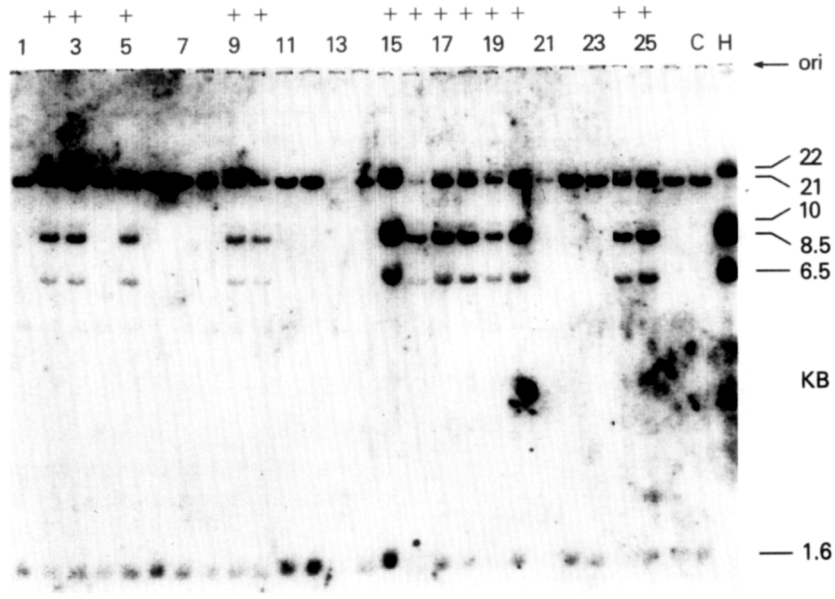

FIG. 2. Hybridization of *Eco*RI-digested human–hamster hybrid cell DNAs with a 1.6-kb full-length human proteoglycan P2 cDNA probe. A different hybrid cell DNA is present in each lane, and parental Chinese hamster (**C**) and human placental (**H**) DNAs are also shown. Presence of the 6.5-, 8.5-, 10-, and 22-kb human bands is indicated above the lanes (+).

containing only fragments of the human X chromosome in a mouse background following chromosome-mediated gene transfer (Olsen *et al.*, 1981; McBride *et al.*, 1982c) was also examined, and the gene was retained in only one line (A9S2) which contained the entire distal one-third to one-half of the X chromosome long arm. These results permit definite localization of the proteoglycan I gene to Xq13–qter and probable localization to Xq22–qter.

PGII Mapping

The proteoglycan II gene was chromosomally mapped by Southern analysis of the same hybrid cell DNAs with a 1.6-kb cDNA probe (P2) containing nearly the entire PGII coding sequence. This probe detected four human bands in *Eco*RI digests (Fig. 2) which segregated concordantly (i.e., all present or all absent together) and could be assigned to human chromosome 12 (Table 2). These bands segregated discordantly (≥19%) with all other human chromosomes. Analysis of several hybrids containing spontaneous breaks involving chromosome 12 permitted localization of the gene to the long arm, or proximal short arm, of this chromosome (12p12.1–qter) below the K-*ras*2 protooncogene. Two independent human–hamster hybrids and one human–mouse hybrid, containing deletions of much or all of the chromosome 12 long arm, retained KRAS2, triose phosphate isomerase 1 (TPI1), and LDHB but not the PGII gene or five other markers for 12q including the anonymous DNA segment pDL32B (Tsui *et al.*, 1985) at q14.3–qter and PEPB (Hamerton *et al.*, 1975) at q21. Two human–mouse hybrids that had lost the distal portion of 12p (including TPI1 and LDHB) retained the PGII gene, as did a series of subclones of a human–mouse hybrid that had lost these short arm markers including KRAS2.

Analysis for RFLPs

DNAs isolated from peripheral leukocytes of 10 unrelated individuals were digested separately with *Eco*RI, *Hin*dIII, *Bam*HI, *Xba*I, *Sac*I, *Taq*I, *Pvu*II, *Pst*I, *Eco*RV, *Bgl*II, *Msp*I, and *Kpn*I. Southern blots of these restriction digests were hybridized with the 1.7-kb (P16) and 2.8-kb (P6) PGI probes as well as with the 1.6-kb (P2) PGII insert probes. No restriction fragment length polymorphisms were detected with any of these probes.

