Some strains of *Streptococcus pyogenes* isolated from infected human tissues were shown to bind laminin, a major component of basement membranes. Binding of 125I-laminin to bacteria was time dependent and functionally irreversible. Of several unlabeled proteins tested in competition experiments, laminin and fibrinogen inhibited binding of the radiolabeled protein. The inhibitory effect exerted by fibrinogen was apparently not caused by a binding to the laminin receptors. The number of receptors available for laminin on cells of the strain examined ranged from 0 to 10^5 depending on the media used to grow the bacteria and an apparent Kd of 4 x 10^-8 M was calculated for the reaction. Bacterial cells incubated with proteolytic enzymes lose the ability to bind laminin, and a trypsin digest contained active receptors capable of competing with intact cells for 125I-laminin. Active receptors may be adsorbed on a column of laminin-Sepharose but not on Sepharose gels substituted with fibrinogen or fibronectin. After radiolabeling the proteins in the trypsin digest a laminin-binding 125I-labeled protein (Mr > 10^6) was isolated by affinity chromatography from a receptor positive strain. Similar components could not be isolated from a strain apparently lacking laminin receptors. Therefore, this protein was tentatively identified as a laminin receptor of streptococci.

During the last decade the idea that adherence of bacteria to host tissues represents the initial step of the infectious process has gained increasing attention. As a consequence, the adhesion of various bacteria to different cells has been extensively studied (for reviews, see Refs. 1 and 2). These interactions presumably involve unspecific physicochemical forces (charge and hydrophobicity), and the attachment of *Streptococcus pyogenes* to an epithelium may include hydrophobic interaction with lipoteichoic acid and the M protein on the bacteria (2, 3). In addition, specific interactions between bacterial cell wall components and appropriate receptors on the cells of the host are presumably involved in bacterial adherence, although the molecular mechanisms of the interactions are not well understood.

Many bacterial infections follow on an initial viral infection or trauma that may damage the protective epithelial lining of organs and expose the basement membrane underlying the epithelial cell layer. Some bacteria, e.g. group A streptococci, are known to produce cytolysin toxins (4) which may affect epithelial cells and make the epithelium permeable to bacteria. For these reasons not only epithelial cells but the underlying basement membrane is a potential substrate for bacterial adherence. Laminin is an abundant basement membrane component and is believed to serve as a substrate for bacterial adherence. Laminin is presumably not exposed in undamaged tissue but may be exposed following trauma or lysis of cells. In the present communication we report that some strains of *S. pyogenes* bind laminin in an interaction that involves distinct receptors present on the surface of the bacteria.

**MATERIALS AND METHODS**

**Chemicals**—Laminin was isolated from mouse Engelbreth-Holm-Swarm tumor as described (10) and labeled with 125I-iodine using the chloramine-T method (11). Fibronectin was purified from human plasma (12).

Egg albumin, bovine serum albumin, trypsin (type III, 1.1 x 10^6 units/mg), soybean trypsin inhibitor, human IgG, a1 acid glycoprotein (orosomucoid), fetuin, and lactoperoxidase were purchased from Sigma. Percoll was purchased from Pharmacia Fine Chemicals, Uppsala, Sweden. Human fibrinogen was a generous gift from Dr. M.C. Poon, V. A. Hospital, Birmingham, AL. Laminin, fibronectin, and fibrinogen were coupled to CNBr-activated Sepharose 4B (Pharmacia Fine Chemicals, Uppsala, Sweden) according to the manufacturer's instructions. Na[125I] (specific activity 15 mCi/µg) was obtained from Amersham Corp.

**Bacteria**—Forty strains of *S. pyogenes*, freshly isolated from patients with pharyngitis, were kindly supplied by Dr. Asa Ljungh, Central Microbiological Laboratory, Stockholm, Sweden. Bacteria were subcultured on blood agar and grown in Todd-Hewitt broth (Difco) for 18 h at 37 °C, unless otherwise stated. For one series of experiments bacteria were grown in brain-heart infusion broth (Difco) and two semisynthetic media described by Holm and Falven (13) and Ikeda et al. (14), respectively. Bacteria were harvested by centrifugation (1500 x g, 20 min), washed twice with PBS (0.13 M sodium chloride, 0.02% sodium azide, 10 mM phosphate buffer, pH 7.4), and suspended in the same buffer. Cells were heated at 80 °C for 10 min, and the cell density was adjusted to 10^10/ml. Bacteria were counted in a Petroff Hauser chamber.

