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BACTERIAL DISEASES: HUMANS VS MICROORGANISMS

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ABSTRACT

Bacteria, one among the group of microorganisms like virus, protozoa and fungi, is universally present in nature. It is in air, water and soil, the basic elements of life closely associated with all living organisms. Present both in the internal and external environment bacteria constitute both beneficial and pathogenic microbes. The beneficial ones contribute to a healthy living, but a slight change in the environment can cause various diseases. This article reviews about the varied taxonomy, morphology, pathogenesis and identification of bacteria and also the various diseases that are caused by them.

KEYWORDS: Bacteria, one among the group of microorganisms like virus.

INTRODUCTION

Throughout history, there has been a continual battle between humans and the multitude of microorganisms that cause infection and disease.[1] The earliest discovery of a pathogenic microorganism was made by Agostino Bassi (1853).[2] Bacteria are the dominant form of life on the planet. There are 105 cells in a mms of sea water and ten times as many bacterial cells on our skin and in our large intestine as cells in our own body.[3]

The oral cavity may serve as a reservoir for many pathogenic bacteria. Opportunistic oral bacteria have been documented in causing systemic diseases.[4] The mouth is similar to other environmentally-exposed sites in the body in having a characteristic (autochthonous) and diverse micro flora in health.[5] Infectious diseases are the leading cause of death worldwide. Not only are new infectious diseases emerging, but the re-emergence of deadly infectious diseases, and the increasing prevalence of antimicrobial resistant strains, present a formidable threat to public health.[6]

Bacterial taxonomy

The methods now employed for bacterial systematics include, the complete 16S RRNA gene sequencing, 16S RRNA because it is ubiquitous among prokaryotic life. Second, its size and high degree of functional conservation result in clock-like mutation rates throughout prokaryotic evolution. Third, the 16S RRNA gene includes both conserved regions, which can be used for designing amplification primers across taxa, as well as nine hyper variable regions (V1-V9), which can be effectively used to distinguish between taxa and its comparative analysis by phylogenetic trees.[7]

Bacterial morphology

Bacterial morphology helps optimize interactions between cells and the surfaces to which they attach. Rod shapes may allow cells to attach more readily in environments with shear stress, by allowing cells to form uniform mat-like sheets. Filamentous cells have more surface area for long-term attachments and can entwine themselves with porous surfaces. Cocci may have access to small pores in a substrate, creating more attachment sites per cell and concealing themselves from external shear forces.[8]

Pathogenesis of bacterial diseases

Infectious diseases are the leading cause of death worldwide. Many diverse bacterial pathogens share common mechanisms in terms of their abilities to adhere, invade, and cause damage to host cells and tissues, as well as to survive host defences and establish infection.[6]

Capsule- Some bacteria overproduce and excrete copious amounts of high molecular weight polysaccharides, also called exopolysaccharides. This extracellular sugar coating is termed capsule. Capsule provides bacteria with protection from the host immune response as well as antibiotics.[6]

Cell wall- Bacteria can be divided into two major groups based on differences in cell wall structure: Gram positive and Gram negative bacteria.

Characteristic	Gram-positive	Gram-negative
Gram reaction	Retain crystal violet dye and stain dark violet or purple	Can be decolorized to accept counterstain (safranin); stain red
Peptidoglycan layer	Thick (multilayered)	Thin (single-layered)
Teichoic acids	Present in many	Absent
Periplasmic space	Absent	Present
Outer membrane	Absent	Present
Lipopolysaccharide (LPS) content	Virtually none	High
Lipid and lipoprotein content	Low (acid-fast bacteria have lipids linked to peptidoglycan)	High (due to presence of outer membrane)
Flagellar structure	2 rings in basal body	4 rings in basal body
Resistance to physical disruption	High	Low
Inhibition by basic dyes	High	Low
Susceptibility to anionic Detergents	High	Low
Resistance to sodium azide	High	Low