PGI (BGN) Gene Copy Number and Size

Several hybridizing bands were found in most human DNA restriction digests after Southern hybridization with the PGI (P16) cDNA probe (Table 3). The number of bands observed in *Hin*dIII, *Taq*I, *Eco*RI, and *EcoRV* digests was identical to that predicted on the basis of restriction sites for these enzymes in the cDNA. This result strongly suggests the presence of a single PGI gene in humans. The number of hybridizing bands observed in digests with other restriction enzymes requires the presence of restriction sites for these enzymes in at least two introns. These results also allow estimation of the size of the PGI gene, which is 5.8–6.5 kb in length including 3.2–3.9 kb of intronic sequences.

TABLE 2

Segregation of Proteoglycan P2 (*DCN*) Gene with Human Chromosome 12

Human chromosome	Gene/chromosome +/+	+/−	−/+	−/−	% Discordancy
1	24	10	10	47	22
2	20	14	6	51	22
3	21	13	14	43	30
4	28	5	30	27	40
5	21	13	4	53	19
6	28	6	18	39	26
7	18	16	18	39	37
8	21	13	14	43	30
9	23	11	8	49	19
10	14	20	5	52	27
11	22	12	5	52	19
12	34	0	0	57	0
13	20	14	12	45	29
14	23	11	17	40	31
15	25	9	19	38	31
16	15	19	21	36	44
17	28	6	25	32	34
18	21	13	26	31	43
19	19	15	9	48	26
20	28	6	12	45	20
21	28	6	31	26	41
22	17	17	10	47	30
X	23	11	23	34	37

Note. The human proteoglycan P2 gene was detected as 6.5- and 8.5-kb bands in EcoRI-digested human–rodent somatic cell hybrid DNAs after Southern hybridization with a full-length human P2 proteoglycan cDNA probe. These bands were readily resolved from 1.6-, 5.2-, and 21-kb cross-hybridizing bands in Chinese hamster DNA digests (Fig. 2) and hybridization with mouse DNA (not shown) was too weak for detection. Detection of the human bands is correlated with the presence or absence of each human chromosome in the group of somatic cell hybrids. The human–hamster hybrids contained 27 primary clones and 13 subclones (18 positive of 40 total), and the human–mouse hybrids represented 15 primary clones and 36 subclones (16 positive of 51 total). Analysis of several hybrids containing spontaneous chromosome 12 breaks and translocations permitted regional localization of the gene to 12p12.1–qter (see text).

PGII (DCN) Gene Copy Number

Multiple bands hybridized with the P2 probe in most restriction digests. These Southern blots were also hybridized with contiguous 0.55-kb 5′- and 1.05-kb 3′-cDNA subfragment probes to permit an estimate of the PGII gene copy number (Table 4). The maximum number of potential PGII genes (and/or pseudogenes) can be estimated from the number of bands hybridizing with either of these probes. Moreover, no more than one common band should be detected by two contiguous probes in any restriction digest if a single gene is present. The results are most compatible with the presence of two PGII sequences at the locus on chromosome 12. In *Eco*RI digests, four bands hybridize with the 0.55-kb 5′-cDNA probe and two of these bands are also detected with the 3′-cDNA probe, which suggests the presence of two PGII sequences each containing an *Eco*RI site (intronic) within the region spanned by the 5′-cDNA probe. A single *Sac*I site within the PGII cDNA separates the 5′- and 3′-cDNA probes. The hybridization pattern in *Sac*I digests again suggests the presence of two hybridizing PGII sequences, with the

TABLE 3

Hybridization with PGI Probe

Enzyme	Sizes of hybridizing bands (kb)	Restriction sites in cDNA (bp)	Restriction sites in introns (No.)	Gene size (kb)[a]
*Eco*RI	17	—	0	<17
*Hin*dIII	(14.8)[b]	238	0	5.8–28.3
	(9.0)[c]	1507		
	4.5			
*Bam*HI	9.1	—	1	<12.5
	(3.4)			
*Xba*I	12.7	—	1	<17
	(4.25)[b]			
*Sac*I	3.25	432	2	4.9–8.0
	1.85	972		
	1.43			
	1.03			
	0.46			
*Pvu*II	(4.5)[b]	1233	2	3.25–10.0
	2.24	1328		
	1.61			
	1.02			
	(0.62)[b]			
*Pst*I	2.6	411	1	<6.5
	(1.85)[b]	1236		
	1.23			
	0.79			
*Eco*RV	>25		0	<25
*Bgl*II	27	382	2	3.2–41.2
	(11)[b]	496		
	1.37			
	1.19			
	(0.68)[b]			
*Kpn*I	32	—	1	<34
	1.86			
*Taq*I	5.1	684	0	3.6–8.7
	1.55	735		
	1.13	924		
	0.57	1373		
	0.37			
	2.65[d]			

[a] The maximum possible size of the gene was assumed to represent the sum of all hybridizing bands in each restriction digest. Since two bands in each digest represent fragments extending for unknown distances into the two flanks, the minimum size was computed by omitting the two largest restriction fragments.

