Decorin Binds Near the C Terminus of Type I Collagen*

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Decorin belongs to a family of small leucine-rich proteoglycans that are directly involved in the control of matrix organization and cell growth. Genetic evidence indicates that decorin is required for the proper assembly of collagenous matrices. Here, we sought to establish the precise binding site of decorin on type I collagen. Using rotary shadowing electron microscopy and photoaffinity labeling, we mapped the binding site of decorin protein core to a narrow region near the C terminus of type I collagen. This region is located within the cyanogen bromide fragment CB6 of the α(I) chain, in a zone that coincides with the C1 band of the collagen fibril α-period. This location is very close to one of the major intermolecular cross-linking sites of collagen heterotrimers. Thus, decorin protein core possesses a unique binding specificity that could potentially affect the structure and cross-linking of collagen.

In this study we sought to establish the exact binding site of decorin on type I collagen. Employing two independent strategies, rotary shadowing electron microscopy and photoaffinity labeling, we mapped the major binding site of decorin protein core near the C terminus of type I collagen. This region is located within the cyanogen bromide fragment CB6 of the α(I) chain, in a zone that coincides with the C1 band of the collagen fibril α-period. This location is very close to one of the major intermolecular cross-linking sites of type I collagen. Thus, decorin possesses a unique binding specificity that could potentially affect the structure and cross-linking of collagen.

EXPERIMENTAL PROCEDURES

Purification of Decorin and Procollagen and Rotary Shadowing Electron Microscopy—Decorin proteoglycan and its protein core were purified to homogeneity from the medium conditioned by HT1080 cells as described previously (15). The purity of the final decorin preparations was determined by SDS-PAGE following labeling (~10 μg each) to high specific activity (~9 × 10^17 cpm/mol) using IODO-GEN (Pierce) and 125I (Amersham Pharmacia Biotech). As an additional control, we iodinated the 175-kDa immunopurified EGF receptor (17). Type I procollagen, isolated from cultured chick tendon fibroblasts (18), was incubated at 23°C for various intervals (15 s, 2 min, 15 min, and 60 min). Procollagen, decorin, and decorin core were also evaluated individually for purity. The samples were dissolved in 0.1 M ammonium bicarbonate buffer, pH 8.0, mixed to a final concentration of 70% glycerol, sprayed onto freshly cleaved mica, and rotary shadowed at 6 degrees using a mixture of platinum and carbon in a Balzers BAE 250 evaporator (19). Images were collected using a Philips 410 TEM, calibrated using a carbon grating replica (Fullam), confirmed by graphitized carbon (Polaron), and enlarged to a final magnification of ×100,000. Measurements were taken using a 10× loupe fitted with a reticle having scale divisions of 0.1 mm. The length of the procollagen triple helix was measured beginning at the N-terminal end of the C-propeptide and continuing to the middle of the bound decorin core.

Photoaffinity Labeling—The reconstituted decorin core was derivatized with SADAS and was previously radiolabeled with 125I using sodium iodide and IODO-BEADS. Decorin core was reacted at a 1:50 ratio with SADAS for 30 min which resulted, on the average, in derivatization of each I domain molecule with three SADAS groups. Photoaffinity-derivatized decorin core was separated from reactants by gel filtration chromatography. All of the above procedures were carried out in the dark. Derivatized decorin was then allowed to bind to reconstituted collagen fibrils in a glass Petri dish. After removing unbound decorin, bound decorin was covalently cross-linked to collagen by irradiation with long structure itself, several molecules can regulate and fine tune this process (7, 8). Decorin induces a delayed fibril assembly and a subsequent reduction in the average fibril diameter (9). This process is mediated by the protein core (10), likely by the central LRRα–ℓ (11–13), and requires preservation of disulfide bonding (14) and proper folding of the protein core (15). In support of these biochemical findings is genetic evidence derived from decorin null animals in which aberrant collagen formation in the dermis causes a skin fragility phenotype (16). In the absence of decorin, the collagen network of the null animals is loosely packed and exhibits irregular collagen contours, with numerous thin fibrils abnormally fused to larger collagen shafts. Thus, the skin fragility phenotype can be ascribed to the generation of an abnormal collagen fibril and matrix structure in the absence of the regulatory protein decorin.

