General Considerations on Infectious Diseases

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Ralph M. Trüeb

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As opposed to noncommunicable diseases, i.e., cardiovascular diseases (heart attack and stroke), cancers, respiratory diseases (chronic obstructive pulmonary disease and asthma), and diabetes, which represent the leading cause of death globally today, infectious diseases, also known as communicable diseases or transmissible diseases, are an illness resulting from the invasion of an organism's body tissues by disease-causing agents, their multiplication, and the reaction of host tissues to the infectious agents and the toxins they produce. They can be spread from one person to another and under certain conditions may cause a large number of people to get sick.

Infections are caused by a wide range of pathogens, including bacteria, viruses, fungi, parasites, and arthropods.

R. M. Trüeb (✉)
Dermatologische Praxis und Haarcenter Professor Trüeb, Wallisellen, Switzerland
e-mail: r.trueeb@derma-haarcenter.ch

R. M. Trüeb et al. (eds.), *Hair in Infectious Disease*,
https://doi.org/10.1007/978-3-031-30754-6_2

The infectious agents are spread in a number of ways including:

- Physical contact with an infected person, through contact with skin, sexual contact, fecal/oral contact, or respiratory droplets
- Mother to unborn child
- Contact with a contaminated surface or object, food, blood, or water
- Bites from insects or animals capable of passing the specific disease
- Through the air

Epidemiology is the study and analysis of who, why, and where disease occurs and what determines whether various populations have a disease. Epidemiologists may determine differences among groups within a population, such as whether certain age groups have a greater or lesser rate of infection, whether groups living in different environs are more likely to be infected, and by other factors, such as gender and ethnicity. Researchers also may assess whether a disease outbreak is sporadic or just an occasional occurrence; endemic, with a steady level of regular cases occurring in a region; epidemic, with a fast arising and unusually high number of cases in a region; or pandemic, which is a global epidemic.

Contagious skin diseases present with a wide range of symptoms. Some have similarities such as rashes, but most are very different.

If the cause of the infectious disease is unknown, epidemiology can be used to assist with tracking down the sources of infection.

The control of certain communicable diseases is required by law. The respective public health office prevents and controls these diseases through prevention programs, including immunization, and by monitoring and following up on cases when they are reported. Doctors, nurses, the laboratories, and eventually others report the respective communicable diseases when they occur.

2.1 Definition

An infectious disease is a disease that spreads from one person or animal to another. Some people also refer to infectious diseases as “communicable” or “transmissible” diseases.

Once a pathogen has entered a person’s body, it will begin replicating. The individual may then begin to experience symptoms. Some symptoms are a direct result of the pathogen damaging the body’s cells; others are due to the body’s immune response to the infection. The host reacts to infections with an innate response, often involving inflammation, followed by an adaptive response.

Communicable diseases are often mild, and symptoms pass after a few days. However, some can be serious and potentially life-threatening.

The skin is our body’s defense, protecting it from harmful environmental forces. And yet, viruses, bacteria, or fungi may sometimes penetrate the skin barriers and

cause infectious diseases. These infections are called contagious skin diseases. They present with a wide range of symptoms. Some have similarities, such as rashes, while others present in a quite specific manner for the causative infectious agent.

Infections of the scalp include bacterial infection of hair follicles (folliculitis), infestation with head lice (pediculosis capitis), and fungal infection or scalp ringworm (tinea capitis). Itching and excessive flaking of the scalp are seen with dandruff and seborrheic dermatitis, of which both are associated with perturbations of the normal scalp microbiome [1]. But also systemic infectious disease may manifest with hair shedding, such as syphilis [2], and systemic febrile infections, like dengue hemorrhagic fever [3] or COVID-19 [4].

2.2 Classification

Infections are caused by a wide range of pathogens, including bacteria, fungi, viruses, and parasites. The invasion of a human or animal body with an arthropod is usually termed infestation.

The symptoms of an infection depend on the type of disease. Some signs of infection affect the whole body generally, while others are specific to individual body parts, such as the skin and hair. An infection is not synonymous with an infectious disease, as some infections do not cause illness in a host. In certain cases, infectious diseases may be asymptomatic for much or even all of their course in a given host. The condition may only be defined as a disease, which by definition means an illness, in hosts who secondarily become ill after contact with an asymptomatic carrier. Persistent infections occur when the body is unable to clear the organism after the initial infection. Persistent infections are characterized by the continual presence of the infectious agent, often as latent infection with occasional recurrent relapses of active infection.

Since the introduction of specific therapeutic agents, the focus of attention has been on the determination of the specific infectious agents so that the proper choice of therapy can be made. This has rendered pointless, and in fact out-of-date, descriptions of some of the dermatologic entities whose status depended on ill-defined morphologic criteria rather than on etiologic considerations. From the pragmatic perspective, the approach has been to classify the infectious diseases by their microbial causation.

