Fibrin is formed at sites of tissue injury and provides the temporary matrix needed to support the initial endothelial cell responses needed for vessel repair. Basic fibroblast growth factor (bFGF) also acts at sites of injury and stimulates similar vascular cell responses. We have, therefore, investigated whether there are specific interactions between bFGF and fibrinogen and fibrin that could play a role in coordinating these responses. Binding studies were performed using bFGF immobilized on Sepharose beads and soluble 125I-labeled fibrinogen and also using Sepharose-immobilized fibrinogen and soluble 125I-bFGF. Both systems demonstrated specific and saturable binding. Scatchard analysis indicated two classes of binding sites for each with \( K_d \) values of 1.3 and 260 nM using immobilized bFGF; and \( K_d \) values of 0.9 and 70 nM using immobilized fibrinogen. After conversion of Sepharose-immobilized fibrinogen to fibrin by treatment with thrombin, bFGF also demonstrated specific and saturable binding with two classes of binding sites having \( K_d \) values of 0.13 and 83 nM. Fibrin binding was also investigated by cloting a solution of bFGF and fibrinogen, and two classes of binding sites were demonstrated using this system with \( K_d \) values of 0.8 and 261 nM. The maximum molar binding ratios of bFGF to fibrinogen were between 2.0 and 4.0 with the four binding systems. We conclude that bFGF binds specifically and saturably to fibrinogen and fibrin with high affinity, and this may have implications regarding the localization of its effect at sites of tissue injury.

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† The abbreviations used are: bFGF, basic fibroblast growth factor; FGFR, FGF receptor.
support with active N-hydroxy succinimide esters in 0.1 M sodium phosphate buffer, pH 7.4, containing 0.5 M NaCl and gently mixed at 25 °C for 2 h, and over 97% of antibody was bound to the beads. Residual active ester sites were then blocked by the addition of 1 M ethanolamine, pH 8.0, and the suspension was washed several times with 0.1 M sodium phosphate buffer, pH 7.4, containing 0.5 M NaCl. bFGF (50 µg/ml) was then added to this suspension and gently mixed at 25 °C for 1 h, following which the unbound bFGF was removed by washing with 0.1 M sodium phosphate buffer, pH 7.4, containing 0.25 M NaCl. The amount of bFGF immobilized on the beads was 24.3 µg/ml as determined by enzyme-linked immunosorbent assay. For binding studies, 125I-fibrinogen at concentrations from 0.15 to 300 nM was incubated at 37 °C with a 0.02 ml suspension of immobilized bFGF in a final volume of 0.1 ml. Nonspecific binding was determined in parallel experiments using a 20-fold molar excess of unlabeled fibrinogen. Preliminary experiments demonstrated maximum specific binding after a 30 min incubation in 0.1 M sodium phosphate buffer, pH 7.4, containing 0.25 M NaCl, and these conditions were used for all subsequent experiments. Following incubation, the beads were separated by centrifugation at 3,000 × g for 10 min after which the supernatant was removed, and the beads were then washed rapidly twice with 0.1 M sodium phosphate buffer, pH 7.4, containing 0.5 M NaCl at 4 °C to minimize nonspecific association. The amount of bound fibrinogen was calculated from the radioactivity associated with the beads.

To characterize the protein that was bound to immobilized bFGF, 125I-fibrinogen (1 mg/ml) was passed through a column of immobilized bFGF, and unbound radioactivity was eluted with 0.1 M sodium phosphate buffer, pH 7.4, containing 0.25 M NaCl. Bound protein was then eluted by the addition of 2 mg/ml unlabeled fibrinogen or ovalbumin as a control, and aliquots of 200 µl were collected and counted. Aliquots of selected fractions were electrophoresed on sodium dodecyl sulfate 7% polyacrylamide gels after disulfide bond reduction, dried, and used to prepare autoradiograms.

