Fibronectin Receptors from Staphylococcus aureus*

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Fibronectin which is recognized for its ability to mediate substrate adhesion of eucaryotic cells has also been shown to bind to Staphylococcus aureus (Kuusela, P. (1978) Nature (Lond.) 276, 718-720). A further characterization of the interaction of fibronectin with staphylococci is presented here. The binding of 125I-fibronectin to S. aureus, strain Cowan 1, is specific, time-dependent, functionally irreversible, and occurs to both live and heat-killed cells. Furthermore, staphylococci may be saturated with fibronectin at a level which suggests the presence of 250 receptors/cell. A lysate produced by digestion of staphylococcal cells with a bacteriolytic enzyme (lysostaphin) inhibited the binding of 125I-fibronectin to bacteria. The lysate was depleted of its inhibitory activity by passage through a column of Sepharose substituted with fibronectin. The inhibitory activity was destroyed when the lysate was incubated with trypsin or pronase, and a lysate prepared from trypsin-treated cells did not have inhibitory activity. These data suggest that the inhibitory activity of the lysate is due to solubilized surface proteins acting as receptors for fibronectin. Staphylococcal mutants that selectively had lost protein A or fibronectin receptors could be isolated, which suggests the presence of fibronectin receptors distinctly different from protein A. Externally 125I-labeled proteins from the different mutants were analyzed by affinity chromatography on fibronectin-Sepharose followed by gel electrophoresis. The fibronectin receptor was tentatively identified as a protein with an apparent Mr = 18,000. This component was found in fibronectin-binding strains but was absent in strains deficient in fibronectin receptors.

Fibronectin (cold insoluble globulin) is a glycoprotein of high molecular weight which is found in connective tissue and plasma. It is produced by most mammalian cells in tissue culture, and the main function of this protein in vitro is presumably related to its ability to mediate cell-substrate adhesion in vitro (for recent reviews, see Refs. 2-3). Fibronectin has also been reported to act as an opsonin, mediating phagocytosis of gelatin-coated particles by macrophages (4). These processes probably involve an interaction between fibronectin and specific receptors present on the surface of the eucaryotic cells. Kuusela (5) demonstrated that fibronectin binds to Staphylococcus aureus, while one strain of Mycobacterium kansasii did not bind fibronectin which was taken as an indication of the presence of specific staphylococcal components interacting with fibronectin.

Besides interacting with different types of cells, fibronectin also binds to a number of macromolecules, e.g. collagen (6, 7), fibrin (8), heparin, heparan sulfate, dextran sulfate (9, 10), DNA (11), actin (12), and hyaluronic acid (13). These interactions exhibit different degrees of specificity and strength. Information on the structure of fibronectin is now being gathered by several laboratories. The locations along the molecules of binding sites for cells and for the different macromolecules are being elucidated. For example, a staphylococcal binding site has recently been located close to the NH2-terminus of the protein (14). In contrast, virtually nothing is known about the identity of the cellular structures which are interacting with fibronectin.

S. aureus also binds plasma proteins other than fibronectin, e.g. IgG (15) and fibrinogen (16). Protein A present on the surface of the staphylococci is responsible for the binding of immunoglobulins (15), whereas the surface components involved in fibrinogen binding have not been identified. In the present communication, we report on the isolation of fibronectin receptors from S. aureus.

MATERIALS AND METHODS

Fibronectin was isolated from human plasma by the method described by Vuento and Vaheri (17). Rabbit IgG was purified by serum chromatography on a column of protein A-Sepharose. Proteins were iodinated by the chloramine-T method (18). Chicken egg albumin, trypsin (type III), soybean trypsin inhibitor (type I-S), deoxyribonuclease (type I), ribonuclease (type 1A), lysostaphin, laccoperoxidase, protein A, and sodium β-glycerophosphate were purchased from Sigma Chemical Co, St Louis, MO. Pronase was obtained from Calbiochem, Los Angeles, CA; lysozyme from Miles Laboratories, Elkhart, IN; and trypsin or soy broth from BBL Microbiology Systems, Cockeysville, MD. Fibrinogen was a generous gift from Dr. M.C. Poon, the Veterans Administration Hospital in Birmingham. Percoll and CNBr-activated Sepharose 4B were purchased from Pharmacia Fine Chemicals, Uppsala, Sweden. The method recommended by the manufacturer was used for coupling of proteins to the activated Sepharose. Rabbit antiserum against protein A was kindly provided by Dr. John Sjöquist, Department of Medical Chemistry, University of Uppsala, Sweden. Plasma was obtained from healthy