The normal skin of healthy individuals is highly resistant to invasion by bacteria to which it is constantly exposed. Bacteria are unable to penetrate the keratinized layers of normal skin and, when applied to the surface, rapidly decrease in number. The susceptibility to infection with pathogenic bacteria is determined by specific and unspecific factors, such as immunocompetence, nutritional state, and integrity of the cutaneous barrier. Meanwhile, certain bacterial species colonize the skin surface successfully. The microorganisms that characteristically survive and multiply in various ecologic niches of the skin constitute the normal cutaneous flora or microbiome. An appreciation of the composition of this flora and the attributes of its

major elements has gained the focus of attention for understanding and treating dermatologic diseases including the scalp.

Bacterial skin infections have been classified into (1) primary infections, (2) secondary infections, and (3) the cutaneous manifestations of systemic bacterial disease.

Primary bacterial infections are produced by the invasion of seemingly normal skin by a single species of pathogenic bacteria. Treatment aimed at the pathogen almost invariably results in the cure of the lesion, since there is usually no doubt as to the primary etiologic role of the specific agent in the pathogenesis of the lesion. Examples are follicular impetigo, folliculitis decalvans, sycosis barbae, furuncles, and carbuncles.

Secondary bacterial infections develop in areas of already damaged skin, and the appearance of these lesions is largely determined by the underlying skin condition. Although the bacteria did not produce the underlying skin condition, their proliferation and invasion of the skin may aggravate and prolong the disease. Secondary infections often show a mixture of organisms on culture, and not infrequently, it is difficult to determine which plays the major role. Examples are bacterial infections complicating traumatic lesions, eczematous dermatitis or dermatophytosis, dissecting cellulitis of the scalp, and pilonidal sinus.

The dermatophytes are a group of taxonomically related fungi capable of colonizing keratinized tissues such as the stratum corneum of the epidermis, the hair, and the nails, since dermatophytes can use keratin as a source of nutrients. The classification of the dermatophytes emphasizes ecologic features of the pathogens, as well as the pattern of infection of the hair.

Those species found only in soil are called *geophilic*, those in association with domestic or wild animals *zoophilic*, and those found only in association with human beings *anthropophilic*.

A number of different species of fungi are involved in dermatophytosis. Dermatophytes of the genera *Microsporum* and *Trichophyton* are the most causative agents.

Tinea capitis is a dermatophytosis of the scalp and associated hair. When suspected hairs are placed on microscope slides with clearing solution to be examined by low-power microscopy, one out of three patterns of infection may be seen: *ectothrix* with fungal arthrospores outside the hair shaft, *endothrix* with arthrospores contained within the hair shaft, or *favic* with a linear arrangement of hyphal fragments in chains along the longitudinal axis of the hair shaft. Finally, the different organisms causing tinea capitis may present with different clinical patterns: (1) noninflammatory, human, or epidemic type, (2) inflammatory type, (3) “black dot” tinea capitis, and (4) tinea favosa, with their respective causative agents, (1) *M. audouinii* and *M. ferrugineum*, (2) *M. canis* and *M. gypseum*, (3) *T. tonsurans* and *T. violaceum*, and (4) *T. schoenleinii*, respectively. Kerion celsi is the most pronounced type of inflammatory tinea capitis which is a result of the host’s response on the fungal infection of the hair follicles. It can be accompanied by secondary bacterial infection. It usually appears as raised, spongy lesions and typically occurs

in children. This honeycomb is a painful inflammatory reaction with deep suppurative lesions on the scalp and follicles discharging pus.

Tinea barbae is a fungal infection limited to coarse hair-bearing areas such as the beard and moustache.

Piedra is a condition in which the fungus causes formation of nodules on the hair shaft. Depending on the fungal agent, the clinical presentation, and climatic preferences, a classification is made into white and black piedra.

Yeast infections of the scalp can be classified into candidiasis and pityrosporum infections.

Candida albicans is the most important fungal opportunistic pathogen that can cause infection when the host becomes debilitated or immunocompromised. Systemic candidiasis usually follows dissemination of *Candida* spp. from the gastrointestinal tract or via the bloodstream. Skin lesions may occur particularly in two situations: (1) in neutropenic patients with severe disseminated disease and widespread muscle pain and (2) in intravenous drug abusers, where candidiasis may present with a follicular pustular rash in the beard area and scalp. *Candida* infection of the scalp in immunocompetent patients is rare [5].

Meanwhile, *Pityrosporum* spp. are part of the normal flora, particularly in sebum-rich areas of the skin, including the scalp. Under appropriate conditions, it may convert from the saprophytic yeast to the predominantly parasitic mycelial morphology associated with clinical disease, such as tinea versicolor. Dandruff, seborrheic dermatitis, the head-and-neck type atopic dermatitis, and folliculitis are typical scalp conditions related to *Pityrosporum*.