Binding of 125I-bFGF to Fibrinogen and Fibrin Monomer—A similar approach was used with incubation of 125I-bFGF with fibrinogen or fibrin monomer immobilized on Sepharose beads. Affi-gel 15 beads were first incubated with purified monoclonal antibody J88B (1 mg/ml) in 0.2 M sodium bicarbonate buffer, pH 8.3, and gently mixed at 25 °C for 2 h. Residual sites were blocked by incubation in 1 X ethanolamine, pH 8.0, and the suspension was washed several times with 0.2 M sodium bicarbonate buffer, pH 8.3, containing 0.5 M NaCl. Gel containing bound antibody was incubated with fibrinogen in sodium phosphate buffer, pH 7.4, containing 0.25 M NaCl and then incubated at 25 °C with gentle mixing for 1 h. After this, the beads were washed with 0.1 M sodium phosphate buffer, pH 7.4, containing 0.25 M NaCl to remove unbound fibrinogen. This was continued until no further fibrinogen was removed as determined by monitoring the optical density at 280 nm. To convert bound fibrinogen to fibrin monomer, beads were incubated with 0.5 units/ml of thrombin at 37 °C for 90 min. Characterization of binding of 125I-bFGF to fibrinogen and fibrin monomer was performed in the same way as 125I-fibrinogen binding to immobilized bFGF (see above). 125I-bFGF at concentrations from 0.05 to 100 nM was incubated with 0.02 ml suspension of beads containing 0.1 µg of fibrinogen or fibrin in a final volume of 0.1 ml. Nonspecific binding was determined in parallel experiments using a 100-fold molar excess of unlabeled bFGF. Specificity of the binding of bFGF to fibrinogen was confirmed by competition experiments in which 0.2 nM of 125I-bFGF was incubated with 1 µg/ml immobilized fibrinogen and fibrin monomer was performed in the same way as 125I-fibrinogen binding to immobilized bFGF (see above). 125I-bFGF at concentrations from 0.05 to 100 nM was added to 100 µg/ml fibrinogen in 0.1 M Tris buffer containing 0.25 M NaCl. Thrombin was then added to a final concentration of 0.5 units/ml, which resulted in clotting of the solution. Following incubation at 37 °C for 30 min, the clot and supernatant were separated by vacuum filtration using GF/C glass microfiber filters (Sigma) previously soaked overnight in a solution of 0.5% polyvinylpyrrolidone and 0.1% Tween 20 to reduce nonspecific binding. The clot on the filter was washed quickly with cold 0.1 M Tris buffer containing 0.25 M NaCl, and the associated radiolabel was measured. Nonspecific binding was determined by parallel experiments incorporating a 100-fold molar excess of unlabeled bFGF.

**Data Analysis**—Unless indicated otherwise data is expressed as mean ± S.D. Scatchard analysis of the data was performed using the Ligand program (18) from Biosoft (Ferguson, MO).

**RESULTS**

Binding of fibrinogen to immobilized bFGF was saturable and specific with nonspecific binding representing less than 20% of the total (Fig. 1A). Saturation of specific binding occurred at a fibrinogen concentration of 150 nM, and only an increase in nonspecific binding was observed at higher concentrations. In control experiments there was a maximum of 3% binding of 125I-fibrinogen over the same range of concentrations to beads with immobilized anti-bFGF immunoglobulin only or to beads with no protein bound and active sites blocked with ethanolamine. A plot of bound versus bound/free fibrinogen (Fig. 1B) was nonlinear, suggesting the presence of more than one binding site. This was confirmed by Scatchard analysis, which indicated that binding was best described by a two-site model with apparent Kₐ values of 1.3 and 260 nM (Table I). Bₐₙₐₓ was 6.3 and 35 nM for the high and low affinity sites, respectively, and the maximum molar binding ratio of bFGF to fibrinogen was 4.0.

