Structural and Functional Comparisons of Chicken and Human Cellular Fibronectins*

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We have investigated the structural and functional differences between chicken and human cellular fibronectin (i) by comparing the tryptic peptide patterns using sodium dodecyl sulfate-polyacrylamide gel electrophoresis and (ii) by analyzing the binding properties of isolated trypsin-resistant polypeptide fragments. Although the overall functional organization of chicken and human cellular fibronectin was similar, the tryptic patterns obtained from these two molecules were strikingly different. For example, the tryptic digest of chicken cellular fibronectin contained two unique peptide fragments having molecular sizes of 45 and 70 kilodaltons. The previously unidentified carboxyl-terminal 45-kDa fragment is an intermediate that appears between 15 to 120 s of digestion. The 70-kDa fragment binds to gelatin, to fibrin (with unusually high apparent affinity), to heparin (at low ionic strength), and to fixed Staphylococcus aureus cells; it also contains an acceptor site for factor XIII, (plasma transglutaminase).

These results suggest that the functional domains of chicken and human fibronectins remain constant and that structural variations occur in the protease-susceptible regions of the molecule. The present findings are discussed in terms of the previously existing discrepancies in the literature on fibronectin.

KDa fragment is held together by disulfide bonding near their carboxyl termini.

Fibronectins are adhesive high molecular weight glycoproteins found on the surface of most cells, as components of extracellular matrices, and as constituents of plasma (1–8). They have been found in most invertebrates and vertebrates tested (9, 10) and have been shown to be involved in a variety of cell functions including cell-substratum and cell-cell adhesion, motility, and phagocytosis (1–9). These biological activities are related to the specific binding of fibronectin to a series of macromolecules including collagen, glycosaminoglycans, and fibrin, and to the bacterium Staphylococcus aureus (2–9). The various binding activities have been assigned to specific protease-resistant domains or regions of the fibronectin molecule, which consists of two similar or identical subunits of molecular size 220 ± 20 kDa† held together by disulfide bonding near their carboxyl termini.

Plasma and cellular fibronectins are not identical (11–15), yet they show very similar immunological, ligand-binding, and biological properties (7, 11, 16, 17). One area where these two forms of fibronectin do show the greatest difference is in biological properties involving cell-cell adhesion. This distinction is best demonstrated in assays for hemagglutination and for restoration of a more normal fibroblastic morphology to transformed cells (2, 11).

Fibronectins isolated from different species can also show very similar physical, immunological, and biological properties. Chicken cellular fibronectin has hydrodynamic properties which vary only slightly from those of human plasma fibronectin (18, 19). Moreover, antibodies elicited against human fibronectin can cross-react with fibronectins from different species (10, 20) such as newts (21), fish (22), sea urchin (23), and sponges (10, 24). Also, fibronectin from human plasma is incorporated into the extracellular matrix of mouse tissue (25). The apparent absence of species specificity in these studies may have been the result of examining only gross structural aspects of the different fibronectins. Several investigators, using more sensitive probes, have recently succeeded in detecting possible species-specific differences. For instance, differences have been detected by two-dimensional peptide mapping of surface-iodinated fibronectins (26, 27), monoclonal (28, 29) and polyclonal antibody reactivities, and detailed analyses of fragments generated by proteolytic enzymes (16, 30).

There are a number of apparent discrepancies in the literature that might be the result of species differences among fibronectins. For example, chicken cellular fibronectin was initially claimed to be cleaved at a site removed from the interchain disulfide bond, creating a disulfide-linked species larger than the monomer (31). In another study, it was not possible to definitively map the actin-binding domain because the origin of this major 70-kDa fragment was not entirely clear (17). These results have been difficult to reconcile with the present structural map of human fibronectin.

We report here the finding of species-specific structural differences distributed throughout the entire lengths of chicken and human cellular fibronectins. Fibronectins from

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The abbreviations used are: kDa, kilodaltons; SDS, sodium dodecyl sulfate.

