The Glycosylphosphatidylinositol-anchored Surface Glycoprotein Thy-1 Is a Receptor for the Channel-forming Toxin Aerolysin*

Kim L. Nelson, Srikumar M. Raja, and J. Thomas Buckley†

From the Department of Biochemistry and Microbiology, University of Victoria, Victoria, British Columbia V8W 3P6, Canada

Aerolysin is a channel-forming protein secreted by virulent Aeromonas spp. Some eukaryotic cells, including T-lymphocytes, are sensitive to very low concentrations of the toxin (<10⁻⁹ M). Here we show that aerolysin binds selectively and with high affinity to the glycosylphosphatidylinositol (GPI)-anchored surface protein Thy-1, which is found on T-lymphocyte populations as well as in brain. Less than 1 ng of purified Thy-1 could be detected by probing Western blots with the toxin. Mutant T-cell lines that lack the ability to add GPI anchors to Thy-1 and other surface proteins were much less sensitive to aerolysin, as were wild-type cells that were pretreated with phosphatidylinositol-specific phospholipase C to remove GPI-anchored proteins. Phosphatidylcholine/cholesterol liposomes containing purified Thy-1 in their membranes were much more sensitive to aerolysin than protein-free liposomes.

Aerolysin is one of the best studied of all of the bacterial cytolytic toxins. The protein is secreted as a 52-kDa inactive precursor called proaerolysin by a number of Aeromonas spp., and it is known to be required for the virulence of Aeromonas hydrophila in mice (see Ref. 1 for a recent review). Proaerolysin exists as a freely soluble dimer in solution. It is converted to the active form of the protein by proteolytic processing near the C terminus. Aerolysin is also a soluble dimer; however, in contrast to the protoxin form, it is capable of oligomerization, producing heptameric structures that transform the protein into an insertion-active state.

Cells that contain a receptor for aerolysin are much more sensitive to the toxin than other cells. Among erythrocytes that have been compared, those of the rat are most sensitive (2, 9), and we have shown that this is because they contain a 47-kDa glycoprotein that binds both proaerolysin and aerolysin with considerable affinity (4). Presumably the primary function of the receptor is to concentrate the toxin on the cell surface, thereby indirectly promoting oligomerization. However, it is also possible that receptor binding has a more direct role in oligomerization. For example, it could facilitate dissociation of the aerolysin dimer, which is presumably an essential step in formation of the heptamer.

Most studies with cytolytic toxins have been carried out with erythrocytes because of their uncomplicated structure and metabolism, the ease with which they can be obtained free of other cells, and the simplicity of hemolytic assays. However, there is no reason to believe that these cells would be the primary targets of the bacterial pathogens that secrete cytolsins. Components of the immune system are more obvious candidates for toxin targeting. In this report, we show that Thy-1, a major surface glycoprotein of rodent T-lymphocytes, is a high affinity receptor for aerolysin.

EXPERIMENTAL PROCEDURES

Cell Culture—The murine lymphoma cell lines BW5147.3 and BW5147.3(Thy-1–) were generously supplied by R. Hyman (Salk Institute). All cell lines were maintained in Dulbecco's modified Eagle's high glucose medium supplemented with 10% bovine fetal clone I serum, 100 units/ml penicillin, and 100 μg/ml streptomycin in 5% CO₂ at 37 °C.

Cell Death Experiments—Cells (5 x 10⁶/ml) in complete Dulbecco's modified Eagle's medium were incubated with 7 x 10⁻¹⁰ M aerolysin for 1 h at 37 °C. Live/dead counts were made using a hemocytometer after adding an equal volume of 0.1% trypan blue in phosphate-buffered saline.

Killing of EL4 cells by aerolysin was also measured using the membrane-impermeant probe Po-Pro 1 (Molecular Probes, Inc.), which fluoresces when bound to nucleic acids made available by the action of aerolysin on the cells. The time dependence of cell death was measured at various aerolysin concentrations with a Photon Technology QM-1 spectrophotometer. Excitation and emission wavelengths were 435 and 455 nm, respectively, and slit widths were 4 nm.