To further characterize the protein that bound to bFGF, 125I-fibrinogen was passed over a column of immobilized bFGF. Following washing, the specifically bound protein was eluted with 2 mg/ml unlabeled fibrinogen (Fig. 2), and approximately 90% of bound label rapidly eluted in two fractions. SDS-polyacrylamide gel electrophoresis of the eluted protein showed bands consistent with the Aα, Bβ and γ chains of fibrinogen.
Binding of Basic Fibroblast Growth Factor to Fibrinogen

Table I

| System                       | $K_d1$   | $K_d2$   | $B_{max1}$ | $B_{max2}$ | Maximum molar binding ratio of bFGF/fibrinogen |
|------------------------------|----------|----------|------------|------------|-----------------------------------------------|
| Fibrinogen/bFGF-Sepharose    | 1.3 ± 0.1| 260 ± 64 | 6.3 ± 3.1  | 35 ± 2.1   | 4.0 ± 0.5                                     |
| bFGF/fibrinogen-Sepharose    | 0.9 ± 0.1| 70 ± 12  | 0.3 ± 0.2  | 5.9 ± 0.7  | 2.0 ± 0.5                                     |
| bFGF/fibrin-Sepharose        | 0.13 ± 0.1| 83 ± 24  | 0.03 ± 0.1 | 5.9 ± 1.4  | 2.0 ± 0.9                                     |
| bFGF/fibrin clot             | 0.8 ± 0.7| 261 ± 70 | 0.5 ± 0.3  | 60 ± 22    | 2.0 ± 0.8                                     |

FIG. 2. Elution of bound protein from immobilized bFGF. $^{125}$I-Fibrinogen (1.0 mg/ml) was passed through a 1-ml column of Sepharose-immobilized bFGF. Following washing, the column was eluted with 2 mg/ml unlabeled fibrinogen, and fractions of 200 μl were collected. Approximately 90% of the bound radioactivity eluted in fractions 5 and 6 (1.0–1.2 ml elution volume). These fractions were pooled, and an aliquot was electrophoresed on a 7% SDS-polyacrylamide gel electrophoresis gel and used to prepare autoradiograms (inset). The polypeptide chain pattern in the eluted pool showed αα, ββ, and γ chains of fibrinogen and was similar to that in the starting material. (Fig. 2, inset) establishing that the bound protein was fibrinogen and not a minor contaminant. In control experiments, less than 5% of bound radioactivity was eluted from the column with 2.0 mg/ml ovalbumin, demonstrating specificity of the elution.

The association of bFGF and fibrinogen was also characterized using soluble $^{125}$I-radiolabeled bFGF and fibrinogen immobilized on Sepharose beads (Fig. 3). With this system, saturable and specific binding was also observed, and nonspecific binding represented 20% or less of the total. Saturation of specific binding was observed at a bFGF concentration of approximately 75 nM. Scatchard analysis (Fig. 3B) indicated the presence of two binding sites of different affinities with apparent $K_d$ values of 0.9 and 70 nM and a maximum molar binding ratio of 2.0 (Table I) as compared with 4.0 with radiolabeled fibrinogen binding to Sepharose-immobilized bFGF (Fig. 1 and Table I). Competitive inhibition of the binding was performed to further characterize the specificity and the degree of nonspecific association. $^{125}$I-bFGF at a concentration of 0.2 nM was incubated with Sepharose-immobilized fibrinogen and then varying concentrations of unlabeled bFGF was added. The binding of $^{125}$I-bFGF to fibrinogen was reduced in a dose-dependent manner with increasing concentrations of unlabeled bFGF (Fig. 3C), but 28% of the radiolabel remained at 100 nM bFGF, which we interpreted as nonspecific binding.

Fibrinogen is converted to fibrin by thrombin, which cleaves fibrinopeptides A and B from the αα and ββ chains respectively, forming fibrin monomer, which can then polymerize to form a branching network of fibers. To characterize the association of bFGF with fibrin in the absence of polymerization, we incubated Sepharose-immobilized fibrinogen with thrombin. The antibody-mediated immobilization of fibrinogen to the Sepharose beads prevents or limits association of the resulting fibrin, forming a surface with immobilized fibrin monomer. Binding of bFGF to fibrin (Fig. 4A) was similar to that seen for fibrinogen using the same system with specific binding approaching saturation between 75 and 100 nM bFGF (Fig. 4A). Nonspecific binding was low at bFGF concentrations below 15 nM and increased at higher concentrations. In contrast to the binding seen with fibrinogen, the bFGF binding curve at concentrations below 1 nM bFGF suggested the presence of a high affinity binding site of low capacity. This was confirmed by Scatchard analysis (Fig. 4B), indicating the presence of two binding sites with apparent $K_d$ values of 0.13 and 83 nM (Table I).