**Binding of 125I-Laminin to Bacteria**—The binding of 125I-laminin to bacteria was quantitated essentially as described previously for binding of fibronectin to staphylococci (15). Briefly, 2 x 10^8 bacteria were incubated with 5 x 10^5 cpm (0.08 µg) of the radiolabeled protein.

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1 The abbreviations used are: PBS, phosphate-buffered saline; SDS, sodium dodecyl sulfate.
pared to heat-killed cells, although the binding reactions tively. Binding of lZ5I-laminin to live bacteria incubated with 0.13 pg of laminin/ml was approximately 15% higher compared to 2 and 250 for bacteria incubated with 0.13 pg of laminin/ml was approximately 15% higher compared to 15% higher.

The plastic tubes used in the experiments were preincubated overnight with 1 mL of 0.1% bovine serum albumin solution in PBS to minimize unspecific binding of bacteria and proteins to the walls of the tubes.

Digestion of Streptococci with Trypsin—Cell surface components of streptococci were solubilized by treatment of cells with trypsin. One hundred mg (wet weight) of bacteria was heated at 80 °C for 10 min, centrifuged (3000 × g, 15 min), and resuspended in 0.5 ml of PBS without azide. This suspension was digested with trypsin (25 μg/ml) for 1 h at 37 °C. The reaction was stopped by the addition of soybean trypsin inhibitor (50 μg/ml), the cells were pelleted by centrifugation, and the supernatant was incubated at 80 °C for 10 min and stored at −20 °C.

Affinity Chromatography—Samples (300 μl) of material released from the bacteria by trypsin were applied to columns containing 0.5 ml of unsubstituted or protein-substituted Sepharose equilibrated with PBS. The columns were washed with 4 ml of PBS followed by 4 ml of 0.5 M NaCl in 10 mM phosphate, pH 7.4, to elute loosely bound proteins. Material tightly bound to the laminin-Sepharose was eluted with a buffer consisting of 4% sodium dodecyl sulfate, 10% 2-mercaptoethanol, and 20% glycerol in 0.125 M Tris·HCl, pH 6.8.

Gel Electrophoresis—Electrophoresis in polyacrylamide gels was performed according to Studier (16). The running gel consisted of a linear 5 to 15% acrylamide gradient overlayed with stacking gel (3% acrylamide concentration). Samples consisting of 2 μg of each protein; Pharmacia Fine Chemicals, Uppsala, Sweden) were applied per lane after boiling in the presence of SDS-containing buffer. Following electrophoresis the gels were stained with Coomassie brilliant blue R, destained, dried under vacuum and subjected to autoradiography using X-Omat AR film (Kodak, Rochester, NY).

**RESULTS**

Screening of 40 randomly selected strains of *S. pyogenes* representing clinical isolates showed that 21 strains bound 125I-laminin. Strains binding less than 2% of the added labeled protein (i.e. less than 150 cpm over the background level) were considered as nonbinders. Among the positive strains the ability to bind laminin varied considerably; some strains bound up to 35% (corresponding to 8.8 × 10^6 cpm/10^8 bacteria) of the added radiolabeled protein. Two strains were chosen for more detailed studies, i.e. strain 57, which was a nonbinder, and strain 1321, which had intermediate binding capacity. Most of the analyzed strains including 57 and 1321 bound fibronectin (17), fibrinogen, and human IgG (data not shown).

The binding of 125I-laminin to heat-killed (80 °C for 10 min) cells of strain 1321 was time and concentration dependent, as shown in Fig. 1. When the assay was performed with a low concentration of laminin (0.13 μg/ml), the binding reaction required 6–7 h for completion, whereas at a higher concentration (16.7 μg/ml) the binding reaction proceeded much faster and was essentially completed after 2 h. The average number of laminin molecules bound per cells was 2 and 250 for bacteria incubated with laminin at 0.13 μg/ml and 16.7 μg/ml, respectively. Binding of 125I-laminin to live bacteria incubated with 0.13 μg of laminin/ml was approximately 15% higher compared to heat-killed cells, although the binding reactions showed the same time dependence (data not shown). Bacteria used in this study were routinely heat-treated to prevent degradation of bacterial cell envelope components by lytic enzymes and/or degradation of 125I-laminin by streptococcal proteases. Heat-treated suspensions of bacteria kept at 4 °C retained the ability to bind laminin for several months.