SDS-PAGE and Detection of Aerolysin-binding Proteins by Western Blotting—SDS-PAGE was carried out by the method of Neville (5). Gels were blotted onto nitrocellulose and developed with sandwich Western blotting as described by Gruber et al. (4), which involves probing blots with 2 x 10⁻⁵ M aerolysin, followed by polyclonal anti-aerolysin antibody and anti-rabbit horseradish peroxidase. Blots were then developed by enhanced chemiluminescence (Amersham Corp.).

Tissue Homogenates—Tissues were quickly excised and homogenized in five volumes of 50 mM Tris-HCl, 0.16 M NaCl, 1 mM EDTA, 1 mM phenylmethylsulfonyl fluoride, and 1 μM pepstatin A, pH 8, using a Polytron homogenizer (Brinkmann Instruments) at 4 °C. Protein was determined as described by Markwell et al. (6).

N-Deglycosylation of Glycoproteins—An equal volume of 40 mM NaH₂PO₄, 100 mM EDTA, 1% SDS, and 10% β-mercaptoethanol, pH 7.5, was added to 5 μl of 4 x 10⁵ cells/ml in phosphate-buffered saline, and the mixture was boiled for 2 min. After cooling to room temperature, 3.3 μl of a protease inhibitor mixture consisting of 0.6 mM phenylmethylsulfonyl fluoride, 60 μl of apotinin, 120 μl leupeptin, and 12 μl pepstatin A were added, followed by 2.5 μl of 10% octyl glucoside and 7.5 μl of peptide N-glycosidase F (Oxford Glycosystems) containing 1.5 units of the enzyme. A control incubation was also carried out in which 7.5 μl of buffer were added in place of the enzyme. After 18 h at 37 °C, sample buffer was added, and aliquots were separated by SDS-PAGE and sandwich Western-blotted.

Thy-1 Purification—Affinity-purified Thy-1 from rat thymus (7) was

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† To whom correspondence should be addressed: Dept. of Biochemistry and Microbiology, University of Victoria, P. O. Box 3055, Victoria, British Columbia V8W 3P6, Canada. Tel.: 250-721-7081; Fax: 250-477-0579.

‡ To whom correspondence should be addressed: Dept. of Biochemistry and Microbiology, University of Victoria, P. O. Box 3055, Victoria, British Columbia V8W 3P6, Canada. Tel.: 250-721-7081; Fax: 250-477-0579.

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a generous gift from Dr. R. McMaster (University of British Columbia). The only band visible on a silver-stained SDS-polyacrylamide gel corresponded to the monomeric protein. In addition to the monomer, a small band corresponding to the dimer was detected by sandwich Western blotting.

**Liposome Preparation and Incorporation of Thy-1**—Dried lipid films were prepared from a mixture of egg yolk 1-phosphatidylycholine (Avanti Polar Lipids, Inc.) and cholesterol (mole ratio of 7:1) and rehydrated in 2 ml of 100 mM carboxyfluorescein in 20 mM HEPES and 150 mM NaCl, pH 7.4. Large unilamellar vesicles (liposomes) were prepared by extrusion through 25-mm polycarbonate filters using an Extruder (Lipex Biomembranes Inc., Vancouver, British Columbia, Canada) as described by Hope et al. (8). The liposome concentration was determined by measuring the phosphate content using a standard assay (9).

Thy-1 was incorporated into the liposomes following the procedure of Rigaud et al. (10). Briefly, 250 μl of Thy-1 (42.5 μg) in 10 mM Tris, pH 8.0, containing 1% octyl glucoside were added to 500 μl of carboxyfluorescein-entrapped liposomes (1.3 μmol of lipid). As a negative control, 250 μl of 10 mM Tris, pH 8.0, containing 1% octyl glucoside were added to 500 μl of carboxyfluorescein-entrapped liposomes. To both samples, were added 250 μl of a 2.2% stock of octyl glucoside in 20 mM HEPES and 150 mM NaCl, pH 7.4, so that the final octyl glucoside concentration was 0.8%. After overnight dialysis against 20 mM HEPES and 150 mM NaCl, pH 7.4, the liposomes were passed over a Sephacryl S-300 column (18 ml) to remove unincorporated Thy-1.