Characterization of binding to polymerized fibrin presents technical and interpretive problems because of transport of bFGF into the gel is limited, and access to potential binding sites within individual fibrin fibers may also be restricted. We chose, therefore, to add $^{125}$I-bFGF to a solution of fibrinogen, which was then clotted by the addition of thrombin to avoid problems of transport of bFGF into the gel. Total binding was measured with this clotting system in the absence of an unlabeled competitor, whereas nonspecific binding was measured in the presence of 100-fold molar access of unlabeled bFGF (Fig. 5). Nonspecific binding represented up to 40% of the total (Fig. 5A). This was higher than that seen with binding to fibrinogen (Figs. 1 and 3) or to fibrin monomer (Fig. 4), possibly reflecting some entrapment of radiolabel within the fibrin gel. A plot of bound versus bound-free $^{125}$I-bFGF was nonlinear (Fig. 5B), and Scatchard analysis identified two distinct binding sites with apparent $K_d$ values of 0.8 and 261 nM, similar to those for fibrinogen (Table I). The maximum molar binding ratio of bFGF to fibrin was 2.0.

DISCUSSION

The results presented demonstrate that bFGF binds specifically and saturably to fibrinogen and fibrin. Two different experimental systems were used to characterize the association with either bFGF or fibrinogen immobilized on Sepharose beads. The results were similar, with both systems identifying high affinity binding sites with $K_d$ values of 1.3 and 0.9 nM and lower affinity sites with $K_d$ values of 260 and 70 nM. The association of bFGF with fibrin was also characterized using two systems with either surface-immobilized fibrin or polymerized fibrin. The results of binding to fibrin were similar to those found using fibrinogen with two distinct binding sites. The $K_d$ values for the high and low affinity sites were 0.13 and 0.8 nM and 83 and 261 nM for surface-immobilized and -polymerized fibrin, respectively.

The maximum molar binding ratios for bFGF to fibrinogen or fibrin were between 2.0 and 4.0 with the different systems used. Considering that fibrinogen is a dimerically symmetric molecule (19) and that two binding sites with different $K_d$ values were identified, the ratio of 4 bFGF to 1 fibrinogen would be expected and consistent with the presence of two structurally distinct and independent sites on each half-mole-
The expected ratio of 4 was, however, only found using a system in which fibrinogen bound to immobilized bFGF, whereas the ratio was lower using the other three approaches. One potential explanation for the lower binding ratio is that the access of bFGF to potential binding sites was limited. This would be reasonable with polymerized fibrin, as sites in both the D and E domains are involved in the reciprocal binding required for polymerization (20). This could prevent concurrent binding of bFGF to any sites in close proximity or to those otherwise affected by polymerization. A similar explanation could explain reduced binding of bFGF to Sepharose-immobilized fibrinogen or fibrin if the binding site was either close to that recognized by the antibody J88B or altered by antibody binding. An alternative explanation for the lower than expected binding ratio is that one of the binding sites is present on only a minor variant of fibrinogen. There are several such fibrinogen variants including those due to heterogeneity at the carboxyl terminus of the \( \gamma \) chain (21, 22) or to variations in serine phosphorylation. These sites are known to be important in the molecular interactions and function of fibrinogen and fibrin as the \( \gamma \) chain site is involved both in binding to platelets (23) and in factor XIII cross-linking (24), and phosphorylation of Ser\(^3\) of the \( \alpha \) chain affects thrombin action (25). This explanation could account for the ratio of 4 found with the system using bFGF immobilized on Sepharose as only those fibrinogen molecules with the high affinity site would bind. By contrast,