With increasing concentrations of 125I-laminin in the incubation mixture the amount of radiolabeled protein bound to the bacteria increased to a maximum of 1.55 pg of laminin/10^6 cells at a laminin concentration of 60 μg/ml (Fig. 2). If we assume a molecular weight of 9 × 10^6 for laminin (18) and further that all 125I-laminin associated with the bacteria represents receptor-bound ligand, the average number of receptors per cell of strain *S. pyogenes* 1321 grown in Todd-Hewitt medium is 10^4.

The number of receptors available for laminin binding depended on the media used to grow the bacteria. When strain 1321 was grown in brain-heart infusion broth or a semisyn-
thetic medium described by Holm and Falsén (13) the maximum binding was markedly reduced (Fig. 2) and corresponded to an average number of laminin receptors of 270 and 570 per cell, respectively. Bacteria grown in the semisynthetic medium described by Ikeda et al. (14) bound only traces of labeled laminin. The reduced number of available receptors present in bacteria grown in media other than Todd-Hewitt broth was not due to release of active receptors in the medium as culture supernatants failed to inhibit the binding of 125I-laminin. Thus, the added unlabeled protein exceeded by 2000 times the amount blocked binding of the 125I-labeled laminin to bacteria (Table 1).

Addition of an excess of unlabeled laminin effectively blocked binding of the 125I-labeled laminin to bacteria (Table I). This demonstrates that the bacterial receptors recognize both forms of the protein. The binding of 125I-laminin was not significantly reduced by fetuin, a1 acidglycoprotein, IgG, or fibronectin. However, somewhat surprisingly, fibrinogen also failed to inhibit the binding of 125I-laminin to bacteria. As streptococci of group A are known to bind fibrinogen, the possibility was considered that these two proteins bind to the same receptor. All but one of the strains that did not bind laminin bound 125I-fibrinogen (data not shown) and hence possess fibrinogen but not laminin receptors. Thus, the receptors involved in binding of the two proteins are different. This conclusion was supported in experiments where solubilized laminin receptors were shown to bind to laminin (but not to fibrinogen) Sepharose (see below).

Whereas addition of excess amounts of unlabeled laminin together with the labeled derivative effectively blocked the binding of 125I-laminin to bacteria, attempts to displace bacteria-bound 125I-laminin by additions of unlabeled laminin failed (data not shown). In this experiment the amount of added unlabeled protein exceeded by 2000 times the amount of labeled laminin, and the incubation time of the standard assay (3 h) was used. The presence of other proteins including fibrinogen also failed to displace bound 125I-laminin. Thus the binding of laminin to streptococci is functionally irreversible, and the requirements for analyzing the binding data in a Scatchard plot are, therefore, not fulfilled. However, an apparent KD of 4 × 10^-10 M can be calculated from the concentration of laminin giving half-maximum binding.

**Solubilization of Laminin Receptors—Digestion of streptococcal cells** (strain 1321) with trypsin reduced their binding of 125I-laminin to 6% of the control value. Functional receptors were solubilized in the process since material released by trypsin inhibited binding of 125I-laminin to intact bacteria (Fig. 3). Fifty per cent inhibition of binding of labeled laminin to 3 × 10^9 cells was observed after addition of 30 μl of trypsinate, i.e., material obtained after digestion of 6 mg (wet weight) of cells (approximately 2 × 10^9 cells). Increasing the amounts of added trypsinate resulted in an almost complete inhibition of binding. Incubation of bacteria with Pronase or papain also released the receptors, although the digest did not inhibit binding of 125I-laminin to bacteria. These data suggest that the laminin receptor is a protein inactivated (degraded?) by Pronase or papain digestion. On gel chromatography of the trypsinate-released material on a column of Sepharose 2B the receptor (as indicated by inhibitory activity) was eluted at the void volume (data not shown). The binding of 125I-laminin to strain 1321 was not markedly inhibited by addition of up to 100 μl of trypsinate obtained from strain 57, which does not bind laminin. However, when the amount of trypsinate from this strain was increased, up to 30% inhibition of binding was observed. Materials released by trypsin treatment of cells of strains 1321 and 57, respectively, were both passed through columns of unsubstituted Sepharose as well as affinity chromatography on a column of laminin-Sepharose. The results of these experiments (Table II) showed that the inhibitory activity of the trypsinate-released material from strain 1321 was adsorbed to laminin-Sepharose only. This indicates a specific affinity for laminin of the inhibiting components in the trypsinate from strain 1321 which is consistent with the presence of solubilized receptors. The inhibitory activity of trypsinate from strain 57 was not adsorbed to any gel and presumably cannot be ascribed to the presence of specific laminin receptors.