Toxins- Are analogous to biological weapons in that these are proteinaceous or non proteinaceous molecules produced by bacteria to damage the host cell. Proteinaceous toxins (exotoxins) are generally enzymes which are delivered to eukaryotic cells by two different methods: (1) secretion into the surrounding milieu or (2) direct injection into the host cell cytoplasm. Bacterial exotoxins can be roughly categorised into four major types based upon their amino acid composition and function: (1) A-B toxins, (2) proteolytic toxins, (3) pore forming toxins, and (4) other toxins.[6]

Adhesins- A key step in the host-pathogen interaction is adherence of the pathogen to host surfaces. Once adhered to a specific host cell surface, the pathogen is then able to initiate its specific biochemical processes that results in disease.[6]

Invasion- Once adhered to a host surface, some pathogens gain deeper access into the host to perpetuate the infection cycle. This pathogenic principle, termed invasion, can be divided into two types: extracellular and intracellular. These species secrete several enzymes that degrade host cell molecules.[6]

Bacterial identification

Bacterial identification initially entails:

- Inspection of the colonial characteristics: size, shape, elevation, margin, color, smell & texture
- Examination of microscopic morphology & staining characteristics: a stained film of the colony helps identification
- Identification of growth condition: aerobic, anaerobic, capnophilic.[2]

Principles of identification:

- Two major categories-

A. Based on genotypic characteristics of bacteria
B. Based on phenotypic characteristics.[2]

Bacterial diseases

Tuberculosis

Tuberculosis (TB) is one of the chronic granulomatous lesions affecting various body systems. It is usually caused by *Mycobacterium tuberculae*, although *Mycobacterium bovis*, *Mycobacterium kansasii* and *Mycobacterium scrofulaceum* have also been implicated. The main cause of TB is *Mycobacterium tuberculosis*, a small, aerobic, nonmotile bacillus. It divides every 16 to 20 hours, which is an extremely slow rate. Mycobacteria have an outer membrane lipid bilayer and stain weakly for "Gram-positive" dye.[9]

Pulmonary Tb

If a tuberculosis infection does become active, it most commonly involves the lungs. Symptoms may include chest pain and prolonged cough producing sputum. Occasionally, people may cough up blood in small amounts. Sometimes, the infection may erode into the pulmonary artery, resulting in massive bleeding (Rasmussen's aneurysm).[10]

Extrapulmonary Tb

In 15–20% of active cases, the infection spreads outside the lungs, commonly seen in immunosuppressed persons and young children. Common sites include the pleura, the central nervous system, the lymphatic system, the bones and joints. TB in many cases follows a general pattern as described by Wallgren. In the first stage, after *M.tuberculosis* contained in inhaled aerosols becomes implanted in alveoli, the bacteria are disseminated to regional lymph nodes in the lung, forming the so-called primary or Ghon complex. The second stage, lasting about 3 months, is marked by hematogenous circulation of bacteria to many organs.[9] Pleurisy or inflammation of the pleural surfaces can occur during the third

stage. The last stage, where the disease does not progress, several extrapulmonary lesions may develop.[10]

Oral lesions in Tuberculous may be either primary, or secondary to pulmonary tuberculosis. The typical oral lesions consist of a stellate ulcer, most commonly on the dorsum of the tongue. The ulcer may be ragged and indurated and is often painful. Tongue is the commonest site for oral tuberculous lesions.[9]

Syphilis

Syphilis, a chronic, systemic sexually transmitted infection, is caused by the spirochaete; *Treponema pallidum*. (25) The primary mode of transmission is by sexual contact, or by transfer across the placenta.[11] Primary syphilis of the mouth manifests as a solitary ulcer usually of the lip or, more rarely, the tongue. The ulceration is usually deep, with a red, purple, or brown base and an irregular raised border. The features of secondary syphilis reflect the hematogenous spread of *T. pallidum*. The 2 principal oral features are mucous patches and maculopapular lesions.[12]

Maculopapular lesions include

- *Macular syphilides*: Macular lesions tend to arise on the hard palate as slightly raised, firm, red lesions.
- *Papular syphilides*: Manifest as red, raised, firm round nodules with a grey center.
- *Mucous patches*: Manifest as oval-to-crescenteric erosions, covered by a grey mucoid exudate with an erythematous border. The mucous patches may coalesce to give rise to, serpiginous lesions termed “snail track ulcers”.