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2 The abbreviations used are: LRR, leucine-rich repeat; PAGE, polyacrylamide gel electrophoresis; SADAS, sulfosuccinimidyl-2-[γ-azidocaproylamido]ethyl-1,3'-dithiopropionate; EGF, epidermal growth factor.
wave ultraviolet light. The covalent linkage between decorin and collagen was then severed by reduction with β-mercaptoethanol, resulting in tagging of the decorin-binding site on collagen. The labeled collagen was then subjected to SDS-PAGE and autoradiography before or after cleavage with CNBr, followed by staining with Coomassie Blue or autoradiography to identify the labeled cross-linking site.

RESULTS AND DISCUSSION

Arch-shaped Structure of Decorin—To establish the purity of the preparations, we radiiodinated decorin or its protein core and subjected them to SDS-PAGE and autoradiography. There were no contaminant bands in any preparation of decorin core (43–50 kDa) or decorin proteoglycan, a polydisperse population centering around 100 kDa (Fig. 1A). As an additional molecular weight marker, we labeled the 175-kDa EGF receptor (Fig. 1A). The same preparations of decorin and decorin core were capable of binding to and inducing phosphorylation of the EGF receptor in A431 squamous carcinoma cells (17) and concurrently inducing growth suppression (not shown). Thus, the preparations used in this study are pure and biologically active.

Using rotary shadowing electron microscopy, arch-shaped structures were clearly discernible for nearly all the molecules (Fig. 1B). The overall dimensions were ~7 nm (the distance between the two arms) × ~5 nm (the distance between the base of the arch and the apex). These measurements are very close to those obtained with the three-dimensional model of decorin (6.5 × 4.5 × 3 nm) (20). These data are in agreement with those reported for a mixed population of leucine-rich proteoglycans (including decorin, fibromodulin and lumican) isolated from bovine sclera (21). Our study is the first to demonstrate that biologically active human decorin, based on its ability to modulate EGF receptor activity, folds into an arch-shaped structure similar to the ribonuclease inhibitor (22, 23).

Decorin Binds to a Region Near the C terminus of α1(I) Collagen Chain—Binding of decorin to collagen interferes with horizontal accretion of collagen molecules and prevents lateral growth of the fibrils (2). Thus, the exact location of the decorin-binding site on type I collagen is of general interest. To address this point, we first examined the in vitro binding of decorin or its protein core to mature type I collagen isolated from bovine skin. Although binding could be localized near the end of these molecules (not shown), the lack of polarity of the mature collagen precluded any meaningful interpretation of the data. To circumvent this problem, we took advantage of the presence of the relatively large, globular C-propeptide in procollagen molecules. To this end, we purified type I procollagen from freshly isolated embryonic chick tendon fibroblasts and analyzed it by rotary shadowing electron microscopy. Approximately 60% of

![Fig. 1. Purity of the decorin preparation and arch-shaped structure of the decorin core. A, autoradiograph of decorin protein core and decorin proteoglycan following iodination with 125I. Notice the lack of contaminants in the preparations of decorin core (43–46 kDa) or decorin proteoglycan, a polydisperse population centering around 100 kDa (Fig. 1A). As an additional molecular weight marker, we labeled the 175-kDa EGF receptor (Fig. 1A). The same preparations of decorin and decorin core were capable of binding to and inducing phosphorylation of the EGF receptor in A431 squamous carcinoma cells (17) and concurrently inducing growth suppression (not shown). Thus, the preparations used in this study are pure and biologically active.](image)

![Fig. 2. Gallery of electron micrographs of rotary shadowed molecules of procollagen alone (A) or interacting with either decorin proteoglycan (B, C) or decorin protein core (D, E). A, type I procollagen shadowed alone. The C-propeptide is positioned to the right in each figure. B, decorin most commonly binds to a region close to the C-propeptide, although other sites are occasionally identified (arrowheads). C, linear aggregates of procollagen B decorin complexes display periodicity (arrowheads). D, decorin protein core almost uniformly binds to a region near the C terminus (arrowheads). E, occasionally, a loop is formed when the N terminus of a triple helix crosses at the decorin core-binding region, a profile not seen in samples of procollagen shadowed alone. Examples of aggregates of two or more molecules complexed with decorin core are also shown. The aggregates are often joined at the site bound by decorin core and can often be recognized by a close proximity of C-propeptide globular domains. This theme is also recognized in larger aggregates (arrowheads). All images excluding two lowermost; bar = 100 nm. Lowermost two images; bar = 200 nm.](image)
FIG. 3. Decorin core binds to the C terminus of procollagen. Quantitative analysis of decorin/procollagen (A), decorin core/procollagen (B), and combined decorin and decorin core/procollagen (C) interactions. The histograms were generated with a bin size of \( \sim 10 \) nm, and the frequency distribution of binding events along the procollagen monomers were plotted as a distance from the N terminus. Measurements were taken between the center of decorin bound to procollagen and the point at which the procollagen triple helix joins the C-propeptide.