To further characterize the laminin-binding components, trypsinate of both strains were passed through columns of Sephadex G-200. Material having receptor activity (i.e., possessing inhibitory activity) was eluted at the void volume. This material was labeled with 125Iiodine and subjected to affinity chromatography on a column of laminin-Sepharose. After applying the radiolabeled material, the column was

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**TABLE I**

**Inhibition of binding of 125I-laminin to S. pyogenes 1321 by unlabeled proteins**

Heat-killed cells (2 × 10^9) of *S. pyogenes* strain 1321 were incubated for 3 h in the presence of 5 × 10^5 cpm of 125I-laminin (approximately 0.08 μg) and 100 or 500 μg of unlabeled proteins. Controls were assayed in the absence of test proteins.

| Protein          | Inhibition by unlabeled proteins added (mean ± S.E.) | %  |
|------------------|-----------------------------------------------------|----|
|                  | 100 μg                                               | 500 μg |
| No additions     | 0                                                   | 0    |
| Laminin          | 84.8 (±9.3)                                         | ND*  |
| Fibrinogen       | 62.9 (±7.4)                                         | 84.8 (±11.5) |
| Fibronectin      | -3.2 (±2.8)                                         | 4.3 (±3.0) |
| IgG              | -2.4 (±2.6)                                         | 4.6 (±2.5) |
| a1 acidglycoprotein | 4.7 (±3.1)                                      | 8.5 (±2.8) |
| Fetuin           | 5.5 (±2.2)                                         | 12.0 (±3.7) |

* ND, not determined.
Adsorption of inhibitory activity on protein-substituted Sepharose gels

Solubilized receptors of *S. pyogenes* strains 1321 and 57 produced by trypsin treatment were passed through columns of Sepharose (bed volume 0.5 ml equilibrated with PBS), and 300 μl of each unfractionated and adsorbed trypsinate were checked for the inhibitory activity in the standard assay. Controls were as described in the legend to Fig. 3. Data are presented as percentages of controls. For experimental details see under "Materials and Methods."

**Table II**

| S. pyogenes 1321 cells incubated in the presence of | Inhibition of ¹²⁵I-laminin binding by trypsin-released material from |
| --- | --- |
| S. pyogenes 1321 | S. pyogenes 57 |
| Unfractionated trypsinate | 93 | 25 |
| Trypsinate passed through Sepharose CL-4B | 88 | 18 |
| Laminin-Sepharose | 9 | 15 |
| Fibrinogen-Sepharose | 95 | 21 |
| Fibronectin-Sepharose | 87 | 19 |

Controlled autoradiography on laminin-Sepharose of ¹²⁵I-labeled high molecular weight components of trypsin-released material. Trypsinates of strain 1321 (○) and strain 57 (△) were labeled with ¹²⁵I iodine and applied to a column (90 × 1 cm) of Sephadex G-200 in PBS. High molecular weight components eluting at the void volume of the column were applied to a column of laminin-Sepharose (bed volume 0.5 ml) equilibrated with PBS. The columns were washed with PBS (fractions 1-8) or 0.5 M NaCl in 10 mM phosphate buffer, pH 7.4 (fractions 9-16) followed by a sodium dodecyl sulfate-containing buffer. Five hundred-pl fractions were collected and 20 μl of each sample were analyzed for radioactivity.

Washed with PBS, followed by 0.5 M NaCl in 10 mM phosphate buffer, pH 7.4, which does not elute the receptor. Finally the column was washed with a buffer containing 4% SDS. As shown in Fig. 4 the material originating from strain 57 was largely washed through the column, and very little radioactivity was eluted by the SDS-containing buffer. On the other hand, a significant portion of the material from strain 1321 bound to laminin-Sepharose and was eluted with the SDS buffer.