Molds growing in mycological cultures (Petri dishes) taken from the scalp and hair usually represent external contaminations. Molds are a large and taxonomically diverse number of fungal species in which the growth of hyphae results in discoloration and a fuzzy appearance. They reproduce by producing large numbers of small spores. Some molds produce small, hydrophobic spores that are adapted for wind dispersal and may remain airborne for long periods. Although molds can grow on dead organic matter everywhere in nature, their presence is visible to the unaided eye only when they form large colonies. A mold colony does not consist of discrete organisms but is an interconnected network of hyphae called a mycelium. In artificial environments, humidity and temperature are often stable enough to foster the growth of mold colonies, commonly seen as a downy or furry coating growing on surfaces.

Viruses represent a diverse group of infectious agents that share a distinctive composition and a unique mode of replication. They are not cellular organisms. A virus is a submicroscopic infectious agent that replicates only inside the living cells of an organism. Viruses infect all life forms, from animals and plants to microorganisms, including bacteria. The most important element of a virus is its genetic information, which it transfers to a susceptible host. When infected, a host cell is forced to rapidly produce thousands of copies of the original virus. Viruses are by far the most abundant biological entities on Earth, and they outnumber all the others put together. They display a wide diversity of shapes and sizes. Accordingly, the animal

viruses have been divided into several large families according to their morphologies, the structure of the virion, and the type of viral nucleic acid (DNA or RNA). Finally, the range of structural and biochemical effects that viruses have on the host cell is also extensive. These are called cytopathic effects. Most virus infections eventually result in the death of the host cell. The causes of death include cell lysis, alterations to the cell's surface membrane, and apoptosis. Some viruses cause no apparent changes to the infected cell. Cells in which the virus is latent and inactive show few signs of infection and often function normally. This causes persistent infections, and the virus is often dormant for many months or years. This is often the case with herpesviruses.

By definition, parasitism is a symbiotic relationship between species, where one organism, the parasite, lives on (ectoparasites) or inside (endoparasites) another organism, the host, causing it some harm, and is adapted structurally to this way of life. Parasites reduce host fitness by general or specialized pathology while increasing their own fitness by exploiting hosts for resources necessary for their survival, in particular by feeding on them and by using intermediate (secondary) hosts to assist in their transmission from one definitive (primary) host to another. Parasitism has an extremely wide taxonomic range, including animals, plants, fungi, protozoans, bacteria, and viruses. Parasites use a variety of methods to infect animal hosts, including physical contact, the fecal-oral route, free-living infectious stages, and vectors, suiting their differing hosts, life cycles, and ecological contexts.

Infestation is the state of being invaded by parasites. In general, the term refers to parasitic diseases caused by animals such as arthropods, such as mites, ticks, and lice, and worms, but excluding conditions caused by protozoa, fungi, bacteria, and viruses, which are called infections. Medically, the term infestation is also reserved for external ectoparasitic infestations, while the term infection refers to internal endoparasitic conditions.

Bacteriology is the study of bacteria and their relation to medicine, virology that of the viruses, mycology that of the fungi, parasitology that of the parasites, and entomology that of the arthropods. Tropical medicine is the study of the world's major diseases endemic to the tropics and related conditions. Because of the similarity of thinking and working with microorganisms other than bacteria, fungi, viruses, and protozoa, there has been a tendency for the field of bacteriology to extend as microbiology. The dermatologist should be knowledgeable of the respective specialties as they relate to the skin, hair, and nails. Infectiology is the medical specialty dealing with the diagnosis and treatment of complex infections. An infectious disease specialist's practice consists of managing nosocomial (healthcare-acquired) infections or community-acquired infections and is historically associated with travel medicine and tropical medicine.

2.3 Pathophysiology

There is a general chain of events that leads to infections. The chain of events involves several steps, which include the infectious agent, reservoir, entering a susceptible host, exit, and transmission to new hosts. Each of the links must be present in a chronological order for an infection to develop and spread.

Infection begins when the pathogen successfully enters the body, grows, and multiplies. This is referred to as colonization. The variables involved in the outcome of a host becoming inoculated by a pathogen and the ultimate outcome include:

- The route of entry of the pathogen and the access to host regions that it gains
- The intrinsic virulence of the particular organism
- The quantity or load of the initial inoculant
- The immune status of the host being colonized

Hosts can fight infections using their immune system. Mammalian hosts react to infections with an innate response, often involving inflammation, followed by an adaptive response. Disease arises if the host's protective immune mechanisms are compromised and the organism inflicts damage on the host.

The disease-producing capacity of bacteria is determined by (1) the invasion potential of the microorganism and (2) its toxigenic properties. Though it is useful to distinguish between these two major pathogenic mechanisms, most bacterial infections result from a combination of the invasive and toxigenic properties of the organism.