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these two sources vary with respect to the number and size of tryptic fragments generated, suggesting that differences exist in the primary structure of the proteins. We also identify a 70-kDa tryptic fragment at the amino terminus of chicken fibronectin that contains a high affinity fibrin-binding site and is a substrate for factor XIII (plasma transglutaminase).

**EXPERIMENTAL PROCEDURES**

Chicken cellular fibronectin was isolated from confluent tertiary cultures of 10-day-old chicken embryo fibroblasts by the urea extraction procedure described previously (32). Human cellular fibronectin (obtained from Bethesda Research Laboratories) was extracted from confluent monolayers of human foreskin fibroblasts (passage five) using 2 M urea as described (32) and then further purified on a gelatin-Sepharose affinity column.

Radiolabeled fibronectin was obtained by incubating the chicken embryo fibroblast cultures with [U-14C]leucine (5 μCi/ml, specific activity 353 mCi/mmol) or [3S]cysteine (100 μCi/ml, 394 Ci/mmol) for 24 h prior to extraction.

Trypsin digestion of the fibronectins was performed in 0.1 M NaCl, 10 mM CaCl₂, and 50 mM Tris-HCl, pH 7.0, buffer at 30 °C as previously described (33) using the enzyme-substrate ratios and digestion times indicated in the figure legends. Fibronectin heparin-, fibrin-, gelatin-, and Staphylococcus aureus-binding fragments were isolated as described previously (33).

Factor XIII-catalyzed incorporation of [3H]putrescine was performed essentially as described by Mosher et al. (34). One hundred micromolars of fibronectin were dissolved in 200 μl of 50 mM Tris (pH 7.0) containing 10 mM CaCl₂, 100 mM NaCl, 0.5 mM putrescine, 6 μM [2,3-3H(N)] putrescine (39 Ci/mmol), and 50 μg/ml of factor XIII. The reaction was started by adding thrombin to a concentration of 1 unit/ml, incubated at 37 °C for 90 min, and stopped by adding EDTA to a final concentration of 20 mM.

SDS-polyacrylamide gel electrophoresis and fluorography were performed as previously described (16), and peptides in the gels were visualized by Coomassie Blue staining. The results shown in Fig. 2 indicate that the mobilities of both the 34- and the 31-kDa fragments are significantly altered by chemical reduction, which strongly suggests the presence of internal disulfide bonding in these fragments, as previously reported for human plasma fibronectin (37–39). In contrast, the electrophoretic mobilities of the comparision of lanes 3 and 5 with lanes 7 and 9, it is apparent that the mobilities of both the 34- and the 31-kDa fragments are significantly altered by chemical reduction, which strongly suggests the presence of internal disulfide bonding in these fragments, as previously reported for human plasma fibronectin (37–39). In contrast, the electrophoretic mobilities of the

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**FIG. 1. SDS-polyacrylamide gel electrophoresis of human and chick cellular fibronectins.** Human (H) and chicken (C) cellular fibronectin (0.5 mg/ml) were compared by electrophoresis on a 4/7.5/12% SDS-polyacrylamide composite gel (see Ref. 14) without (A) and with (B) reduction with 0.1 M dithiothreitol (DTT). The lanes S represent molecular weight standards.

**FIG. 2. SDS-polyacrylamide gel electrophoresis of the major tryptic fragments of human and chicken cellular fibronectins.** Human (H, lane 1) and chicken (C, lane 2) cellular fibronectin (0.5 mg/ml) were digested by trypsin at an enzymesubstrate ratio of 1:100 for 15 min (lanes 3, 4, 7, and 8) and 30 min (lanes 5, 6, 9, and 10) at 30 °C. The 1-lane fragments (20 μl) were separated by electrophoresis on the same 8% SDS-polyacrylamide gel with (lanes 1–6) or without (lanes 7–10) reduction by 0.1 M dithiothreitol (DTT). The proteins were visualized by Coomassie Blue staining. The numbers at the left indicate the relative molecular sizes in kilodaltons of protein standards.
large fragments (Fig. 2, lanes 3, 5, 7, and 9) are not significantly affected by the presence or absence of reducing agent. This is consistent with published observations that one of the earliest tryptic cleavages must separate the domain containing the interchain disulfide bond(s) from the remainder of the molecule (16, 36, 37, 39).