The oral complications of tertiary syphilis center upon gumma formation. Gummas tend to arise on the hard palate and tongue. A gumma manifests initially as one or more painless swelling. When multiple, they tend to coalesce, develop into areas of ulceration, with areas of breakdown and healing.[12]

Leprosy

Leprosy, also known as Hansen's disease (HD), is a chronic infection caused by the bacterium *Mycobacterium leprae*.[13] its incubation period ranges from 6 months to 20 years, due to its very slow growth. Bacterial culture is not possible. Leprosy has no specific vaccine against *M. leprae*, also diagnostic and prognostic tests are not feasible in clinical routine.[14]

It is primarily a granulomatous disease of the peripheral nerves and mucosa of the upper respiratory tract; skin lesions are the primary external sign. Left untreated, leprosy can cause permanent damage to the skin, nerves, limbs and eyes. Leprosy does not cause body parts to fall off, although they can become numb or diseased as a result of secondary infections. These infections, in turn, can result in tissue loss causing fingers and toes to become shortened and deformed, due to cartilage resorbtion.[13]

The skin may show macules, plaques, papules, nodules, diffuse infiltration, secondary lesions like burns, blisters, fissures, ulcers and scars. Lesions, may be present with extreme variability in colour, size, edge, texture, location, distribution and number.[15]

Actinomycosis

Actinomycosis is a chronic disease characterized by abscess formation, tissue fibrosis, and draining sinuses. It is caused by non-spore-forming, anaerobic or microaerophilic bacterial species of the genus *Actinomyces*.[16] Of the 14 *Actinomyces* species, six may cause disease in humans, including *A. israelii, A. naeslundii, A. odontolyticus, A. viscosus, A. meyeri,* and *A.* Gerencseriae.[17]

They are opportunistic pathogens that rely on breaks in the normal mucosa for portals of entry. Among oral mucosal membrane, actinomycosis usually involves the tongue and the oro-alveolar mucosa.[18] The developing granulomatous and suppurative lesions disrupts the mucosa and progresses into the deeper tissues producing a permanent discharge of purulent exudates.[17]

A general characteristic of actinomycosis includes- chronic disease state, suppuration, formation of draining sinuses or fistulas as well as presence of “sulphur granules” in exudates. “Sulfur granules” are small, white-to-yellow granular aggregates of bacterial filaments, 1-5mm in diameter. Healing with scar formation in one area and formation of new sinuses in contiguous tissues are typical of actinomycosis.[19]

Diptheria

It is an acute, life-threatening, infectious, and communicable disease of the skin and mucous membrane caused by toxemic strains of *Corynebacterium diphtheriae*, anaerobic gram-positive bacteria. There are 4 biotypes of the bacterium (gravis, mitis, intermedius, and belfanti), and each differs in the severity of disease it produces. The disease is characterized

by local inflammation and formation of a greyish adherent pseudo membrane, which bleeds on removal.[20]

Diphtheria is an acute bacterial disease that can involve almost any mucous membrane. Symptoms include sore throat, fever, and malaise, difficulty in swallowing and breathing. Individuals with severe disease may develop neck swelling and enlarged neck lymph nodes, leading to a "bull-neck" appearance. Extension of the pseudo membrane into the larynx and trachea can lead to obstruction of the airway with subsequent suffocation and death.[21]

Scarlet fever

Scarlet fever is caused by infection with group A Streptococcus (GAS). It begins on the upper trunk, travelling distally and sparing the soles and hands. Characteristic clinical features include: an initial white covering of the tongue, followed by enlargement of the papillae, giving a distinctive ‘strawberry tongue’ appearance, flushing of the cheeks and circumoral pallor.[22]