Analysis by gel electrophoresis of the radiolabeled material of high molecular weight from strain 1321 showed the presence of high molecular weight components that remained on top of the gel and in addition two proteins of lower molecular weight (Fig. 5). The ¹²⁵I-labeled material that did not bind to the laminin-Sepharose contained the low molecular weight components as well as a portion of the high molecular weight material. The radiolabeled material that bound to the gel and was eluted in the presence of 4% SDS consisted of a high molecular weight component ($M_r > 10^6$).

**Discussion**

We have examined 40 strains of group A streptococci (*S. pyogenes*) for the ability to bind the basement membrane protein laminin. About half of the strains tested bound ¹²⁵I-labeled laminin in a time-dependent reaction, and one strain (1321) was selected for further studies. Cells of this strain could be saturated with ligand indicating the presence of a limited number of surface receptors. The numbers of available receptors present on the bacteria depended on the media used for growing the cells, e.g., whereas the average number of receptors per cell was 10² on bacteria grown in Todd-Hewitt broth and 0.5 × 10³ on bacteria grown in Holm-Falsen's medium (13), hardly any receptors could be detected on bacteria grown in the semisynthetic medium described by Ikeda et al. (14). Similarly, the number of available fibronectin receptors on *S. aureus* has been shown to depend on growth conditions (19). The reasons for these variations are presently unclear.

Binding of laminin to streptococci was specific in that a number of other proteins, with the exception of fibrinogen, did not interfere in the binding reaction. Our data indicate that fibrinogen and laminin bind to separate receptors located in close proximity to each other and that fibrinogen bound to its receptor interferes sterically with the binding of laminin. However, other explanations of the inhibitory effect of fibrinogen cannot be ruled out at the present time.

The laminin receptor on streptococcal cells was solubilized by trypsin digestion of the bacteria and partially purified by gel chromatography and affinity chromatography on laminin-Sepharose. Digestion of bacteria with papain or Pronase also removed the laminin receptor but in a degraded form. Gel electrophoresis of the purified receptor indicates that it is a
high molecular weight component (\( M_r > 10^9 \)). After centrifugation of the trypsinate at 90,000 \( \times g \) for 90 min the receptor activity remained in solution (data not shown) which indicates that the receptor is not part of particulate material. In a recent report Stinson and Bergey (20) demonstrated a time-dependent binding of a cell wall protein from \( S. \) \( pyogenes \) to the glomerular basement membrane of monkey kidney. The relation between this protein and the presently described laminin receptor is unclear.

Binding of laminin to streptococci was functionally irreversible which may suggest that the initial recognition between the receptor and its ligand is followed by conformational rearrangements in the interacting proteins. As a consequence new antigenic sites may be exposed in the laminin molecule that could be recognized by the host and trigger an immune response. Rheumatic fever and glomerulonephritis are diseases with postulated autoimmune pathogenesis (21). They are common sequelae of throat and skin infections caused by \( S. \) \( pyogenes \). It is presently not known if anti-laminin antibodies are found in any of these conditions. The presence of laminin-binding antibodies in infections caused by \( Trypanosoma cruzi \) was recently reported (22). It was postulated that the parasite contains laminin-like structures which trigger the immune response resulting in the synthesis of antibodies that cross-react with laminin of the host tissue although alternative explanations cannot be ruled out.

It is tempting to speculate that the ability of streptococci to bind laminin represents a mechanism of bacterial tissue adherence and that laminin may serve as a substrate for adhesion of not only eucaryotic cells (4) but also of bacteria. Most likely the adherence of streptococci to host tissue involves also mechanisms other than laminin binding. Group A streptococci bind both fibrinogen (23) and fibronectin (17), which may represent other potential substrates for bacterial adherence.

Laminin may act as an attachment protein for normal and malignant eucaryotic cells in vivo mediating growth, differentiation, and locomotion. Laminin receptors of eucaryotic cells have recently been isolated and characterized (24–27). The affinity of binding (2 \( \times 10^9 \) M) is similar to that reported by us for the binding of laminin to streptococcal cells; the number of receptors per eucaryotic cell ranges from 5 \( \times 10^4 \) to 10\(^6\), compared to 10\(^8\) for the strain of \( S. \) \( pyogenes \) examined here. It is unclear if these two types of laminin receptors are related.

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