When chicken cellular fibronectin was analyzed in an identical manner, a strikingly different tryptic peptide pattern was obtained. Six major polypeptide fragments were detected after incubation with trypsin for 15 min with apparent molecular sizes of 210, 180, 160, 140, 70, and 24 kDa (Fig. 2, lane 4). The relative amounts of the high molecular size fragments tend to decrease with longer incubation times, while the 70- and 24-kDa fragments appear to be particularly resistant to further digestion by trypsin (Fig. 2, lanes 4 and 6). Also, the electrophoretic mobilities of the 70- and 24-kDa fragments are significantly lower after reduction with 0.1 M dithiothreitol (Fig. 2, compare lanes 4 and 6 with lanes 8 and 10). This finding strongly suggests the presence of internal disulfide bonds as described earlier for the collagen-binding region of human fibronectin (39, 43). The differences we observed between human and chicken fibronectins cannot be attributed to different methods of purification. The pattern of tryptic fragments was essentially unaltered when the chicken fibronectin is extracted with 2 M urea and then further purified on a gelatin-Sepharose affinity column followed by 4 M urea elution (results not shown).

To examine the origin of the high molecular weight polypeptides identified in the tryptic digest of chicken cellular fibronectin, the protein was digested with trypsin at an enzyme substrate ratio of 1:1000 (w/w) for brief intervals (see legend to Fig. 3). Under these conditions, the primary cleavage appears to occur near the carboxyl terminus, generating 210-kDa monomers which are no longer linked by interchain disulfide bonds (Fig. 3, compare lane 2 in A and lane 2 in B). This 210-kDa fragment is the earliest major cleavage product observed under these conditions. Within 1 min, three other polypeptide fragments of molecular sizes 180, 45, and 30 kDa appear, and after 2 min of digestion, fragments having molecular sizes of 160, 140, 95, 70, and 24 kDa also become visible (Fig. 3B). After 5 min, the amount of 30-kDa fragment appears to be significantly reduced, accompanied by a concomitant increase in the amount of the 24-kDa fragment (Fig. 3B, lane 8).

It is apparent from examination of the digestion pattern at early times (Fig. 3) that one additional unusual cleavage can also occur. As early as 15 s and throughout the first 2 min of incubation, a 45-kDa fragment is detected only in the reduced gel (Fig. 3B), and a 270-kDa fragment is detected in only the nonreduced gel (Fig. 3A). The relative intensity of the band corresponding to intact monomer in Fig. 3B diminishes at the same rate as the 270-kDa band in A. In addition, the disappearance of the 45-kDa band coincides with the appearance of the 30- and 24-kDa fragments. These data are consistent with a first cleavage occurring in only one of the two monomer chains, 45 kDa from the carboxyl terminus (termed "asymmetric" cleavage). This 45-kDa fragment is subsequently cleaved, possibly generating the 30-kDa and eventually the 24-kDa fragments.

To summarize, the earliest major tryptic digestion products from chicken cellular fibronectin are (i) 30- and 180-kDa fragments from the "symmetric" cleavage and (ii) 45- and 180-kDa fragments from the "asymmetric" cleavage. The 24-kDa fragment always appears subsequent to the 45- and 30-

![Figure 3](image-url)

**Fig. 3.** Time course of trypsin digestion of chicken cellular fibronectin. Chicken cellular fibronectin (lane 1) (1 mg/ml) was digested by trypsin at an enzyme substrate ratio of 1:1000 for 15 s (lane 2), 30 s (lane 3), 45 s (lane 4), 1 min (lane 5), 2 min (lane 6), 5 min (lane 7), and 12 min (lane 8) at 30 °C. The tryptic fragments were separated by SDS-polyacrylamide gel electrophoresis as described in Fig. 1 without (A) and with (B) reduction by 0.1 M dithiothreitol (DTT). The 270-, 210-, 180-, 70-, 45-, 30-, and 24-kDa fragments are indicated by arrowsheads. Note that the 270-kDa product of the "asymmetric cleavage" (see text) clearly has a slower mobility than the fibronectin monomer in A and is absent from B.
Comparisons of Chicken and Human Cellular Fibronectins

