Pathogenicity and virulence

Pathogenicity is the ability of microbes to cause disease in a particular host species. Such diseases are caused by microbes, which we call pathogens. **Virulence** is then a quantitative expression of the pathogenicity of a certain strain of bacteria.

Pathogenicity

Pathogenicity factors are genetically encoded. Either in the bacterial chromosome (most strains of a given species are able to act pathogenically) or in plasmids. The presence of genetic information on plasmids gives bacteria **enormous veriability**, which is the cause of variable virulence of strains. The simultaneous occurence of several pathogenic genes may give rise to new, highly virulent clones (carnivorous streptococci, *Neisseria meningitidis*).

According to the host's susceptibility, pathogens can be divided into:

Primary pathogens (obligatory)
: They have the ability to cause disease in healthy people. There are few of these species and preventive vaccination directed againt them. These pathogens include: *Streptococcus pyogenes*, *Treponema pallidum*, *Shigella*, *Vibrio cholerae*, *Bordetella pertussis*, *Salmonella typhi*, *Neisseria gonorrhoeae*, *Bacillus anthracis*, *Corynebacterium diphtheriae*, *Mycobacterium tuberculosis*, *Yersinia pestis*.

Oppotunistic pathogens (conditionally pathogenic)
: Species are able to be pathogens only when the **defense mechanisms are damaged** (injuries to the skin, mucous membranes, impaired physiological processes, after medical procedures) and **immunity is reduced**. The source of infection can be pathogens from the external environment, but also from their own flora (originally symbiotic bacteria). For example, *Escherichia coli*.

Virulence

The individual property indicated the degree of pathogenicity of a particular strain of microbe. Within a pathogenic specues, strains can be **highly virulent** (killing most of their hosts, thereby losing the ability to spread), **virulent** (surviving and can spread) to **avirulent**.

LD_{50} (50% lethal dose) is used to determine virulence. Virulence may be increased, for example, by repeated transmission of the strain to the same host species, which is a complication of nosocomial infections.

We can artificially reduce virulence, which is called attenuation. This can be used in the preparation of vaccines.

Pathogenicity factors

Portability

The portability of the microbe depends mainly on:

- the number of microbes excreted from the organism;
- microbial resistance;
- infectious dose (low in *Mycobacterium*, *Shigella*);
- host behavior (cough, sneezing, diarrhea, behavior change).

Transmission is usually mediated by, for example, the saliva, the fecal-oral route, sexually, or via an animal vector (fleas, cats,etc.).

Toxicity

The ability of a microbe **to harm a host**. Bacteria are toxic through their toxins – endotoxins a exotoxins. Damage to the body can be **direct**, which can lead to cell death (microbially induced apoptosis), metabolic damage or mechanical damage (parasites, diphtheria). Or the body may be damaged by **a defensive reaction to the microbe** – inflammation, edema, scars, immunopathological hypersensitivity.

Invasiveness

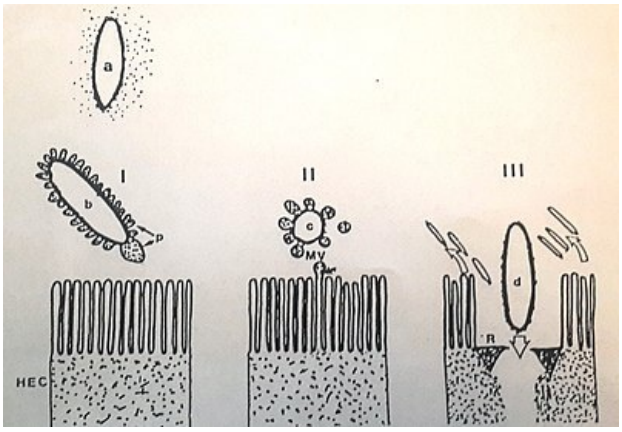
Invasineness is the ability of a microorganism **to enter a host**. It depends on several factors, adherence, penetration into the internal environment, proliferation in tissues, spread within the body and overcoming the host's defense mechanisms.