**Liposome Release Assay**—Carboxyfluorescein release was monitored spectrophotometrically. The excitation wavelength was set at 490 nm, and the emission wavelength at 520 nm. A 4-nm slit width was used for both monochromators. Small aliquots of the liposome preparations (21.5 nmol of lipid) were added to 3 ml of 20 mM HEPES and 150 mM NaCl, pH 7.4, and carboxyfluorescein release was followed with time. Activated aerolysin was added to a concentration of 4 × 10^{-3} mM at 2 min. Total entrapped carboxyfluorescein in the liposomes was measured by adding Triton X-100 to a 0.1% (w/v) final concentration at the end of each run. All of the experiments were carried out at room temperature.

**Phosphatidylinositol-specific Phospholipase C Treatment**—Five-hundred μl of 9 × 10^7 EL4 cells/ml were incubated with 200 milliunits of phosphatidylinositol-specific phospholipase C (PI-PLC; Boehringer Mannheim) for 2 h at 37°C in phosphate-buffered saline. A control sample was incubated without the enzyme. The cells were subsequently pelleted at 80,000 rpm for 20 min at 4°C in a Beckman TLA 100.2 rotor. Aliquots of the supernatants and pellets were used for the sandwich Western blotting procedure.

Liposomes (86 nmol of phosphatidylincholine) containing reconstituted Thy-1 (0.85 μg) and control liposomes containing no protein were treated with 600 milliunits of PI-PLC for 60 min at room temperature. Liposomes containing Thy-1 incubated under the same conditions without PI-PLC also served as controls.

**RESULTS**

**Mouse Brain and Thymus Contain a 30-kDa Protein That Binds Aerolysin**—We have shown that there is a 47-kDa glycoprotein in rat erythrocyte membranes that binds aerolysin with high affinity (4). The protein can be detected by exposing Western blots of membrane proteins to low concentrations of proaerolysin (10^{-9} M) and probing with an anti-aerolysin antibody after washing to remove unbound protein (4). We decided to screen a number of mouse tissues in a similar way for proteins that might bind proaerolysin. The tissues were homogenized and dissolved directly in sample buffer for SDS-PAGE. Bands indicating aerolysin binding were observed in all of the tissues, but by far the most intense were those observed in the region of 30 kDa in the lanes containing thymus and brain (Fig. 1).

**T-cell Lines Also Contain a 30-kDa Aerolysin-binding Component**—The appearance of an aerolysin-binding protein on blots of homogenized thymus prompted us to examine several T-cell lymphomas in the same way as we screened the tissues. The results for two cell lines, EL4 and BW5147.3, are shown in Fig. 2. It is clear that a protein corresponding to those observed in the brain and thymus is also found in these T-cells.

**The T-cell Aerolysin-binding Protein Is GPI-anchored**—We have obtained evidence that the rat erythrocyte receptor is anchored to the cell surface by glycosylphosphatidylinositol attached to its C terminus (1). To determine if the 30-kDa protein in the T-cells might be anchored in a similar way, we took advantage of the fact that there are EL4 and BW5147.3 cell lines that lack the ability to attach these anchors to any of their surface proteins (11). When proteins from these cells were studied by the Western blotting technique, it was clear that they contained far lower quantities of the 30-kDa species (Fig. 2).

