Physiology and Genetics of Virulence

Antibody-mediated immunity to group B streptococcus

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Group B streptococcus can be a serious infection of neonates and, in the past, specific antibody has been shown to mediate immunity against this organism. However, specific antibody is not required for phagocytosis of highly pathogenic organisms by neutrophils. Furthermore, the bacteria subsequently survive and tend to accumulate or multiply in these cells, which they then disrupt. Highly pathogenic streptococci are killed by neutrophils in the presence of specific antibody. Moreover, the addition of specific antibody to neutrophils after phagocytosis of the organisms had occurred stimulated the phagocytes to kill the ingested bacteria. This post-phagocytic role for antibody in the killing of bacteria by neutrophils, as opposed to their ingestion, has not been previously described. Once inside the cell, specific antibody binds to protective surface determinants on the bacteria and allows the organisms to be killed. Present work involves an examination of the surface virulence determinants that are responsible for the survival of highly pathogenic group B streptococci within neutrophils.

Adsorption of fibronectin to Teflon followed by *S. aureus* adherence

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Infection of implanted prostheses or catheters represent a major problem in modern medicine. We have recently demonstrated that fibronectin (FN) is an important ligand of *Staphylococcus aureus* adherence to polymethylmethacrylate (PMMA) material, which was implanted into animals and used as an experimental model of foreign body infection (J. infect. Dis. *150*, 546). In a related in vitro study, we described FN adsorption by PMMA coverslips (J. infect. Dis. *45*, 768). Using similar techniques, we studied quantitative adsorption of ^{125}I-labeled FN and promotion of *S. aureus* adherence onto Teflon®, which is a widely used material in the clinic. The adsorption of purified FN for Teflon was dose-dependent and resembled that for PMMA. Promotion of *S. aureus* adherence by Teflon® adsorbed FN was dose-dependent, leading to increased bacterial adherence up to a maximum of 6.5×10^4 bacteria per coverslip. In contrast, Teflon unexposed to FN bound 3×10^3 bacteria by coverslip. FN adsorption on Teflon® and its promotion of bacterial adherence were prevented by other serum proteins (= FN-depleted serum pool). In summary, FN adsorption and its promotion of *S. aureus* adherence by Teflon are similar to those found previously with PMMA.

Mechanisms of bacterial persistence in the long-term catheterized bladder

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A prospective study of urinary tract infections in long-term catheterized spinal injury patients revealed their complexity. Mixed bacterial communities of up to eight multi-drug resistant species were commonly isolated. In the absence of chemotherapy some species were persistent and others transient inhabitants. In vitro, tests showed that there was no correlation between adherence of the bacteria to bladder uroepithelial cells and persistence. *Pseudomonas aeruginosa* gave significantly higher adherence to silicone- and teflon-coated catheter sections than all the other isolates. Overall however there was no relationship between persistence and the ability to adhere to the catheters. The observed emergence of *P. aeruginosa* during the chemotherapy may be explained by its growth and adherence characteristics. Adherence exhibited by this organism appeared not to be related to the surface hydrophobicity or to the possession of fimbriae.

The regulation of hemolysin secretion in *E. coli* strains of patients from Bern and Essex

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In the past years results from several groups point to a contribution of hemolysin production to *E. coli* virulence. We investigated the occurrence of hemolytic *E. coli* strains and plasmid encoded hemolysin production. From 1818 screened strains 395 showed hemolytic activity where of 5 strains contained a Hly-plasmid. These plasmids were compared with 2 Hly-plasmids isolated in Essex regarding their genetic and phenotypical properties. We found the following similarities: 1) a high transfer-frequency, 2) a derepressed state and 3) the suppression of their hemolysin secretion by $FeCl_3$. The plasmids from Bern differed from those of Essex in the much lower amount of secreted hemolysin. Furthermore the hemolysin secretion was increased by iron chelators (2,2'-dipyridil and others) only in strains containing the plasmids isolated in Bern. By means of cloning and hybridization experiments strong homology in the structural genes and differences in the flanking regions of the Hly-genes in plasmids from Bern and Essex were found. However, the left flanking region among the Bernese plasmids seemed to be identical. Therefore we suggest that the genes responsible for the induction of hemolysin secretion by iron chelators are located in this region.

Inhibition of *Pseudomonas aeruginosa* elastase production by subinhibitory concentration of gentamycin

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Proteases, mainly elastase produced by *Pseudomonas aeruginosa* (PA) are thought to mediate tissue destruction in patients with burns and cystic fibrosis (Pavlovski, Rev. infect. Dis., 1983). We characterized the kinetics of elastase production during bacterial growth in vitro in strains of PA isolated from CF patients in the presence and absence of gentamycin. MIC for gentamycin was 0.5 µg/ml in five of these strains. 10^3 PA were inoculated in 15 ml of broth and grown with various concentrations of gentamycin. Viable counts and elastase activity were determined at various time points. Without antibiotics, elastase production was first detected at 16 h of culture growth with a peak at 34 (27.3 mg of elastin-H^3 solubilized at 37 °C/ml of supernatant/h). Even at subinhibitory concentrations of gentamycin, without impairment of bacterial growth, a decrease in elastase production was found. Similar results were obtained for all five strains at 20 h of bacterial growth. These results suggest that treatment with gentamycin reaching only subMIC (such as found in CF bronchial secretions) may still be efficient through inhibition of bacterial production of elastase.