The molecules existed as monomers of 293.7 ± 2.83 nm (mean ± S.D., \( n = 70 \)) and included an intact globular C-propeptide (10–12 nm in diameter) that was used as a reference point for polarity (Fig. 2A). When decorin was incubated with procollagen (1:1), it bound not only near the C terminus of procollagen, but also in multiple regions along the monomer (Fig. 2B). Particularly common in the decorin + procollagen mixtures were linear aggregates (Fig. 2C), not seen in the pure procollagen samples. These aggregates, which formed quickly even in samples sprayed within 15 s of mixing, demonstrated periodicity (Fig. 2C), due to the presence of bound decorin and/or retained C-propeptide.

When decorin core was mixed with procollagen, it clearly bound near the procollagen C-propeptide (Fig. 2D). Notably, the decorin core appeared smaller when bound to collagen (compare Fig. 2D with 2B). We interpret this as due to the intercalation of the procollagen monomer within the groove of the arch-shaped decorin core. The rotary shadowing profile of two intercalated molecules should, in fact, increase by a relatively small amount, because part of the shadow would be generated by the procollagen triple helix. The larger appearance of decorin proteoglycan bound to procollagen does agree with this concept, since the two molecules could interact via the glycosaminoglycan chain and thus would not be intercalated. Occasionally, two procollagen molecules were joined near their C-propeptide or single molecules formed loops when a portion of the radiolabeled photoaffinity probe. Notice the presence of a major band migrating at \( \sim 43 \) kDa (asterisk). Molecular mass markers are in lane 2. B, the vast bulk of the labeling of decorin is incorporated into a fragment co-migrating (lane 2, arrow) with the \( \alpha(1) \) CB6 peptide of type I collagen (lane 1). In lane 1, CNBr-generated peptides of type I collagen were separated on a reducing SDS-PAGE and stained with Coomassie Brilliant Blue. Lane 2 is an autoradiograph of a similar gel. C, schematic representation of human \( \alpha(1) \) and \( \alpha(1) \) chains illustrating the major CNBr peptides. The position of each methionine residue is indicated by vertical lines. The bottom panel shows the sequence of the decorin-binding site within the \( \alpha(1) \) chain as predicted from the rotary shadowing data. Hyd*, one of the two major intermolecular cross-linking sites, is labeled by an asterisk.

Quantitative analysis of decorin binding to procollagen revealed a major interactive site (41% of binding events) centering at \( \sim 275 \) nm from the N terminus (Fig. 3A), in addition to other binding sites. However, the data obtained with the decorin core were quite clear, with \( \sim 75\% \) of all the observations (\( n = 72 \)) again falling within a short interval between 260 and 280 nm from the N terminus (Fig. 3B). When the data were combined (Fig. 3C), the vast majority (\( \sim 67\% \) of the observations, \( n = 96 \)) again fell within the narrow interval between 260 and 280 nm from the N terminus, thereby positioning the decorin-binding site at \( \sim 25 \) ± 4 nm from the C-propeptide (see below).

Decorin Protein Core Binds Specifically to the CNBr Peptide \( \alpha(1) \) CB6—To confirm the rotary shadowing data, we utilized a photoafffinity labeling and cross-linking approach. We derivatized decorin core with SASD, allowed it to interact with collagen fibrils, and covalently cross-linked the \( ^{125}\text{I}-\)decorin core to the collagen with long wave UV light. The covalent linkage
between decorin and collagen was then severed by reduction with β-mercaptoethanol, resulting in tagging of the decorin-binding site on collagen followed by CNBr digestion and SDS-PAGE. An aliquot of the SASD-labeled decorin core was analyzed by SDS-PAGE for purity, and a single band of ~43 kDa was detected by autoradiography (Fig. 4A). A major binding site of decorin corresponded to the α1(I) CB6 collagen peptide (Fig. 4B), the most C-terminal fragment (Fig. 4C). The identity of all the CNBr peptide bands was confirmed by N-terminal sequencing (not shown).