For many disease-producing bacteria, including the suppurative lesions of *S. aureus*, a clear understanding of the basis for pathogenicity has been lacking. And yet, it is recognized that exotoxins constitute essential components of the virulence mechanisms of *S. aureus*. Nearly all strains secrete lethal factors that convert host tissues into nutrients required for bacterial growth. Panton-Valentine leukocidin (PVL) is one of many toxins associated with *S. aureus* infection. It was initially discovered by Van deVelde in 1894 due to its ability to lyse leukocytes. It was named after Sir Philip Noel Panton and Francis Valentine when they associated it with soft tissue infections in 1932 [6]. The presence of PVL is associated with increased virulence of certain strains of *S. aureus*. It is present in the majority of community-associated methicillin-resistant *S. aureus* isolates studied [7] and is the cause of necrotic lesions of the skin.

What's more, the role of bacterial biofilms as pathogenetic factors in chronic recurrent bacterial disease of the skin and hair has only recently gained attention. A biofilm comprises any syntrophic consortium of microorganisms in which cells stick to each other and to a surface. These adherent cells become embedded within a slimy extracellular matrix that is composed of extracellular polymeric substances, such as polysaccharides, proteins, lipids, and DNA. Biofilms may form on living or

nonliving surfaces in response to a number of different factors, which may include cellular recognition of specific or nonspecific attachment sites on a surface and nutritional cues. A cell that switches to the biofilm mode of growth undergoes a phenotypic shift in behavior in which large suites of genes are differentially regulated. Biofilms are not just bacterial slime layers but biological systems; the bacteria organize themselves into a coordinated functional community. The biofilm bacteria can share nutrients and are sheltered from harmful factors in the environment, such as desiccation, antibiotics, and a host body's immune system.

It is suggested that around two-thirds of bacterial infections in humans involve biofilms [8, 9]. Infections associated with biofilm growth usually are challenging to eradicate due to the fact that mature biofilms display antimicrobial tolerance and immune response evasions [10, 11]. Biofilms often form on the inert surfaces of implanted devices such as catheters, prosthetic cardiac valves, and intrauterine devices. Some of the most difficult infections to treat are those associated with the use of medical devices [8]. Biofilms can form on the teeth as dental plaque, where they may cause tooth decay and gum disease. Biofilm formation in the skin has been identified in soft tissue fillers [12] and more recently in folliculitis decalvans [13].

In the case of clinical fungal infection of the skin and hair, a suitable environment on the host skin is of critical importance: trauma, hydration, and occlusion all interfere with the barrier function of the stratum corneum. Once the host skin is inoculated under the suitable conditions, there follow several stages through which the dermatophyte infection progresses. During an initial incubation period, the dermatophyte grows in the stratum corneum, sometimes with minimal clinical symptoms of infection. A *carrier status* has been postulated when the presence of a dermatophyte is detected on seemingly normal skin. Once the infection is established in the stratum corneum of the skin, (1) the rate of growth of the organism, in its relation to (2) the epidermal turnover rate, is determinant, since the fungal growth rate must equal or exceed the epidermal turnover rate or the organism will be shed quickly. Proteolytic enzymes, including keratinases, and toxins produced by the organism and the host immunologic response finally account for the clinical presentation of disease.

Resistance to fungal infection may involve non-immunologic as well as immunologic mechanisms.

Noteworthy is the increase in saturated fatty acids on the skin that occurs after puberty, explaining for the higher incidence of tinea capitis during childhood.

The major immunologic defense mechanism is the type IV delayed hypersensitivity response. Infections by dermatophytes naturally stimulate the immune system as in those by other microorganisms. However, differing from other infections, the infecting organisms cannot become a direct target of antibody response or phagocytosis because they reside only in the barrier membrane of the body surface, specifically, in the stratum corneum. In dermatophytosis, a unique behavior of the epidermis as noted in contact dermatitis plays an important role in the defense against infection. Dermatitic changes induced by fungal products, particularly those due to contact sensitivity to a fungal antigen, trichophytin, enhance epidermopoiesis, which leads to increased turnover of the epidermis with their resultant elimination from the

skin surface. Furthermore, the dermatophytes in the stratum corneum provoke transepidermal leukocyte chemotaxis by generating chemotactic C5a anaphylatoxin in exudating serum via alternative complement pathway activation in addition to a release of low-molecular-weight chemotactic factors. Such neutrophilic migration with the formation of subcorneal pustules also enhances epidermal proliferation [14].

In the past, skin testing has been used both to determine the causative organisms of infection or to discover a respective immunologic deficiency state. Characteristic skin lesions usually develop after intradermal injection of the microbial antigen. However, false-positive and false-negative reactions, which antigens to use in an antigen battery, their lack of standardization, and the method of administration have been important issues dealing with the skin tests [15].