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donors and depleted of fibronectin by passage through a column of gelatin-Sepharose.

Protein was determined according to Lowry et al. (19) using bovine serum albumin as standard.

Polyacrylamide gel electrophoresis in the presence of SDS was carried out according to Studier (20). The gels consisted of linear gradients of 4% acrylamide, and the gel was 3% acrylamide in 0.25 M Tris-glycine. Samples were dissolved in 0.065 M Tris-HCl, pH 6.8, 2% SDS, 10% glycerol, 0.001% bromophenol blue and heated at 100°C for 5 min. After removal of particulate material by centrifugation at 100,000 g for 1 h, the supernatant (denoted lysate) was stored lyophilized. Before use, the powder was dissolved in 200 ml of 0.145 M NaCl, 0.1% egg albumin. The tubes containing the digested samples. Cells were then incubated at 5°C for 5 min, suspended in PBS, and digested with trypsin (250 units/ml) for 10 min at 100°C, washed once with PBS, and resuspended in PBS.

Digestion of Bacteria with Trypsin—Cells were heat-inactivated for 10 min at 100°C, suspended in PBS, and digested with trypsin (25 mg/ml of bacteria). Indicated times, samples were removed from the incubation mixture and soybean trypsin inhibitor (50 µg/ml) was added to the digested samples. Cells were then incubated at 100°C for 5 min, washed once with PBS, and reconstituted in PBS.

Solubilization of Bacteria—Bacteria, 20 g wet weight, were suspended in 200 ml of 0.145 M NaCl, 50 mM Tris-HCl, pH 7.4 containing 2 mg of lysozyme, 2 mg of DNase, and 2 mg of RNase. The suspension was incubated at 37°C under constant rotation (150 rpm) in a New Brunswick environmental incubator shaker. The reaction was stopped after 2 h by heating the suspension to 85-90°C for 30 min followed by centrifugation at 3,000 x g for 30 min. The supernatant was stored lyophilized. Before use, the powder was dissolved in 200 ml of PBS. After removal of particulate material by centrifugation at 100,000 x g for 1 h, the supernatant (denoted lysate) contained 470 mg of protein. In experiments where 125I-labeled bacteria were used, the procedure described above was scaled down.

Characterization of the Binding of 125I-Fibronectin to S. aureus—Incubation of S. aureus with 125I-fibronectin resulted in a time-dependent binding of the labeled protein to staphylococcal cells (Fig. 1). Live and heat-inactivated (100°C for 10 min) bacteria bound 125I-fibronectin with the same apparent kinetic constants. Maximal binding was reached within 3 h of incubation. When staphylococci were incubated with increasing amounts of 125I-fibronectin, the amounts of radiolabeled protein bound increased in a logarithmic fashion.
tein associated with the bacteria increased in two phases. Initially, the amounts of fibronectin bound to bacteria increased rapidly, but when more than 0.25 µg of fibronectin was added/sample, only a small increase in fibronectin associated with bacteria was observed (Fig. 2). These data were compatible with the presence of a limited number of fibronectin binding sites that became saturated with ligand and, in addition, an unspecific binding of fibronectin to the bacteria that was dependent on the amounts of fibronectin added to the samples. Assuming that fibronectin was bound only to specific receptors and that these receptors are saturated with ligand when 0.25 µg of fibronectin was added to the system, it could be estimated that each bacterial cell possesses around 250 receptor molecules.