Fig. 4. Two-dimensional tryptic peptide mapping of the 180-, 45-, 30-, and 24-kDa fragments of chicken cellular fibronectin. Fragments generated by tryptic digestion and separated by SDS-polyacrylamide gel electrophoresis were radioiodinated, cleaved exhaustively with trypsin, and peptides were electrophoretically separated and chromatographed on cellulose thin layer plates as described in Ref. 21. A, map of the 180-kDa fragment in Fig. 3B, lane 7; B, map of the 45-kDa fragment in Fig. 3B, lane 7; C, an analysis of A, B, and D (see below) for similarities, where O represents major spots appearing in A, B, and D, • represents spots appearing in A, but not in B or D, ○ represents spots appearing in B or D but not in A, and □ represents minor spots which appear in A, B, and D and cannot be reliably used for the comparison; D, map of the 30-kDa fragment in Fig. 3B, lane 7; E, map of the 24-kDa fragment in Fig. 3B, lane 7; F, an analysis of B, D, and E for similarities where • represents major spots in common among B, D, and E, ○ represents spots which appear in B but in D or E, □ represents spots which appear in D and E but not B, • represents spots which are unique to D, and ○ represents minor spots which cannot reliably be used for comparisons among maps B, D, and E. All spots appearing in E are also present in D.

The peptide map of the 30-kDa fragment contains all the spots seen in the map of the 24-kDa fragment (Fig. 4E) plus additional major spots (see Fig. 4F). This demonstrates that the 24-kDa fragment is derived from further cleavage of the 30-kDa fragment. When comparing the peptide maps of the 45- and 30-kDa fragments (Fig. 4, B and D, respectively), most of their major spots are present in common.

As expected, there are a number of major peptides in the 45-kDa fragment which are not found in the 30-kDa fragment. However, there are several major peptides in the 30-kDa fragment which are absent from the 45-kDa fragment (Fig. 4, B, C, and F). These data suggest that although the 45- and 30-kDa fragments are very closely related, probably coming from the same region of the fibronectin molecule (the carboxyl terminus) and sharing many peptides, the 30-kDa fragment cannot have been generated by further tryptic digestion of the 45-kDa fragment. Possible pathways for the formation of the major tryptic fragments of chick and human fibronectins are shown schematically in Fig. 5. The pathways for the digestion of human fibronectin are based on previously published reports (30, 36, 41).

To further characterize the 70-kDa tryptic fragment unique to chicken cellular fibronectin, we investigated its binding and biological activities with respect to the previously defined properties of specific protease-resistant domains of the molecule (16, 36, 40–47). As shown in Fig. 6A, the 70-kDa fragment is the major gelatin-binding fragment present after 30 min of trypsin digestion of chicken cellular fibronectin at an enzyme:substrate ratio of 1:100. The minor lower molecular weight gelatin-binding fragments appearing in lane 5 of Fig. 6A are probably the result of further tryptic digestion of the 70-kDa fragment. The 70-kDa fragment also binds to heparin at low ionic strength (Fig. 6A) and to S. aureus at physiological ionic strength (Fig. 6B). The 70-kDa fragment is quantita-
The arrows indicate a relationship between two fragments, although the smaller model for human fibronectin is based on previously published reports (see text for references).

The numbers represent the apparent size in kilodaltons of the different fragments. The heavy arrows illustrate the major pathways of fragmentation, the light arrows show the minor pathways, and dashed arrows indicate further processing of fragments. The dotted line indicates a relationship between two fragments, although the smaller fragment is not the result of further proteolysis of the larger. The model for human fibronectin is based on previously published reports (see text for references).