Diagnosis is not established until the characteristic diffused, bright, scarlet-skin rash appears on the second or third day of illness. Small papules of normal colour erupt through these rashes giving a characteristic sandpaper feel to the skin and is called “pastia sign”.[23]

Noma

Noma is a devastating infectious disease which destroys the soft and hard tissues of the oral and para-oral structures. Also referred to as "cancrum oris” is described as a "gangrenous affection of the mouth, especially attacking children in whom the constitution is altered by bad hygiene and serious illness, especially from the eruptive fevers, beginning as an ulcer of the mucous membrane with edema of the face, extending from within out, rapidly destroying the soft parts and the bone, and almost always quickly fatal.”[24]

The mucous membrane becomes inflamed and develops an ulcer. The infection then spreads to the skin causing necrosis of the lips and cheeks. Foul smelling, purulent oral discharge, associated with profuse salivation, anorexia and palpable cervical lymphadenopathy.[25]

Tetanus

Tetanus is now a rare disease in the developed world. Is caused by a Gram-positive bacillus, Clostridium tetani.[26] Tetanus toxin is a zinc-dependent metalloproteinase that targets a protein (synaptobrevin/vesicle-associated membrane protein—VAMP) that is necessary for

the release of neurotransmitter from nerve endings through fusion of synaptic vesicles with the neuronal plasma membrane.[27]

A clinical trial of rigidity, muscle spasms and, autonomic dysfunction is seen. Neck stiffness, sore throat, and difficulty in opening the mouth are early symptoms. Masseter spasm causes trismus or "lockjaw".[26] Spasm progressively extends to the facial muscles causing "risus sardonicus", and muscles of swallowing causing dysphagia.[27]

Pyogenic granuloma

Pyogenic granuloma (PG) is a kind of inflammatory hyperplasia. There are two kinds of PG namely lobular capillary hemangioma (LCH type) and non-LCH type, which differs in their histological features. Oral PG, affects the lips, tongue, and buccal mucosa. The surface is characteristically ulcerated and friable, may be covered by a yellow, fibrinous membrane and its color ranges from pink to red to purple, depending on the age of the lesion.[28]

Erysipelas

This disease affects epidermis and superficial dermis of the face, legs, and other sites and may also involve the lymphatics. Group A β hemolytic streptococcus (GABHS, *Streptoccocus pyogenes*) is the usual etiologic agent. Patients typically have a small erythematous patch that rapidly becomes bright red, edematous, indurated, and shiny with well-defined borders.[29]

Granuloma inguinale (donovanosis)

Donovanosis, is a chronic, benign condition caused by an intracytoplasmic, gram-negative bacillus called *Klebsiella granulomatis*. It begins with a nodule at the site of bacterial inoculation, which bursts, leading to formation of a painless ulcer that grows slowly and bleeds easily.[30] Four clinical patterns of disease presentation are seen. The commonest is the ulcero-granulomatous type: presents with a single, well-defined, friable, beefy red, non-tender, non-indurated ulcer with profuse granulation tissue that bleeds on touch. The necrotic type is most often seen in patients with long-standing donovanosis. The hypertrophied type, extensive lesions involve the genitalia and surrounding areas. The sclerotic type is an uncommon form, characterised by early formation of extensive fibrous tissue.[31] Ulcero vegatative form: there is abundant granulation tissue at the base of the lesion, which bleeds easily.[30]

Impetigo

Impetigo is a contagious superficial bacterial skin infection, most frequently encountered in children. It is typically classified as primary and secondary impetigo.[32] *Staphylococcus aureus*, is the main causative organism for this infection. Impetigo is classified as bullous or non-bullous type. The initial lesion is a thin-walled vesicle on previously normal skin that rapidly ruptures leaving a superficial eroded area covered with honey-coloured crusts. These crusts dry and disappear leaving a red mark that heals without scarring.[33]

Cat-scratch disease

Cat-scratch disease (CSD) typically features subacute regional lymphadenitis with an associated inoculation site papule due to a cat scratch or bite. *Bartonella henselae* is the major etiologic agent associated with the necrotizing granulomatous lesions of classic CSD.