Addition of unlabeled fibronectin, but not of fibrinogen, together with the 125I-labeled fibronectin readily blocked binding of the radiolabeled fibronectin to staphylococcal cells (Fig. 3A). Furthermore, addition of human plasma to the incubation mixture inhibited 125I-fibronectin binding to bacteria, whereas fibronectin-depleted plasma did not affect binding (Fig. 3B). These data suggest that the staphylococci recognized fibronectin in plasma and that other plasma proteins including fibrinogen did not interfere with the binding of fibronectin to bacteria and, hence, that fibronectin was specifically recognized by the bacterial receptors. However, 125I-fibronectin bound to staphylococci was not displaced by addition of unlabeled fibronectin (100 µg), which indicated that the fibronectin bacterial binding was functionally irreversible.

Solubilization and Assay of Fibronectin-binding Proteins—A lysate of S. aureus obtained after incubating bacteria with lysostaphin inhibited the binding of 125I-fibronectin to staphylococci (Fig. 4). The observed inhibitory effect of the lysate could be caused by the presence of (A) solubilized fibronectin-binding components (e.g. fibronectin receptors) competing with the cell-bound receptors for the available fibronectin; (B) enzymes degrading the receptor; or (C) enzymes degrading the fibronectin molecules. Preincubation at 100 °C for 10 min did not destroy the activity of the lysate (Table I), indicating that the inhibitory effect of the lysate was not due to the action of heat-labile enzymes. Furthermore, analysis on polyacrylamide gel electrophoresis in SDS (data not shown) indicated that the 125I-fibronectin was not degraded during incubation with the lysate.

Fig. 2. Effect of ligand concentration on the binding of fibronectin to S. aureus, strain Cowan I. Heat-killed bacteria (10⁶ cells/incubation) were incubated with increasing amounts of 125I-labeled fibronectin at 22 °C. Binding was monitored as described under “Materials and Methods” and expressed as micrograms of fibronectin bound to 10⁶ bacteria. Indicated values represent means of duplicate samples.

Fig. 3. Inhibition of the binding of 125I-labeled fibronectin to S. aureus strain Cowan I by various plasma fractions. Heat-killed bacteria (10⁶ cells) were incubated with 125I-fibronectin in the presence of increasing amounts of (A) fibronectin (△—△) and fibrinogen (△—△) and (B) fresh human plasma (○—○) and fibronectin-depleted human plasma (■—■). The effect of the plasma fractions is expressed as per cent inhibition. 0% inhibition corresponds to the amount of fibronectin bound to bacteria in the absence of plasma. 100% inhibition represents the background recorded from incubations performed in the absence of bacteria.

Fig. 4. Effects of bacterial lysate on the binding of fibronectin to staphylococci. Crude lysate (○—○), lysates passed through a Sepharose substituted with IgG (△—△), and fibronectin (●—●), respectively, were tested as inhibitors for the fibronectin-staphylococci interaction. Absorptions were performed as follows. Fifteen ml (90 mg of protein) of concentrated lysates were passed through columns (6.5 x 1 cm) of immobilized protein. Components with no affinity for the gels were collected and tested at different concentrations for inhibitory activity. Comparison was made on an equal protein basis. Bars indicate variations in duplicate samples.
If the activity of the lysate is due to the presence of solubilized fibronectin receptors, the inhibitory molecules in the lysate should express affinity for fibronectin. In accordance with this prediction, material passed through a column of fibronectin-Sepharose was no longer capable of inhibiting the binding of $^{125}$I-fibronectin to staphylococcal cells (Fig. 4). On the other hand, the lysate largely retained the ability to inhibit binding of $^{125}$I-Fibronectin to cells after passage through a column of Sepharose 4B substituted with IgG (Fig. 4). These results indicated that the inhibitory molecules in the lysate could specifically be absorbed on a column of fibronectin-Sepharose.