**Fig. 3.** A, binding of bFGF to fibrinogen. \( ^{125}\)I-bFGF was incubated with fibrinogen immobilized on Sepharose beads, and the amount of bound protein was determined as radioactivity associated with the beads following centrifugation and washing. Nonspecific binding (squares) was determined in the same way in the presence of a 100-fold molar excess of unlabeled bFGF. Specific binding (triangles) was calculated by subtracting the nonspecific from the total bound (diamonds). Each point represents the mean ± S.D. of three different experiments. B, Scatchard plot. The best fit of the data was determined by analysis with the Ligand program and is most consistent with involvement of two distinct binding sites. C, competitive inhibition of binding. Increasing concentrations of unlabeled bFGF were used to competitively inhibit the binding of \( ^{125}\)I-bFGF to fibrinogen. Each point represents mean ± S.D. of three different experiments.

**Fig. 4.** Binding of bFGF to fibrin monomer. Fibrinogen was immobilized on Sepharose beads and then converted to fibrin monomer by incubation with thrombin. \( ^{125}\)I-bFGF was incubated with immobilized fibrin, and bound and free ligand were then separated by centrifugation. Nonspecific binding (squares) was measured in the presence of a 100-fold molar excess of unlabeled bFGF, and specific binding (triangles) was determined by subtraction of nonspecific from total binding (diamonds). Each point represents the mean ± S.D. of three different experiments. B, Scatchard plot. The best fit of the data was determined using the Ligand program, and the presence of two distinct binding sites was indicated.
two distinct binding sites was indicated. Data was determined using the Ligand program, and the presence of
bFGF and to the availability of other sites for binding within the vasculature. At normal plasma concentrations of fibrinogen (7 μM) and of bFGF (up to 6 μM) nearly all bFGF should be bound to fibrinogen considering the \( K_d \) values in the nanomolar range. However, other bFGF binding proteins, \( \alpha_2 \) macroglobulin (44) and soluble forms of FGF receptor (45), have also been identified in blood. The binding of bFGF to \( \alpha_2 \) macroglobulin involves formation of covalent bonds and is slow, requiring up to 4 h to reach completion (44). Three soluble truncated forms of the high affinity cell receptor FGFR1 have also been identified in plasma as binding proteins for bFGF (45), but neither the plasma concentration nor binding affinities have been described. Further studies will be required to elucidate the distribution of bFGF binding to these plasma proteins and their role in influencing plasma half-life or bFGF activity.

The binding of bFGF with fibrinogen and fibrin may have effects locally at sites of vessel injury or disease. Fibrinogen is found in both normal and atherosclerotic arterial walls (46–48) and could, therefore, serve as a binding site for bFGF within the matrix. This may also occur with fibrin that is also present in atherosclerotic vessels (47) as well as at sites of injury, inflammation, or tumor growth. Binding of bFGF to fibrin could, therefore, localize both molecules to sites where they are needed to support endothelial cell migration, proliferation, and angiogenesis.

The binding of bFGF to fibrinogen and fibrin may also have effects on interactions with cell receptors and with signal transduction. Binding of endothelial cells to matrix glycoproteins through integrin receptors alters their sensitivity to growth factor-induced signaling mechanisms (49). Fibrinogen and fibrin can support endothelial cell attachment through occupancy of \( \alpha_5 \beta_3 \) and the resultant formation of a focal adhesion complex. Studies in vitro demonstrate the co-localization of integrin receptors and the high affinity FGF with multiple signaling molecules within the focal adhesion complex (50).
This organization may foster signal integration between integrins and growth factor receptors. The binding of bFGF to fibronogen and fibrin could facilitate such close association between their separate receptors. Further interactions between bFGF with fibronogen and fibrin in vascular responses may result from the role of bFGF in modulating endothelial cell surface integrin expression (51). The binding of bFGF as described in this work may, therefore, serve to both localize its activity and coordinate with fibronogen and fibrin in supporting angiogenesis and the vascular response to injury.

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Binding of Basic Fibroblast Growth Factor to Fibrinogen and Fibrin
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