The results shown in Fig. 7 indicate that both the 70- and 24-kDa fragments from chicken fibronectin bind to fibrin. However, the 70-kDa fragment has a higher apparent affinity for fibrin, since 4 M urea is required to elute this fragment from fibrin-Sepharose. The results shown in Fig. 7B indicate that the 31- and 34-kDa tryptic fragments of human fibronectin also bind to fibrin as previously reported (36).

To further localize the 70-kDa tryptic fragment within the fibronectin molecule, the fragment was tested as a substrate for transglutaminase. The results shown in Fig. 8 indicate that [3H]putrescine is incorporated into this fragment. This suggests that the 70-kDa fragment must span the area of the fibrin-binding site, which is indicated in Table I. A schematic model for the placement of the tryptic fragments within the primary structure of the fibronectin monomer is shown in Fig. 9 for both chicken and human cellular fibronectins based on data presented both here and elsewhere (16, 30, 36, 40-47).
The stable 70-kDa tryptic fragment appears to be unique to chicken fibronectin, as it has not been observed in tryptic digests of human fibronectin (36) or hamster fibronectin (50). It is, however, possible to generate a similar fragment from human fibronectin with cathepsin D, although this enzyme is substantially less effective than trypsin in cleaving fibronectin (39, 50). The 70-kDa fragment from chicken binds to gelatin, heparin, fibrin, and S. aureus. Furthermore, this fragment contains a transglutaminase acceptor site, as judged by [3H]putrescine incorporation. These data are consistent with assignment of this fragment to the氨基terminus of the molecule for the following reasons: (i) the binding of fibrin is of high affinity and the binding of heparin is of low affinity as is found to be the case with the amino-terminal domain of human fibronectin (36, 41); (ii) the S. aureus-binding region is near the amino terminus of human plasma fibronectin (34); and (iii) the transglutaminase acceptor site is very close to the amino-terminal amino acid. In the case of bovine fibronectin, it is only three amino acids from the amino terminus (51, 52), and there is substantial amino acid homology between chicken cellular and bovine plasma fibronectins (52, 53). The 70-kDa fragment also contains the gelatin-binding site and is extensively disulfide-bonded, as has been previously described (37–39).

In the case of human cellular fibronectin, the amino-terminal domain is the 31-kDa fragment which interacts with factor XIII, and binds to S. aureus, heparin, and fibrin (17, 34, 36, 37, 41, 45). The 34-kDa fragment binds to fibrin (36) and the 170-kDa fragment binds to gelatin and cells (36, 37). Although these activities were largely characterized using fragments generated from human plasma fibronectin, the tryptic digestion patterns of human cellular and plasma fibronectins are very similar, though not completely identical.

The present studies also indicate that the chicken cellular fibronectin dimer can be cleaved asymmetrically by trypsin. One possible explanation for the finding of an asymmetric tryptic cleavage is that cellular fibronectin may be composed of two nonidentical chains. This characteristic has never been directly observed in mature cellular fibronectin, possibly because the separation of the monomers on polyacrylamide gels is obscured by band spreading caused by glycosylation heterogeneity. Such an explanation is also consistent with the results of some of the peptide-mapping experiments presented here. Earlier work has identified a similar asymmetric cleavage in human plasma fibronectin using cathepsin D (50, 54), but since plasma fibronectin is composed of two chains of distinctly different sizes (cf. Refs. 2–8), it is not surprising that two chains show different susceptibilities to proteolysis. Another possibility is that small subpopulations of chicken fibroblasts or even of messenger RNAs within a cell produce fibronectins which are subtly different near their carboxyl termini.