The nature of the inhibiting molecules was investigated by subjecting the lysate to various treatments (Table I). The inhibitory activity of the lysate was not affected by periodate oxidation or by incubation with lysozyme. Procedures known to modify carbohydrates. However, incubations of the lysate with proteolytic enzymes did result in a loss of inhibitory activity, suggesting that the activity of the lysate resides in protein molecules. In support of a protein nature of the inhibitor(s), the activity was found to be precipitable with ammonium sulfate (data not shown).

These experiments demonstrate the presence of solubilized fibronectin-binding components in the lysate, capable of blocking the binding of fibronectin to receptors on staphylococci.

**Cellular Location of the Fibronectin-binding Proteins**—As shown in Table I, the fibronectin-binding components in the staphylococcal lysate lose their activity when incubated with trypsin. If the fibronectin-binding proteins in the lysate are identical with the fibronectin receptors present at the surface of the bacteria, we would expect that (a) fibronectin binding is abolished after digestion of the staphylococcal cells with trypsin and (b) a lysate from bacteria digested with trypsin should not inhibit binding of fibronectin to staphylococci. Incubation of staphylococci with trypsin resulted in a rapid loss of fibronectin binding (Fig. 5). Likewise, a lysate prepared from staphylococcal cells digested with trypsin did not inhibit the binding of $^{125}$I-fibronectin to staphylococci even though this lysate was added at a concentration 5 times higher than a lysate obtained from untreated bacteria which caused total blockage of fibronectin bacterial binding (Fig. 6). These results demonstrate that the fibronectin receptors on the surface of the *S. aureus* strain Cowan I are susceptible to trypsin digestion and that the fibronectin-binding components responsible for the inhibitory activity in the lysate originate from the cell surface.

**Relationship between Fibronectin Receptors and Protein A**—Initial experiments demonstrated that some but not all preparations of protein A that were tested inhibited binding of $^{125}$I-fibronectin to staphylococci to some extent (see also Ref. 26). Furthermore, a portion of protein A was found to bind to a column of fibronectin-Sepharose and could be eluted by 0.1 M glycine, pH 3.0 (data not shown), whereas solubilized fibronectin receptors showed no or little affinity for IgG-Sepharose (Fig. 4).

These findings prompted us to investigate the possible relationship between the fibronectin receptor and protein A. To this end, we used one strain of *S. aureus* (parent strain SA113(83A)) that bound both $^{125}$I-fibronectin and $^{125}$I-IgG and

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**Table I**

*Inhibition of fibronectin binding to bacteria by staphylococcal lysate*

| Inhibitor                    | $^{125}$I-Fibronectin bound |
|------------------------------|----------------------------|
| None                         | 100                        |
| Lysate                       | 18                         |
| Lysate (preincubated at 100 °C for 10 min) | 20 |
| Lysate incubated with lysozyme | 21 |
| Lysate incubated with trypsin | 20 |
| Lysate incubated with pronase | 99 |
| Lysate incubated with trypsin | 107 |
| Periodate-treated lysate     | 24                         |

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**Fig. 5. Binding of $^{125}$I-fibronectin to trypsin-digested bacteria.** Staphylococci were incubated with trypsin for the indicated periods of time and samples (corresponding to 10⁶ cells) were heat-inactivated and incubated with $^{125}$I-fibronectin. The amount of $^{125}$I-fibronectin bound to treated cells is expressed as percentage of the amount bound to undigested bacteria. For details, see "Materials and Methods."