Adherence

Adherence is the ability of bacteria to **attach to and remain** on the host. Bacteria for adherence use a variety of mechanisms by which they attach to cell surfaces. Bacteria can adhere through fimbriae or adhesins (they bind to cellular receptors).

Penetration

Penetration of the bacterium into the cell can occur in a direct manner, forced by phagocytosis, the enzyme production or in an unknown manner. **Direct manner** can penetrate intact skin (leptospira), small cracks in the skin or mucosa (staphylococci, streptococci), stinging insects (*Yersinia pestis*) or enzymes (*Clostridium perfringens*).
Forced phagocytosis is invasive. These bind to the integrins. This causes the phagocytic vacuole to rupture and the bacteria to be expelled by the cell's actin system.



Salmonella bacterial invasion

Bacteria can enter cells **after cell breakdown or between cells**. Bacteria can also produce enzymes that disrupt the cell wall. These enzymes are also called spreading factors.

Ability to spread

Bacteria can spread to their site of action in the host organism. The methods of dissemination can be:

- via lymph– *Mycobacterium tuberculosis*, *Treponema pallidum*, pyogenic cocci, viruses;
- via blood– *Salmonella typhi*, generalized infections.;
- *per continuitatem* (direct transfer) – *Streptococcus pyogenes*, *Clostridium perfringens*, *Borrelia burgdorferi*;
- along the nerves – herpesviruses, rabies.

Ability to multiply

Pathogenic bacteria can multiply in the body if they find all the conditions necessary for their metabolism. **These requirements vary** from one bacterial species to another. The action of antibiotics focuses on these demands.

Bacteria are **dependent on the gain of iron** from the environment. They can be obtained from the cells by disrupting the membrane with hemolysins. They obtain them from the proteins that transport iron by occupying the binding site for iron with teir own compound - so-called siderophores and taking the free iron. Increased iron concentration increases the virulence of all pathogenic bacteria. This fact is a complication of procedures where serum iron levels increase (blood transfusions, leukemia, hemochromatoses, sickle cell disease).

Ability ro overcome the host's defense mechanisms

This is an important factor in the pathogenicity of bacteria.

Complement resistance
The surface of the bacteria triggers the body's response. The first is the complement reaction. It takes place immediately after the bacteria penetrate the body. **G+ bakteria** (thick layer of peptidoglycan) are better resistant to this process. In addition, they may bind to their receptors another substance (eg. IgA), that does not activate complement (*Neisseria meningitidis*).
Bacteria can also produce inhibitors of complement activation (sialic acid) or they can produce enzymes that break down complement components.

Resistance to phagocytosis
Bordetella pertusis toxin is able to paralyze neutrophils and thus prevent their activity. Bacteria of psudomonads, staphylococci, streptococci and histotoxic clostridia produce leukocidins, which are substances that destroy leukocytes only after phagocytosis (from the inside).
An important factor in pathogenicity is the **bacterial capsule**, which prevents the phagocyte from adhering to the bacterial surface. Another way is, for example, **escape from a phagosome** (*Ricketssia*), prevention of fusion of a phagosome with a lysosome (*Mycobacterium tuberculosis*), inhibition of chemotaxins by succinic acid, elastase, etc.

Escape from specific immunity
An effective mechanism is **rapid multiplication**, when specific immunity does not have time to intervene (respiratory viruses, diarrhea, malaria). Another way is to deceive the host by having the same antigens on their surface as the host cell. Bacteria are recognized by the body as the body's own cells(*Neisseria meningitidis*). Other species may alter the antigenic specificity of their surface antigens (*Neisseria gonorrhoeae*, influenza, HIV, *Borrelia recurrentis*). An important factor is the production of a specific protease that cleaves IgA antibody. Cleavage causes no toher immunoglobins (*Haemophilus influenzae*) to bind to the epitopes. Furthermore, the microbe may be hidden (*Herpes simplex*).

Links

related articles

- The relationship between host and microbe
- Bacterial toxins

Source

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