Patients with chronic dermatophytosis appear to have a relatively specific defect in delayed hypersensitivity to trichophytin, and their cell-mediated responses to other antigens may also be somewhat decreased. However, the subjects do not appear to suffer excessive morbidity from infectious diseases other than dermatophytosis [16].

Chronic mucocutaneous candidiasis (CMC) refers to a heterogeneous group of disorders characterized by recurrent or persistent superficial infections of the skin, mucous membranes, and nails with *Candida* organisms, usually *Candida albicans*, with little propensity for systemic dissemination. CMC does not represent a specific condition but rather a phenotypic presentation with a spectrum of immunologic, endocrinologic, and autoimmune disorders. The unifying feature of these heterogeneous disorders is impaired cell-mediated immunity against *Candida* species.

CMC is inherited either as an autosomal dominant or recessive trait, with nine types depending on the gene and chromosomal locus, including the interleukin 17 (IL-17) family and receptor genes.

The autoimmune polyendocrinopathy syndrome with chronic mucocutaneous candidiasis (APECED) is an autosomal recessive disease caused by mutations in the autoimmune regulator (AIRE) gene and characterized by the clinical triad of CMC, hypoparathyroidism, and adrenal insufficiency. Additional features may be insulin-dependent diabetes mellitus, chronic atrophic gastritis with pernicious anemia, hypogonadism, alopecia areata, and vitiligo. Onset is in childhood, candidiasis is usually the first symptom, and manifestations continue to appear until the fifth decade, including alopecia. AIRE is the first gene identified underlying autoimmune disease. This gene plays a critical role in the body's ability to distinguish between its own proteins and cells and those of bacteria and viruses. The marked susceptibility to mucocutaneous candidiasis without systemic candidiasis in this condition is less understood. Böni and Trüeb reported unsuccessful sensitization to diphenylcyclopropenone (DPCP) and treatment of alopecia universalis in a patient with APECED [17].

The deep fungal infections involve two distinct groups of conditions: (1) the subcutaneous and (2) the systemic mycoses. Neither are common, are largely confined to the tropics and subtropics, or are encountered as opportunistic infections in the immunocompromised, including those with AIDS or with neutropenia

associated with malignancy and solid organ transplants. Subcutaneous fungal infections usually present with signs of skin involvement, while systemic mycoses only occasionally have skin lesions, either as portal of entry or following dissemination from a deep focus of infection.

Mycetoma is a chronic localized infection caused by different species of fungi or actinomycetes, which is characterized by the formation of aggregates of the causative organisms (grains) within abscesses. These communicate via sinuses onto the skin surface or may involve adjacent bone causing a form of osteomyelitis. The organisms are usually soil or plant saprophytes that are only incidental human pathogens implanted subcutaneously, usually after a penetrating injury. Dermatophytic mycetoma of the scalp is a rarity, especially when not associated with tinea capitis [18]. Actinomyctoma of the scalp has been reported after a car accident [19]. Because of this topography and potential spread to the brain, this condition may be a particular diagnostic and therapeutic challenge.

Dermatophytic pseudomycetoma represents a chronic infection characterized by a tumor-like growth containing dermatophytes arranged as clustered aggregates (grains) within the dermis. In contrast to mycetomas, they lack sinus tracts and are more common in the scalp [20]. Both are similar to eumycetoma clinically and histopathology, being distinguished through the isolation of the fungus, which in the case of pseudomycetoma can be *Microsporum* spp. or *Trichophyton* spp. [21].

Skin lesions are a prominent feature of a number of viral diseases. In some instances, the cutaneous lesions may suggest a specific viral disease, of which diagnosis can be quickly established. At other times, the differential diagnosis is broader, particularly in the case of unspecific rashes and diffuse effluvium. In general, virus infections may affect the skin and hair by three different routes: (1) direct inoculation, (2) systemic infection, or (3) local spread from an internal focus. The skin lesions may be produced by the direct effect of virus replication on infected cells, the host response to the virus, or the interaction of replication and host response. Typically, infected cells develop gross cytopathic changes and eventually die. Viral cytopathic effects usually account for the appearance of early lesions, while the host immune response presumably contributes to the evolution of those lesions that subsequently develop an inflammatory response. The severity of illness produced by a particular virus varies significantly from individual to individual, with host factors believed to be determinant for most of this variation. Specific cell-mediated immunity is elicited during viral infections and influences the course of many viral infections. Antibody responses to viral infection represent the major host defense against reinfection by the same virus. Inflammatory cells may produce some of their antiviral effects through the production of interferons (IFNs). IFN, which can be induced by foreign RNA or DNA, is secreted into the extracellular fluid, where it confers resistance to virus infections to those cells that come in contact with the IFN. Finally, genetic factors may play a role in determining the outcome of viral infections. For instance, loss-of-function variants of X-chromosomal TLR7 have been identified in association with impaired IFN responses in young men with severe COVID-19 [22].