The peptide maps demonstrate that chicken cellular fibronectin is cleaved by trypsin into a 180-kDa fragment and either a 30-kDa fragment via symmetric cleavage of both subunits of the dimer or a 45-kDa fragment when the two chains of the dimer are cleaved asymmetrically. The peptide maps show that two major peptides in common between either the 45- or the 30-kDa fragment and the 180-kDa fragment. Surprisingly, the peptide maps show that the 45-kDa fragment cannot be further digested to eventually yield the 30-kDa fragment, even though these two share most of their peptides in common and are very closely related. The
simplest explanation for this finding is that the 30-kDa fragment extends past the amino terminus of the 45-kDa fragment, and the 45-kDa fragment extends past the carboxyl terminus of the 30-kDa fragment. The results of the peptide mapping of these fragments are also consistent with the presence of two slightly different sized monomer units comprising the fibronectin dimer.

The peptide maps also show that the 24-kDa fragment is generated by further cleavage of the 30-kDa fragment. There are no major peptides found in the 24-kDa fragment not also found in the 30-kDa fragment. The peptide maps also show that the 24-kDa fragment is susceptible interdomain regions. The presence of two slightly different sized monomer units com-


differences between chicken and human fibronectins, but it is impossible to rule out conformational differences which could cause altered proteolysis patterns in these two proteins. Resolution of this question would require extensive amino acid sequencing of the fibronectins.

Our present model for the placement of the protease-resistant binding domains of both chick and human cellular fibronectins is summarized in Fig. 9. All the functional domains in human fibronectin are present in the same relative positions in chick fibronectin. This finding accounts for the similar biological and physical properties of these two molecules. The major differences between chick and human fibronectins appear to be preferentially localized to the protease-susceptible interdomain regions.