**Fig. 6. Inhibition of $^{125}$I-fibronectin binding to staphylococci by lysate obtained from intact and trypsin-digested bacteria.** Staphylococci (5 g wet weight) were incubated with (O—O) or without (●—●) trypsin (25 µg/ml) for 30 min at 37 °C in PBS. Subsequently, soybean trypsin inhibitor (50 µg/ml) was added and the incubation was continued for an additional 15 min. The bacteria were collected by centrifugation and suspended in 0.145 M NaCl, 50 mM Tris-HCl, pH 7.4, and solubilized by incubation with lysostaphin. After heat inactivation, the protein concentration of the two lysates was determined and found to be essentially the same (13 mg/ml). The inhibitory activity of different amounts of the two lysates on the binding of $^{125}$I-fibronectin to *S. aureus* strain Cowan I was determined. For details, see "Materials and Methods."
two mutants derived from this strain; one mutant (SA113(83A)prA-320) only bound fibronectin and the other mutant (SA113(83A)hla-6) bound IgG but very little fibronectin (Table II). Lysates obtained from the parent strain and the two mutants by digestion with lysostaphin were analyzed for protein A by immunodiffusion. The strain SA113(83A)prA-320 which did not bind IgG also lacked immunologically detectable amounts of protein A, whereas precipitation lines were formed between the other two lysates and an anti-protein A serum (Fig. 7).

The lysates obtained from the three strains were assayed as inhibitors of fibronectin binding to bacteria. Lysate obtained from the parent strain SA113(83A) and the protein A-negative mutant SA113(83A)prA-320 inhibited the binding of $^{125}$I-fibronectin to S. aureus strain Cowan I, whereas lysate prepared from mutant (SA113(83A)hla-6) had little inhibitory activity (Fig. 8). The relationship between the fibronectin-binding components of these strains was further analyzed in the following way. External surface proteins were labeled by lactoperoxidase-catalyzed $^{125}$I iodination. The bacteria were solubilized and fibronectin-binding components were absorbed on columns of fibronectin-Sepharose. The columns were subsequently washed with PBS and 0.5 M NaCl and finally eluted with 5% SDS. Material eluted from the columns was analyzed by polyacrylamide gel electrophoresis. (For details see legend to Fig. 9.) After drying the gels, the labeled components were

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**Table II**

*Binding of $^{125}$I-IgG and $^{125}$I-fibronectin, respectively, to wild type and mutant strains of S. aureus SA113(83A)*

| Strain                | $^{125}$I-IgG bound | $^{125}$I-Fibronectin bound |
|-----------------------|---------------------|-----------------------------|
| Parent strain, SA113(83A) | 3671            | 571                         |
| SA113(83A)prA-320     | 125               | 791                         |
| SA113(83A)hla-6       | 2243              | 90                          |

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**Fig. 7.** Immunodiffusion of anti-protein A sera against solubilized bacteria. Ten μl of a rabbit antiserum raised against protein A were applied in the center well in an agarose gel on a glass plate. In the peripheral wells, 10 μl of lysate obtained after lysostaphin treatment of S. aureus strain parent strain (A); hla-6 (B); and prA-320 (C). The plate was incubated at room temperature overnight in a humidified chamber and subsequently stained with Coomassie blue.

**Fig. 8.** Inhibition of $^{125}$I-fibronectin binding to S. aureus strain Cowan I by lysates prepared from different strains of staphylococci. Lysates prepared by lysostaphin digestion of strain SA113(83A) (C—C), strain SA113(83A)prA-320 (●—●), and SA113(83A)hla-6 (Δ—Δ) were analyzed for inhibitory activity of $^{125}$I-fibronectin binding to Cowan I. For details, see “Materials and Methods.”