GPI anchoring of the 30-kDa protein found on normal EL4 cells, suggested by the experiment with the mutant cells, was confirmed by treating the wild-type cells with PI-PLC. This enzyme is known to release many proteins anchored to cell membranes in this way (12). The results in Fig. 3 show that reaction with the enzyme led to a pronounced decrease in the amount of cell-associated aerolysin-binding protein, which appeared in the soluble fraction after cell pelleting. This allows us to conclude that this proaerolysin-binding protein is GPI-anchored and also that the lipid portion of the anchor for this protein is a single molecule of diacylglycerol. Several mammalian GPI-anchored proteins, such as human acetylcholinesterase (13) and Trypanosoma brucei procyclic acid repeat protein (14), are known to contain an additional acyl chain esterified to the inositol moiety. These proteins are not released by phospholipase C treatment (13).

**N-Glycosidase Treatment Suggests That the T-cell Protein Is Thy-1**—More than 100 different proteins are known to be attached to the plasma membrane by GPI anchors, and several have been reported to be present on T-lymphocytes (15). Of those for which information is available, we could find only two that have molecular masses in the region of 30 kDa. One of these is RT6, which is not expressed by EL4 cells. The other is Thy-1, a glycoprotein that migrates as two or more bands in the region of 30 kDa on SDS-polyacrylamide gels. It is the smallest known member of the immunoglobulin superfamily (16), and in...
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Fig. 3. The proaerolysin-binding component of EL4 cells is released by PI-PLC. Approximately $9 \times 10^6$ cells were digested with the enzyme as described under "Experimental Procedures." Control incubations without the enzyme were carried out in parallel (UNTREATED lanes). Cells (10$^6$ cells) and supernatants were separated by SDS-PAGE and blotted. Proportionally twice as much supernatant as cells was loaded.

Fig. 4. N-Glycosidase treatment reduces the size of the proaerolysin-binding component in EL4 cells. This is comparable to the experiment in Fig. 3. Cells were either untreated or incubated with or without the enzyme. See "Experimental Procedures" for details.

As little as 10$^{-10}$ M aerolysin leads to 100% killing of wild-type cells, whereas 10$^{-8}$ M Po-Pro 1, a membrane-impermeant probe that fluoresces when it intercalates with double-stranded nucleic acid, when aerolysin is added to wild-type cells, there is an increase in fluorescence after a delay that we have found depends on the concentration of the toxin (data not shown). Live/dead cell counts carried out in parallel showed that all of the cells are dead by the time the fluorescence curve plateaus. As little as 10$^{-10}$ M aerolysin leads to 100% killing of wild-type cells in <1 h at 37°C (data not shown). The results of an experiment comparing the effect of a much higher concentration of aerolysin (1.8 \times 10^{-8} M) on wild-type and EL4(Thy-1$^-$) cells are shown in Fig. 7. It is clear that this toxin concentration, nearly 100-fold higher than that needed to kill wild-type cells, had no measured effect on the mutant cells. The survival of the mutant cells was confirmed independently by live/dead cell counts. Comparable experiments showed that BW5147.3(Thy-1$^-$) cells, which also lack GPI-anchored proteins, were much less sensitive than the corresponding wild-type cells.

Fig. 5. Detection of purified Thy-1 using sandwich Western blotting with proaerolysin. The indicated amounts of Thy-1 were separated by SDS-PAGE and blotted to determine the minimum quantity of Thy-1 that could be detected by proaerolysin.

Fig. 6. Aerolysin binding to blotted whole brain homogenates from different species. Approximately the same amount of protein (30 μg) was separated in each lane.

Thy-1 is a major cell-surface component of thymocytes and brain (17). Thy-1 is known to be N-glycosylated at three positions, and it is not O-glycosylated (18). The molecular mass of the protein after peptide N-glycosidase F treatment should be ~14.5 kDa. The results in Fig. 4 show that when EL4 cell samples were treated with peptide N-glycosidase F, the aerolysin-binding component was reduced to a single band migrating with an apparent mass in this range. This result supports the view that the aerolysin receptor is Thy-1. In addition, it eliminates the possibility that it is RT6 since the molecular mass of the amino acid chain of this protein is much higher (19). The possibility that the 47-kDa rat erythrocyte receptor we have described (4) is simply a more heavily glycosylated derivative of the 30-kDa glycoprotein in the T-cell and brain is also excluded by this result. The molecular mass of the erythrocyte proaerolysin-binding protein is reduced only to 32 kDa by peptide N-glycosidase F treatment (4). We have recently obtained the N-terminal sequence of the erythrocyte receptor, which indicates that the protein is related to a small family of GPI-anchored proteins that are involved in ADP-ribosylation reactions.2 Interestingly, there are no apparent sequence similarities between these proteins and Thy-1.