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REFERENCES

1. Hysey, H. O. (1976) Biochim. Biophys. Acta 458, 73-107
2. Yamada, K. M., and Olden, K. (1976) Nature (Lond.) 275, 179-184
3. Mosher, D. F. (1980) Proc. Natl. Acad. Sci. 77, 1111-1115
4. Mosesson, M. W., and Honn, H. (1980) Blood 56, 145-158
5. Pearlstein, E., Gold, L. I., and Garcia-Fordo, A. (1980) Mol. Cell. Biochem. 50, 129-138
6. Ruscabbi, E., Envall, E., and Hayman, E. G. (1981) Collagen Res. Rev. 1, 95-129
7. Akiyama, S. K., Yamada, K. M., and Hayashi, M. (1981) J. Supramol. Struct. 18, 345-358
8. Hysey, H. O., and Yamada, K. M. (1982) J. Cell Biol. 95, 399-377
9. Akiyama, S. K., and Yamada, K. M. (1983) in Connective Tissue Diseases (Fincham, R. S., Wanstler, B., and Kaufman, N., eds) pp. 55-96, Williams & Wilkins Co., Baltimore, MD
10. Akiyama, S. K., and Johnson, M. D. (1983) Comp. Biochem. Physiol. 70B, 687-694
11. Yamada, K. M., and Kennedy, D. W. (1979) J. Cell Biol. 80, 492-498
12. Fukuda, M., Lavery, S. B., and Hakomori, S. (1982) J. Biol. Chem. 257, 6856-6860
13. Fukuda, M., and Hakomori, S. (1979) J. Biol. Chem. 254, 5401-5407
14. Atherton, B. T., and Hynes, R. O. (1981) Cell 25, 133-141
15. Atherton, B. T., Taylor, J. M., and Hynes, R. O. (1981) J. Supramol. Struct. 19, 153-161
16. Hayashi, M., and Yamada, K. M. (1981) J. Biol. Chem. 256, 1129-1300
17. Kesk-Osa, J., and Yamada, K. M. (1981) Biochem. J. 193, 515-520
18. Alexander, S. S., Jr., Colonne, G., and Edelhoch, H. (1979) J. Biol. Chem. 254, 1901-1905
19. Sekiguchi, J., and Hakomori, S. (1983) J. Biol. Chem. 258, 3893-3897
20. Kruusta, P., Ruscabbi, E., Envall, E., and Vaheri, A. (1976) Immunochim. 13, 69-82
21. Naiti, P. G., Galloway, D., Nicotra, M. R., and De Martino, C. (1981) Connect Tissue Res. 199, 149-200
22. Spiegel, E., Burger, M., and Spiegel, M. (1980) J. Cell Biol. 87, 309-313
23. Labat-Robert, J., Robert, L., Auger, C., and Lethias, C. (1981) Proc. Natl. Acad. Sci. U. S. A. 78, 2621-2625
24. Oh, E., Fierensbacher, M., and Ruscabbi, E. (1981) Proc. Natl. Acad. Sci. U. S. A. 78, 2318-2322
25. Nairn, R., and Hughes, R. C. (1976) Biochim. Biophys. Acta 458, 165-167
26. Hughes, R. C., and Nairn, R. (1979) Ann. N. Y. Acad. Sci. 312, 192-206
27. Kotaniyagi, Y. E., Arensya, E. B., Bregev, T. G., Chernousov, M. A., Glikzyova, M. A., Ishiyama, A., Metz, M. L., Petrovski, M. N., and Rokhlin, O. V. (1982) FEBS Lett. 142, 199-202
28. Schoon, R. C., Bentley, K. L., and Kleibe, R. J. (1982) Hybridon J. 99, 1-99
29. Sekiguchi, J., and Hakomori, S. (1983) J. Biol. Chem. 258, 3893-3897
30. Yamada, K. M., Olden, K., and Pastan, I. (1978) Anu. N. Y. Acad. Sci. 312, 256-277
31. Yamada, K. M. (1982) in Immunochemistry of the Extracellular Matrix (Furnugly, H., ed) Vol. 1, pp. 111-1122, CRC Press, Boca Raton, FL
32. Bernard, B. A., Yamada, K. M., and Olden, K. (1982) J. Biol. Chem. 257, 8555-8554
33. Mesler, D. F., Schad, P. E., and Vann, J. M. (1982) Hybridon J. 1, 107-108
34. Ruscabbi, E., Hayman, E. G., Keusela, P., Shively, J. E., and Envall, E. (1980) J. Biol. Chem. 255, 6554-6554
35. Balan, G., Click, E. M., and Borrstein, P. (1980) J. Biol. Chem. 255, 3234-3238
36. Fure, M. B., Frey, A. B., and Rikfik, D. B. (1980) J. Biol. Chem. 255, 4391-4394
37. Hörnman, H., and Seidl, M. (1980) Hoppe-Seyler's Z. Physiol. Chem. 361, 1429-1434
38. Hahn, L. E., and Yamada, K. M. (1979) Proc. Natl. Acad. Sci. U. S. A. 76, 1160-1163
39. Hahn, L. E., and Yamada, K. M. (1979) Cell 18, 1043-1051
40. Yamada, K. M., Schlesinger, D. H., Kennedy, D. W., and Pastan, I. (1977) J. Biol. Chem. 252, 1332-1340
41. Takasaki, S., Yamashita, K., Suzuki, K., and Kobayashi, A. (1980) J. Biochem. (Tokyo) 88, 1567-1574
42. Richter, H., and Horsmann, H. (1982) Hoppe-Seyler's Z. Physiol. Chem. 363, 351-364
43. McDonagh, R. P., McDonagh, J., Petersen, T. E., Thogersen, H. C., Skorstengard, K., Sottorp-Jensen, L., and Magnusson, S. (1981) FEBS Lett. 127, 174-178
44. Petersen, T., Thogersen, H. C., Skorstengard, K., Vibe-Petersen, K., Sahl, P., Sottorp-Jensen, L., and Magnusson, S. (1983) Proc. Natl. Acad. Sci. U. S. A. 80, 127-131
45. Hirano, H., Yamada, Y., Sullivan, M., de Crombrugghe, B., Pastan, I., and Yamada, K. M. (1983) Proc. Natl. Acad. Sci. U. S. A. 80, 46-56
46. Richter, H., Seidl, M., and Hornman, H. (1981) Hoppe-Seyler's Z. Physiol. Chem. 362, 399-406
47. Courtoy, Y., and Hughes, R. C. (1976) Gerontology 22, 371-379

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