2.4 Transmission

For infecting organisms to survive and repeat the infection cycle in other hosts, they or their progeny must leave an existing reservoir and cause infection elsewhere. Infection transmission can take place via many potential routes:

- Droplet contact—also known as the respiratory route or airborne disease. If an infected person coughs or sneezes on another person, the microorganisms, suspended in warm, moist droplets, may enter the body through the nose, mouth, or eye surfaces.
- Oral transmission—diseases that are transmitted primarily by oral means may be caught through direct oral contact such as kissing or by indirect contact such as by sharing a drinking glass.
- Transmission by direct contact.
- Sexual transmission—with the resulting disease being called sexually transmitted disease.
- Vertical transmission—directly from the mother to an embryo, fetus, or baby during pregnancy or childbirth. It can occur as a result of a preexisting infection or one acquired during pregnancy.
- Fecal-oral transmission—foodstuffs or water becomes contaminated by people not washing their hands before preparing food or untreated sewage being released into a drinking water supply, and the people who eat and drink them become infected.
- Vehicle transmission—transmission by an inanimate reservoir such as food, water, or soil.
- Iatrogenic transmission—due to medical procedures such as injection or transplantation of infected material.
- Vector-borne transmission—transmitted by a vector, which is an organism that does not cause disease itself but that transmits infection by conveying pathogens from one host to another.

Once transmission occurs, the pathogen must establish an infection to continue. The more competent the host immune system, the less chance there is for the pathogen to survive. It may require multiple transmission events to find a suitably vulnerable host. During this time, the invader is dependent upon the survival of its current host. Multiple infections can also result in gene swapping among pathogens, increasing the pathogen's ability to cause damage to a host (virulence). A potential for virulence exists whenever a pathogen invades a new environment, host, or tissue. The new host is likely to be poorly adapted to the intruder.

2.5 Epidemiology

As opposed to the noncommunicable disease, i.e. cardiovascular diseases, cancers, respiratory diseases, and diabetes, infectious diseases, also known as communicable diseases or transmissible diseases, are an illness resulting from the invasion of an organism's body tissues by disease-causing agents, their multiplication, and the reaction of host tissues to the infectious agents and the toxins they produce.

Infections can be caused by a wide range of pathogens, including bacteria, viruses, fungi, parasites, and arthropods.

Epidemiology centers around the idea that disease and illness do not exist randomly or in a bubble. It is the study and analysis of who, why, and where disease occurs and what determines whether various populations have a disease. Epidemiologists conduct research to establish the factors that lead to public health issues, the appropriate responses, interventions, and solutions. By using research, from the field and in the lab, and statistical analysis, epidemiologists can track disease and predict its future outcomes. This type of epidemiology is at the forefront of today's world as epidemiologists work on the front lines to track and trace the spread of COVID-19.

Epidemiology is at the foundation of public health. Epidemiological research helps us understand not only who has a disease but why and how it was brought to this individual or region. Another task of epidemiology is monitoring or surveillance of time trends to show which diseases are increasing or decreasing in incidence and which are changing in their distribution. This information is needed to identify emerging problems and also to assess the effectiveness of measures to control old problems.

Epidemiologists may determine differences among groups within a population, such as whether certain age groups have a greater or lesser rate of infection, whether groups living in different environs are more likely to be infected, and by other factors, such as gender and ethnicity.

Researchers also may assess whether a disease outbreak is sporadic, or just an occasional occurrence; endemic, with a steady level of regular cases occurring in a region; epidemic, with a fast arising and unusually high number of cases in a region; or pandemic, which is a global epidemic. If the cause of the infectious disease is unknown, epidemiology can be used to assist with tracking down the sources of infection.

Like the clinical findings and pathology, the epidemiology of a disease is an integral part of its basic description. Ultimately, epidemiology has developed into a vibrant scientific discipline that brings together the social and biological sciences, incorporating everything from statistics to the philosophy of science in its aim to study and track the distribution and determinants of health events.

With their potential for unpredictable and explosive impacts, infectious diseases have been major actors in human history [23]. In 2010, about ten million people died of infectious diseases.

Nevertheless, microorganisms ordinarily live in harmony with their hosts via mutual or commensal interactions. Diseases emerge when existing microorganisms

become pathogenic or when new pathogenic microorganisms enter a new host. For instance, the introduction of smallpox, measles, and typhus to Central and South America by European explorers during the fifteenth and sixteenth centuries caused pandemics among the native inhabitants. Between 1518 and 1568, disease pandemics purportedly caused the population of Mexico to fall from 20 million to 3 million [24].

Human activity is involved with many emerging infectious diseases, such as environmental change enabling a microorganism to occupy new niches. When that happens, a pathogen that had been confined to a remote habitat has a wider distribution and possibly a new host organism. Pathogens jumping from nonhuman to human hosts are known as zoonoses [25].