Picogram Amounts of Purified Thy-1 Are Detected Using Aerolysin on Western Blots—To confirm that aerolysin binds Thy-1, a blot containing varying amounts of the purified rat thymus protein in the nanogram and picogram range was probed with proaerolysin (Fig. 5). It may be seen that proaerolysin binding provides a very sensitive method to detect Thy-1 after Western blotting. Less than 1 ng of the GPI-anchored protein could be detected in this way.

Thy-1 from Several Species Binds Aerolysin—The results in Fig. 6 show that Thy-1 in the brains of a number of species may bind proaerolysin. Thus, rabbit, human, and pig brains contain proteins of corresponding size and in similar amounts to the mouse. Comparable binding intensity was not observed in cow and sheep brains. However, we did not determine whether this is because Thy-1 levels are low in the brains of these species (none of the commercially available anti-Thy-1 antibodies we could find will detect Thy-1 on Western blots) or whether it is because the cow and sheep Thy-1 proteins differ from the protein from the other species in some way that affects proaerolysin binding.

Cells That Lack GPI-anchored Proteins Are Less Sensitive to Aerolysin—We used a simple assay to determine if EL4 cells containing surface GPI-anchored proteins are more sensitive to aerolysin than cells without these determinants. EL4 cells were incubated with 1 μM Po-Pro 1, a membrane-impermeant probe that fluoresces when it intercalates with double-stranded nucleic acid. When aerolysin is added to wild-type cells, there is an increase in fluorescence after a delay that we have found depends on the concentration of the toxin (data not shown). Live/dead cell counts carried out in parallel showed that all of the cells are dead by the time the fluorescence curve plateaus. As little as 10$^{-10}$ M aerolysin leads to 100% killing of wild-type cells in <1 h at 37°C (data not shown). The results of an experiment comparing the effect of a much higher concentration of aerolysin (1.8 \times 10^{-8} M) on wild-type and EL4(Thy-1$^-\)$ cells are shown in Fig. 7. It is clear that this toxin concentration, nearly 100-fold higher than that needed to kill wild-type cells, had no measured effect on the mutant cells. The survival of the mutant cells was confirmed independently by live/dead cell counts. Comparable experiments showed that BW5147.3(Thy-1$^-\)$ cells, which also lack GPI-anchored proteins, were much less sensitive than the corresponding wild-type cells.

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Results similar to those in Fig. 7 were obtained when we compared EL4 cells with cells that had been pretreated with PI-PLC. The enzyme-treated cells had greatly reduced sensitivity to the toxin.3

Liposomes Containing Thy-1 Are More Sensitive to Aerolysin—The results of the experiments comparing wild-type and mutant cell lines were convincing evidence that GPI-anchored proteins confer aerolysin sensitivity to T-lymphocytes. However, since these cells may contain several different proteins anchored in this way, we could not conclude that Thy-1 itself was acting as an aerolysin receptor. We have shown previously that incorporation of the rat erythrocyte receptor into planar bilayers increases their sensitivity to aerolysin (4). Since GPI-anchored proteins are easily incorporated into liposome membranes, we used entrapped dye release from large unilamellar vesicles as an assay instead of the more cumbersome planar bilayer assay to determine the effect of purified Thy-1 on the ability of aerolysin to form channels. The results in Fig. 8A show that nanomolar concentrations of aerolysin cause the release of carboxyfluorescein from liposomes containing incorporated Thy-1, but not from control liposomes containing only lipid. As expected, when Thy-1 was removed from the membranes by treating the liposomes with PI-PLC, their sensitivity to aerolysin was abolished (Fig. 8B).

