

Microbiology Learning Objectives C15
Microbial Mechanisms of Pathogenicity

1. What is pathogenicity?
2. What is virulence?
3. What do we call the microbe's pathway for entry into the body which results in a disease state? What are the three common entry pathways?
4. What must happen for most pathogens before they can cause a disease state? What is the relationship between *Streptococcus mutans* and *Actinomyces*?
5. What percent of human bacterial infections involve biofilms?
6. What is another term used to describe the capsule associated with some bacteria? Do capsules increase or decrease virulence? What is significant about capsules associated with *Streptococcus pneumoniae*?
7. What is the benefit of the cell wall component called the M protein to *Streptococcus pyogenes*?
8. What is the benefit of the cell wall component called Opa to *Neisseria gonorrhoeae*?
9. How does *Mycobacterium tuberculosis* increase its virulence?
10. What is an exoenzyme?
11. How does *Staphylococcus* use coagulases to increase its virulence?
12. How can *Streptococcus pyogenes* and *Staphylococcus aureus* use kinases (streptokinase) to increase its virulence?
13. How is hyaluronic acid used to form body tissues? How do bacteria like *Streptococcus* spread rapidly through tissues?
14. How is antigenic variation used to increase bacterial virulence?
15. *E. coli* may actually enter into the cytoplasm of an epithelial cell. What protein allows *E. coli* to create a "membrane ruffling" which is the key to entering the host cell's cytoplasm?
16. How do bacteria "steal" iron from multicellular hosts?

17. How may *E. coli*, *Shigella*, *Salmonella*, and *Neisseria gonorrhoeae* cause direct damage to host epithelial cells?
18. How do bacteria cause the most damage to human cells?
19. What is an exotoxin?
20. What is an endotoxin?
21. What is an antitoxin?
22. What is a toxoid?
23. What are the three principle types of exotoxins?
24. In the A-B toxin mechanism, what is the function of the A component and the B component?
25. What two actions are initiated by membrane-disrupting toxins?
26. *Staphylococcus* and *streptococci* both produce hemolysins which lyse RBC. What other type of cell is destroyed by hemolysins?
27. What are superantigens?
28. How do endotoxins differ from exotins?
29. Why might antibiotics make a gram negative infection symptoms and signs worst?
30. How do lipid A exert their effect?
31. What is shock? Septic shock?
32. What are portals of exit?