It is not known whether decorin specifically binds to α1(I) or α2(I) or to both chains. Our results show that decorin binds to the α1(I) but not to the α2(I) chain, despite their significant homology. The specificity of binding further suggests that the α1(I) chain might differ in sequence in the decorin-binding site (see below). Taken together, these findings independently corroborate the rotary shadowing electron microscopy data presented above. Moreover, the data indicate that the binding site of decorin on the procollagen triple helix and the mature reconstituted fibrils is the same.

Precise Location of the Decorin-Binding Site on Collagen α1(I)—The best fit between electron microscopic and sequence-generated data was achieved for the 67-nm t-period length of 234 residues for both α1(I) and α2(I) chains (24), which computes to 0.286 nm/residue (25). Because the triple helical portion of the human α1(I) chain has 1014 residues, and the nonhelical ends have 17 N-terminal and 26 C-terminal telopeptide residues, respectively, the length of type I collagen molecules can be calculated to be ~299 nm (26). Our measurements of procollagen monomers (293.7 ± 2.83 nm) are very close to the predicted size. Thus, in doing our calculations, we corrected the location of the major decorin-binding site by ~2% (293/299 nm). This site centers at residues 961–962 according to Chapman human, calf, and chicken, in contrast with the fact that these authors have utilized decorin extracted with the; binding site at constituting fibrils is the same.

A was detected by autoradiography (Fig. 4). The position of decorin on the collagen α1(I) chain differs from that previously proposed (the d band) to represent the major binding site (28). There are several explanations for this discrepancy. First, previous studies have used cationic dyes that interact with the sulfated glycosaminoglycan chains, which are not required for binding to collagen (10, 14). Second, the tissues were fixed and dehydrated, thus including a substantial intrinsic error in the calculations, since it is well established that significant shrinkage, up to 5–20%, occurs during sample preparation for electron microscopy. Just a 5% inaccuracy would lead to ~15 nm error, which translates into a difference of 52 residues (150/2.86). This error, compounded with the fact that the glycosaminoglycan side chains are known to be highly polydisperse, makes these studies difficult to interpret (28). Indeed, cuprolinic blue-stained filaments can reach 75 nm in length, equivalent to ~12 concatenated decorin molecules. This would imply that multiple decorin molecules are positioned axially within the d-gap region, an image that was never observed in our study.

Other studies have mapped a major and a minor decorin-binding site at ~50 and 100 nm from the N terminus, respectively (29). This discrepancy can be in part attributed to the fact that these authors have utilized decorin extracted with the chaotropic agent guanidinium hydrochloride and used cuproline blue to stain the decorin proteoglycan in solution. The former could unravel cryptic binding sites that would not be present in the native molecule, whereas the latter could generate nonspecific binding, since cuproline blue is known to precipitate the glycosaminoglycan chain of decorin.

Immuno-electron microscopic studies, using either ferritin (30) or gold-labeled (31) antibodies, have detected a major binding site for decorin near the d band in the n-gap region of type I collagen. However, in both studies, a considerable proportion of binding sites was also located in the c band as in our case. Using antibodies (~15 nm in size) and either ferritin (12 nm in size) or gold (20 nm in size) labels, one has to account for these additional measurements when mapping a binding site on a molecule. Thus, decorin core could be displaced by 27–35 nm, equivalent to 94–122 amino acid residues, thereby positioning decorin within the c band.

In conclusion, we have mapped the major decorin-binding site to the c1 band of the collagen fibril, in close proximity to one of the major intermolecular cross-linking sites. Our data favor a model where decorin exhibits a high degree of specificity, since it binds to a unique site, despite the availability of the entire triple helix of collagen and the repetitive nature of the collagen sequence. The decorin core specifically binds to the α1(I) but not to the α2(I) chain, and it could play a role in the stabilization of collagen in vivo.