**Fig. 9.** Gel electrophoreses of $^{125}$I-labeled surface proteins purified by affinity chromatography on a column of fibronectin-Sepharose. S. aureus strains parent SA113(83A) (I), SA113(83A)prA-320 (II), and SA113(83A)hla-6 (III) were labeled by $^{125}$I iodination and cells corresponding to $5 \times 10^6$ cpm of each strain were lysed with lysostaphin. The lysates were passed through columns (2 ml) of Sepharose 4B to remove labeled components that bind to Sepharose gels. The columns were washed with 1 ml of PBS and the eluates were applied to columns (1.5 ml) of fibronectin-Sepharose gels. The columns were washed with 5 ml of PBS followed by 5 ml of 0.5 M NaCl in PBS. Subsequently, the material was eluted with 10-200 μl portions of 5% SDS. Labeled proteins eluted with SDS, corresponding to $10^5$ cpm, were applied to the polyacrylamide gel. For further details, see “Materials and Methods.”
visualized by autoradiography. Strain SA113(83A) (that binds both 125I-fibronectin and 125I-IgG) contained labeled fibronectin-binding components with molecular weights of 45,000, 32,000, 29,000, and 18,000, respectively. The protein A-deficient strain contained only the 18,000 component, whereas the 45,000 component was the only labeled component that could be detected from the strain that bound 125I-IgG but not 125I-fibronectin (Fig. 9). In addition, radiolabeled components that did not penetrate the gel were observed from all three strains. The presence of a fibronectin-binding protein ($M_r \sim 18,000$) in a protein A-deficient mutant SA113(83A)prA-320 but absent in the mutant SA113(83A)hla-6, which virtually lacks fibronectin receptors, suggested that fibronectin receptor activity resides in the 18,000 component. Furthermore, since protein A on gel electrophoresis migrates as a protein with a $M_r$ of 45,000, it appears possible that the radiolabeled 45,000 protein is identical with protein A.

### DISCUSSION

In the present communication, the binding of fibronectin to S. aureus strain Cowan I was characterized. The binding was specific in the sense that the presence of plasma proteins other than fibronectin did not affect the binding of 125I-fibronectin to staphylococci. Both live and heat-killed bacteria bound 125I-fibronectin in a reaction that required about 3 hours for completion. Radiolabeled fibronectin associated with the bacteria could not be displaced by unlabeled fibronectin, which indicated that the fibronectin binding was essentially irreversible and that the receptor-ligand complex was kept together by strong forces.

S. aureus strain Cowan I bound a maximum of $0.29 \times 10^{-6}$ $\mu$g of fibronectin/cell which suggested that 250 receptor molecules were available/cell. Digestion of the staphylococci with trypsin abolished the ability of the bacteria to bind fibronectin which indicated that the receptor was located at the surface of the cells and contained a protein component. A lysate obtained by digesting the bacteria with lysostaphin inhibited the binding of fibronectin to bacteria. The inhibitory activity in the lysate was presumably due to the presence of solubilized receptor molecules since: (A) a lysate obtained from trypsin-digested bacteria did not contain inhibitory activity and (B) the inhibitory activity could be absorbed on a column of fibronectin-Sepharose but not on a column of Sepharose substituted with IgG.

The presence of fibrinogen did not interfere with the binding of 125I-fibronectin to staphylococci which indicated that the two proteins bind to different receptor molecules at the bacterial cell surface. The relationship between protein A, which binds IgG, and the fibronectin receptor is unclear. Conflicting data have been reported as to the ability of purified protein A to inhibit the binding of 125I-fibronectin to bacteria (25, 26). Preliminary studies in our laboratory have shown that a portion of some preparations of protein A will bind to a column of fibronectin-Sepharose. Consistent with these observations is that S. aureus strain SA113(83A) contained a surface protein that bound to fibronectin-Sepharose and had a molecular weight (~ 45,000) similar to that of protein A. This protein which could be labeled by external 125I iodination was present in a strain of S. aureus that expressed protein A but virtually lacked fibronectin binding. The 45,000 protein was, however, absent in a protein A-deficient mutant that binds fibronectin. From these observations, we conclude that the 45,000 protein is not identical with the primary fibronectin receptor, instead the fibronectin receptor activity correlated with a protein from the surface of the staphylococci with a $M_r = 18,000$. Furthermore, the solubilized fibronectin receptor molecules show, in contrast to protein A, little or no affinity for IgG-Sepharose (Fig. 4). The 15,000 protein can be purified from a protein A-negative mutant of S. aureus. However, it can not be excluded that the primary receptor may occur connected with other cell surface components (e.g. protein A) and that treatment of the bacteria with lysostaphin results in the solubilization of complexes containing the receptor in addition to other components.

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