[b] Weakly hybridizing bands shown in parentheses.

[c] Weak hybridization signal with 1.7-kb (P16) probe and strong signal with 2.8-kb (P6) probe. The 4.5-kb fragment probably extends between the two internal cDNA restriction sites, whereas the 9- and 14.8-kb fragments extend into the 3′ and 5′ flanks, respectively.

[d] This strongly hybridizing band was detected only with the P6 probe containing the complete 3′-untranslated region.

TABLE 4

Hybridization with PGII Probes

Enzyme	Sizes of hybridizing bands (kb) 5′-cDNA probe	3′-cDNA probe
*Eco*RI	22	—
	(10.5)	—
	(8.9)	8.9
	6.6	(6.6)
*Hin*dIII	8.75	8.75
	(6.45)	—
	—	6.1
	5.0	—
	(0.92)[a]	
*Bam*HI	>30	>30
*Xba*I	9.0	(9.0)
	3.55	—
	—	3.5
	(3.4)	—
	(1.25)	1.25
*Sac*I	28.5	—
	17	—
	—	10.1
	(6.0)	6.0
*Pvu*II	18	18
	10	—
	2.63	—
	1.95	—
	(1.7)[a]	
*Pst*I	20	20
	4.45	—
*Eco*RV	18	—
	16	16
*Bgl*II	7.9	—
	—	7.3
	—	(6.8)
	1.26	—
*Kpn*I	23	23
	9.1	—

Note. The same membrane was hybridized separately with both probes. The presence of a number on the same line in both columns indicates detection of the same band with both probes. Weakly hybridizing bands are indicated in parentheses. Restriction sites contained within the P2 cDNA were *Hin*dIII at 823 and 1360 bp, *Sac*I at 546 bp, and *Pvu*II at 403 bp.

[a] Denotes detection of a weakly hybridizing band with full-length cDNA probe but not with either subfragment probe.

*Sac*I site conserved in one sequence and possibly further 5′ to this site in the other copy. The observations with *Xba*I and *Pvu*II digestions are also most consistent with the presence of two genomic sequences. The other results (Table 4) do not exclude the presence of a single gene but strongly suggest that no more than two PGII sequences are present.

DISCUSSION

This paper reports the chromosomal assignments and copy number of genes for proteoglycans I and II. PGI (*BGN*) is clearly a single-copy gene located on the long arm of the human X chromosome (Xq13–qter). This gene contains at least two introns and it spans about 6 kb in length.

The proteoglycan II (*DCN*) gene has been localized to the proximal short arm or long arm of human chromosome 12 (12p12.1–qter). The pattern of hybridization with PGII cDNA probes is more complex than that found with PGI, and it appears to reflect the presence of two copies of PGII at the locus on chromosome 12. Krusius and Ruoslahti (1986) also suggested the presence of a PGII-related gene on the basis of results of hybridization with genomic digests. Thus far, however, no evidence for the presence of two functional PGII genes producing two different mRNA transcripts has been presented. Identical cDNAs were cloned in two different laboratories (Krusius and Ruoslahti, 1986; Fisher *et al.*, 1989). An additional cDNA (clone 5E of Krusius and Ruoslahti) did differ from the other three reported clones in the 3′-untranslated region. However, the coding sequence and first 134 nucleotides of the 3′-flanking region were identical to those of all the other clones, whereas the sequence of the 3′-flanking region diverged almost completely after that point. It seems improbable that the coding regions and the 3′-flanking sequences of two functional genes would be conserved precisely while the more distal 3′ sequence would diverge sharply and completely unless this represented an insertion element such as an *Alu* sequence.

Although the precise role of the proteoglycans is not yet known, they are important elements of the extracellular matrix. Thus, they have a potential role in diseases involving bone and connective tissues as well as involvement in cell attachment, cell migration, and probably regulation of cell proliferation. No RFLPs could be detected with the cDNA probes available. Additional studies will be required to identify polymorphic markers suitable for use in evaluation of the role of these genes in hereditary diseases.

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