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REFERENCES
1. Kresse, H., Hauser, H., and Schönher, E. (1995) Experimenta (Basel) 49, 403–416
2. Vogel, R. V. (1995) Annu. Rev. Biochem. 67, 609–652
3. Vogel, R. V. (1995) J. Biol. Chem. 274, 18643–18646
4. Vogel, K. G., Paulusson, M., and Heinegard, D. (1984) Biochem. J. 233, 587–597
5. Bidanet, D. J., Guidry, C. A., Rosenberg, L. C., Choi, H. U., Timpl, R., and Hook, M. (1991) J. Biol. Chem. 267, 5250–5256
6. Hedbom, E., and Heinegard, D. (1993) J. Biol. Chem. 268, 27307–27312
7. Kadar, K. E., Holmes, D. F., Trotter, J. A., and Chapman, J. A. (1996) Biochem. J. 316, 1–11
8. Birk, D. E., Fitch, J. M., Babiars, J. P., Deane, K. J., and Linsenmayr, T. F. (1990) J. Cell Sci. 93, 649–657
9. Vogel, K. G., and Trotter, J. A. (1987) Collagen Rel. Res. 7, 105–114
10. Vogel, K. G., Koob, T. J., and Fisher, L. W. (1987) Biochem. Biophys. Res. Commun. 148, 658–663
11. Svensson, L., Heinegard, D., and Oldberg, A. (1995) J. Biol. Chem. 270, 20712–20716
12. Schönher, E., Hauser, H., Beavan, L., and Kresse, H. (1995) J. Biol. Chem. 270, 8877–8883
13. Kresse, H., Lecio, C., Schönher, E., and Fisher, L. W. (1997) J. Biol. Chem. 272, 18404–18410
14. Scott, P. G., Winterbottom, N., Dodd, C. M., Edwards, E., and Pearson, C. H. (1986) Biochem. Biophys. Res. Commun. 138, 1348–1354
15. Ramamurthy, P., Hocking, A. M., and McQuillan, D. J. (1996) J. Biol. Chem. 271, 19578–19584
16. Danielson, G. K., Baribault, H., Holmes, D. F., Graham, H., Kadar, K. E., and Izoo, R. V. (1997) J. Cell Biol. 136, 729–743
17. Izoo, R. V., Moscatello, D., McQuillan, D. J., and Eichstetter, I. (1999) J. Biol. Chem. 274, 4489–4492
18. Swanson, S., Mayne, P. M., Wright, D. W., Accvittia, M. A., Fitch, J. M., Linsenmayr, T. F., and Mayne, R. (1992) Matrix 12, 56–65
19. Sakai, L. Y., and Keene, D. R. (1994) Methods Enzymol. 245, 29–52
20. Weber, L. T., Harrison, R. W., and Izoo, R. V. (1996) J. Biol. Chem. 271, 31767–31770
21. Scott, J. E. (1996) Biochemistry 35, 8795–8799
22. Kope, B., and Deisenhofer, J. (1995) Nature 374, 183–186
23. Kajava, A. V. (1998) J. Mol. Biol. 277, 519–527
24. Meech, K. M., Chapman, J. A., and Hardest, R. A. (1979) J. Biol. Chem. 254, 10710–10714
25. Kadar, K. E. (1995) Protein Prof. 2, 491–499
26. Piez, K. A. (1984) in Extracellular Matrix Biochemistry (Piez, K. A., and Reddi, A. H., eds) pp. 1–39, Elsevier Science Publishing Co., Inc., New York
27. Chapman, J. A. (1974) Connect. Tissue Res. 2, 137–150
28. Scott, J. E., and Orford, C. R. (1981) Biochemistry 20, 213–216
29. Yu, L., Cummings, C., Sheehan, J. K., Kadar, K. E., Holmes, D. F., and Chapman, J. A. (1993) in Dermatan Sulphate Proteoglycans (Scott, J. E., ed) pp. 183–192, Pudoc Press, London
30. Pringle, G. A., and Dodd, C. M. (1990) J. Histochem. Cytochem. 38, 1405–1411
31. Fleischhauer, R., Fisher, L. W., MacDonald, E. D., Jacobs, L., Jr., Perls, J. S., and Termine, J. D. (1991) J. Struct. Biol. 106, 82–90

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