Several human activities have led to the emergence of zoonotic human pathogens [26, 27] and spread of vector-borne diseases:

- Encroachment on wildlife habitats, hunting, and bushmeat
- Changes in agriculture
- Deforestation, biodiversity loss, and environmental degradation
- Climate change
- Uncontrolled urbanization with crowding
- Greater global interconnectivity driven by international travel and commerce

These understandings underline the way in which infectious diseases are a part of an ecological web that itself is influenced by shifting economic, social, and environmental factors. Ultimately, René Dubos (French-American microbiologist, experimental pathologist, environmentalist, 1901–1982) remarked in 1958 that "microbial disease is one of the inevitable consequences of life in a world where nothing is stable" [28].

2.6 Prevention

Disease prevention is a procedure through which individuals, particularly those with risk factors for a disease, are treated in order to prevent a disease from occurring.

Disease prevention, understood as specific, population-based, and individual-based interventions, aims at minimizing the burden of diseases and associated risk factors, whereby there are three levels of prevention:

- Primary prevention—intervening before health effects occur
- Secondary prevention—screening to identify diseases in the earliest
- Tertiary prevention—managing disease post diagnosis to slow or stop

Primary prevention refers to actions aimed at avoiding the manifestation of a disease. This may include actions to improve health through changing the impact of social and economic determinants on health; the provision of information on behavioral and medical health risks, alongside consultation and measures to decrease them at the personal and community level; nutritional and food supplementation; hygiene education; and clinical preventive services such as immunization and vaccination.

Secondary prevention deals with early detection when this improves the chances for positive health outcomes. This comprises activities such as evidence-based screening programs for early detection of diseases and preventive drug therapies of proven effectiveness when administered at an early stage of the disease.

One of the ways to prevent or slow down the transmission of infectious diseases is to recognize the different characteristics of various diseases. Some critical disease characteristics that should be evaluated include virulence, distance traveled by victims, and level of contagiousness.

Infectious diseases are caused by microorganism harbored in other people, animals, or the environment. Avoiding contact is a means to prevent many infections and diseases. While specific diseases are passed in specific ways, there are basic steps to take to stay healthy and lower the risk of catching and spreading any infectious disease:

Vaccinate and Use Medicines Properly

- Keep immunizations up to date. Follow recommended immunizations for children, adults, and pets.
- Use antibiotics exactly as prescribed. Take them for the full course prescribed by your doctor, but not for colds or other nonbacterial illnesses. Never self-medicate with antibiotics or share them with family or friends.
- Report to your doctor any quickly worsening infection or any infection that does not get better after you take a prescribed antibiotic.
- If you travel internationally, get all recommended immunizations, and use protective medications for travel, especially to areas with malaria.

Keep Clean

- Wash your hands often, especially during cold and flu season.
- Be aware of what you eat, and prepare foods carefully.
- Protect yourself from disease carriers.
- Be cautious around all wild and domestic animals that are not familiar to you.
- After any animal bite, clean the skin with soap and water, and seek medical care immediately.
- Avoid areas where there are ticks.
- Protect yourself from mosquitoes.
- Stay alert to disease threats when you travel or visit undeveloped areas.

- Don't drink untreated water while hiking or camping. If you become ill when you return home, tell your doctor where you've been.

Don't Spread Disease

- If you are sick with a cold or flu, stay at home, and don't spread germs.
- Practice safer sex.
- Do not use intravenous drugs or share syringes.

Key risk factors for communicable diseases identified in the academic literature can be broadly grouped into categories such as water, sanitation, and hygiene (WASH), health and public health system, environment, infrastructure, living conditions, nutrition, and overcrowding [29]. Within those broader categories, individual risk factors are defined more specifically, although the categories themselves serve as general risk factors as well, particularly for communicable disease outbreaks in humanitarian emergencies and disasters.

Knowing key risk factors and their thresholds and weight in different types of infections can help guide risk reduction efforts and emergency response.

Besides the illustration of the pertinent pathogens, the clinical presentations of disease, and the treatment, the following book chapters attempt also to address the epidemiology of risk factors for the infectious diseases with manifestation on the hair and scalp for the respective measures of prevention.