Subacute Regional Lymphadenitis- The most typical form of CSD manifests as tender, regional lymphadenopathy. Histologic changes include characteristic nodal lesion of a stellate microabscess surrounded by a rim of granulomatous inflammation.

Oculoglandular CSD- Patients with conjunctival inoculation sites often have unilateral conjunctivitis with ipsilateral preauricular adenopathy, sometimes referred to as *Bartonella*-related Parinaud oculoglandular syndrome. Histopathologically shows ulcerated epithelium with a mixed inflammatory infiltrate and focal necrosis.[34]

Tularemia

Tularemia is a disesase caused by *Francisella tularensis*, a gram-negative bacterium. Two types of *F. tularensis* (A and B) occur. Type A organisms are classified as *F. tularensis* biovar *tularensis*, and Type B organisms are classified as *F. tularensis* biovar *holarctica*. Infection usually falls into one of the following categories:

Glandular - Present with enlarged and painful lymph nodes that may become filled with pus.

Oculoglandular - Presents with pus-producing conjunctivitis and enlarged lymph nodes.

Oropharyngeal - After ingestion of bacteria in contaminated food or water.

Pneumonic – Due to inhalation of organisms.

Ulceroglandular - Present with large, tender lymph nodes and a non-healing skin ulcer.

Typhoidal.[35]

Rhinoscleroma

Rhinoscleroma is a chronic bacterial infection caused by *Klebsiella rhinoscleromatis*, a Gram-negative, non-motile, encapsulated bacillus. Rhinoscleroma generally progresses in three stages: the initial stage is the catarrhal or exudative phase, second, proliferative or granulomatous stage and third, cicatricial stage. Initially persistent rhinitis and mucopurulent discharge is seen. In the second stage, inflamed mucosa coalesces to form granulomas. These granulomas may infiltrate other portions of the airway and scar, leading to cicatricial stage. Histopathologic evidence of rhinoscleroma includes granulomatous inflammation with vacuolated histiocytes known as "Mikulicz cells."[36]

Botryomycosis

Botryomycosis is an uncommon bacterial disease that clinically and histologically mimics infection by *Actinomyces* species. It is defined by the histological presence of eosinophilic material surrounding densely packed microorganisms associated with a suppurative focus. This unique pattern became known as the Splendore-Hoeppli phenomenon after the two scientists who described it.[37]

The most characteristic feature of the disease is that bacteria, instead of spreading throughout the infected tissue, group together to form conglomerates.[38] Botryomycosis may present in cutaneous or visceral forms. Cutaneous form presents with chronic, suppurative and granulomatous skin lesions. Visceral form is usually with pulmonary involvement, associated with cystic fibrosis.[39]

Sinusitis

Sinusitis can be broadly defined as inflammation of one or more of the paranasal sinuses. Classically, sinusitis is characterized as the following:

Acute- symptoms last less than 4 weeks, Subacute- symptoms last 4 to 8 weeks, Chronic- symptoms last longer than 8 weeks, Recurrent- three or more acute episodes a year.[40]

Gonorrhea

Is primarily a venereal disease affecting the male and female genitourinary tract. It is an infection of epithelium and commonly manifests as cervicitis, urethritis, prostatitis and conjunctivitis. *N. gonorrhoeae* is a gram-negative, intracellular, aerobic diplococcus.[41]

Infection is caused by *Neisseria gonorrhoeae*. Infection is less frequently found in the pharynx, rectum, conjunctivae, liver capsule, skin, heart valves, joints, and meninges, or it can be disseminated in the bloodstream.[42]

Disseminated gonococcal infection typically manifests as arthritis, tenosynovitis, and dermatitis but can also present as perihepatitis.[41]

CONCLUSION

The ability to measure bacterial diversity is a prerequisite for the systematic study of bacterial biogeography and community assembly. Our estimates are hampered by a lack of data on the abundance of even the most abundant organisms in the environment. However, we are confident that more quantitative data will become available in the near future. This in turn will allow us to refine our extrapolations.

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