DISCUSSION

Thy-1 is the major protein on the surface of rodent T-cells, and for this reason alone, it is an ideal target for a bacterial toxin like aerolysin. The T-cells of other species contain variable amounts of the protein. For example, in the human, progenitor cells are the richest lymphocyte source (20). The brains of many species are also rich in Thy-1, but it is unlikely that this could be significant in Aeromonas infections. Interestingly, despite the fact that bovine thymocytes (21) are known to contain a homologue of the rat and human Thy-1 proteins, we found little or no aerolysin binding after blotting cow (or sheep) brain samples when compared with the other species we tested. Presumably, this means that the glycoprotein in these two species differs from Thy-1 of the others in some region of the structure that is crucial for proaerolysin binding.

Our results tell us several things about the interaction of aerolysin with Thy-1. The fact that the receptor could easily be detected in cell-free supernatants following treatment with PI-PLC by probing blots with proaerolysin (Fig. 3) is evidence that the diglyceride portion of the anchor is not required for binding. Similarly, since binding was not reduced when the N-linked sugars were removed (Fig. 4), it is clear that this portion of Thy-1 is also not involved in the interaction with the protoxin.

GPI-anchored glycoproteins are thought to be capable of much higher lateral mobility in the plasma membrane than proteins with conventional peptide transmembrane regions (22), and this may be an advantage in promoting channel formation by aerolysin. Before binding, the toxin is a watersoluble dimer, and to form membrane channels, it must oligomerize and form insertion-competent heptamers (1). Presumably, the toxin, once bound to the receptor, moves laterally in the membrane to form the oligomer. Clearly, the abundance of Thy-1 on murine T-cells should facilitate binding, and the lateral mobility of the receptor should facilitate oligomerization.

It is also easy to speculate that binding to a small protein molecule like Thy-1, close to the cell surface and lipid-anchored, may make it easier for the aerolysin oligomer to insert into the bilayer. Interestingly, such a function has already been proposed for CAMPATH-1, a small GPI-anchored polypeptide on human lymphocytes (23). Monoclonal antibodies against this molecule are very effective for complement-mediated cell lysis (24).

Thy-1 is not the only membrane protein that can act as a

3 K. L. Nelson, S. M. Raja, and J. T. Buckley, unpublished observations.
receptor for aerolysin. We have previously characterized such a protein in the rat erythrocyte (4), and the comparison of mouse tissues by sandwich Western blotting in Fig. 1 identifies a few other bands that may represent proteins with affinity for the toxin. Our recent evidence indicates that the rat erythrocyte protein is a member of another family of proteins that are unrelated to Thy-1, except that they all also contain GPI anchors. It is worth noting that Bacillus thuringiensis δ-toxin has recently been shown to bind to a GPI-anchored aminopeptidase in the midgut of the insect Manduca sexta (25).

Like many other toxins, aerolysin has long been classified as a channel-forming hemolysin, and it is clear that it destroys erythrocytes by breaching the permeability barrier. The mechanism by which it causes death in other cells, such as T-lymphocytes, is worth further study. It seems likely that at high concentrations, where channels would be formed at a rate that would overwhelm the cell, the cause of death may be comparable to that in the erythrocyte. However, at low toxin concentrations, other causes of death may be more important, and they may result from the binding of the toxin to receptors like Thy-1. Although the functions of many GPI-anchored proteins are unknown, there is evidence for some that clustering results in the generation of an intracellular signal. For example, clustering of Thy-1 molecules as a result of binding by an anti-Thy-1 antibody has been shown to cause lymphocyte apoptosis (26). Since oligomerization of aerolysin should promote clustering of Thy-1 in the same way as the antibody, we should expect to see aerolysin apoptosis under some conditions. Aerolysin should prove to be a useful tool in the study of apoptosis and in related studies of the function of Thy-1 and other GPI-anchored proteins.

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