References

1. Polak-Witka K, Rudnicka L, Blume-Peytavi U, Vogt A. The role of the microbiome in scalp hair follicle biology and disease. Exp Dermatol. 2020;29(3):286–94.
2. Hernández-Bel P, Unamuno B, Sánchez-Carazo JL, Febrer I, Alegre V. Syphilitic alopecia: a report of 5 cases and a review of the literature. Actas Dermosifiliogr. 2013;104(6):512–7.
3. Wei KC, Huang MS, Chang TH. Dengue virus infects primary human hair follicle dermal papilla cells. Front Cell Infect Microbiol. 2018;21(8):268.
4. Trüeb RM, Dutra Rezende H, Gavazzoni Dias MFR. What can the hair tell us about COVID-19? Exp Dermatol. 2021;30(2):288–90.
5. Fortuna MC, Garelli V, Pranteda G, Carlesimo M, D'Arino A, Rossi A. Scalp infection by Candida Albicans in an immunocompetent patient: a description of a rare case. J Chemother. 2018;30(5):316–7.
6. Boyle-Vavra S, Daum RS. Community-acquired methicillin-resistant Staphylococcus aureus: the role of Panton–Valentine leukocidin. Lab Invest. 2007;87(1):3–9.
7. Szmiegielski S, Prevost G, Monteil H, et al. Leukocidal toxins of staphylococci. Zentralbl Bakteriol. 1999;289(2):185–201.
8. Del Pozo JL, Rouse MS, Patel R. Bioelectric effect and bacterial biofilms. A systematic review. Int J Artif Organs. 2008;31(9):786–95.
9. Lazar V. Quorum sensing in biofilms—how to destroy the bacterial citadels or their cohesion/power? Anaerobe. 2011;17(6):280–5.
10. Bryers JD. Medical biofilms. Biotechnol Bioeng. 2008;100(1):1–18.

11. Rybtke M, Hultqvist LD, Givskov M, Tolker-Nielsen. Pseudomonas aeruginosa biofilm infections: community structure, antimicrobial tolerance and immune response. J Mol Biol. 2015;427(23):3628–45.
12. Dayan SH, Arkins JP, Brindise R. Soft tissue fillers and biofilm. Facial Plast Surg. 2011;27:23–8.
13. Matard B, Meylheuc T, Briandet R, Casin I, Assouly P, Cavelier-balloy B, Reygagne P. First evidence of bacterial biofilms in the anaerobe part of scalp hair follicles: a pilot comparative study in folliculitis decalvans. J Eur Acad Dermatol Venereol. 2013;27(7):853–60.
14. Tagami H, Kudoh K, Takematsu H. Inflammation and immunity in dermatophytosis. Dermatologica. 1989;179(Suppl 1):1–8.
15. Ponte CD. Delayed cutaneous hypersensitivity testing. South Med J. 1982;75(9):1076–9.
16. Sorensen GW, Jones HE. Immediate and delayed hypersensitivity in chronic dermatophytosis. Arch Dermatol. 1976;112(1):40–2.
17. Böni R, Trüeb RM, Wüthrich B. Alopecia areata in a patient with candidiasis-endocrinopathy syndrome: unsuccessful treatment trial with diphenylcyclopropenone. Dermatology. 1995;191(1):68–71.
18. Diongue K, Boye A, Bréchard L, Diallo MA, Dione H, Ndoye NW, Diallo M, Ranque S, Ndiaye D. Dermatophytic mycetoma of the scalp due to an atypical strain of Microsporum audouinii identified by MALDI-TOF MS and ITS sequencing. J Mycol Med. 2019;29(2):185–8.
19. Welsh O, Morales-Toquero A, Vera-Cabrera L, Vazquez-Martinez O, Gómez-Flores M, Ocampo-Candiani J. Actinomycetoma of the scalp after a car accident. Int J Dermatol. 2011;50(7):854–7.
20. Castro-Echeverry E, Fiala K, Fernandez MP. Dermatophytic pseudomycetoma of the scalp. Am J Dermatopathol. 2017;39(2):e23–5.
21. Ruiz LRB, Zaitz C, Lellis RF, Veasey JV. Pseudomycetoma of the scalp caused by Microsporum canis. An Bras Dermatol. 2020;95(3):372–5.
22. van der Made CI, Simons A, Schuurs-Hoeijmakers J, van den Heuvel G, et al. Presence of genetic variants among young men with severe COVID-19. JAMA. 2020;324(7):663–73.
23. Fauci AS, Morens DM. The perpetual challenge of infectious diseases. N Engl J Med. 2012;366(5):454–61.
24. Dobson AP, Carter ER. Infectious diseases and human population history. BioScience. 1996;46(2):115–26.
25. Krauss H, Weber A, Appel M. Zoonoses: infectious diseases transmissible from animals to humans. 3rd ed. Washington, DC: ASM Press; 2003. ISBN 978-1-55581-236-2.
26. Potter P. Summer buzz. Emerg Infect Dis. 2013;19(3):1184.
27. Daszak P, Cunningham AA, Hyatt AD. Emerging infectious diseases of wildlife—threats to biodiversity and human health. Science. 2000;287(5452):443–9.
28. Dubos R. Infection into disease. Perspect Biol Med. 1958;1(4):425–35.
29. Hammer CC, Brainard J, Hunter PR. Risk factors for communicable diseases in humanitarian emergencies and disasters: results from a three-stage expert elicitation. Global Biosecur. 2019;1(1). https://doi.org/10.31646/gbio.5.