

Detection and prevention of communicable diseases basic concepts.

Joseph Kate*

The Department of Non-communicable Disease Epidemiology, London School of Hygiene and Tropical Medicine, London, UK

Introduction

Understanding the elements affecting transmission in great detail is essential for controlling and preventing infectious diseases. In addition to outlining many of the agent, host, and environmental factors of these illnesses that are particularly important to public health practitioners, this article highlights the basic concepts of infectious disease transmission. Also covered are the fundamental concepts of infectious illness diagnosis, management, and prevention.

The epidemiological triad, a well-known model of infectious disease aetiology, postulates that an infectious illness develops as a result of a confluence of agent (pathogen), host, and environmental variables [1]. Living parasites (*helminths* or *protozoa*), fungus, bacteria, or nonliving viruses or prions can all be infectious agents. The likelihood of a host being exposed to one of these agents is determined by environmental conditions, and the course of the exposure is determined by the interactions between the agent and the host. Infection, illness, healing, or death is only a few of the phases in the cascade of interactions between an agent and a host.

Colonisation is the attachment and early proliferation of a disease agent at an entrance point, such as the skin or the mucous membranes of the respiratory, digestive, or urogenital tract. For instance, methicillin-resistant *Staphylococcus aureus* colonisation of the nasal mucosa does not in and of itself result in illness. A pathogen must infect (invade and develop inside) host tissues for illness to manifest. Although an infection will always disturb the host in some manner, illness is not necessarily the end outcome. Disease denotes a degree of host disruption and damage that produces both subjective symptoms and outward indications of sickness [2].

Factors that influence infectious disease

When a potential host is exposed to an infectious agent, the result of that exposure depends on the dynamic interaction between the intrinsic host determinants of susceptibility to infection and illness and the agent determinants of infectivity, pathogenicity, and virulence. Physical and social behavioural environmental variables are extrinsic determinants of host susceptibility to exposure.

The initial host defences against infection are physical and chemical surface barriers like the skin, the flushing effect of tears, and the trapping effect of mucus. For instance, severe

burns that eliminate the skin's natural barrier against microbial infiltration might result in catastrophic problems such as wound infection and subsequent sepsis [3]. Vaginal acid is microbicidal for many agents of STDs, and lysozyme, which is released in saliva, tears, milk, perspiration, and mucus, has bactericidal characteristics as well (STIs). Through the use of available resources and living space, microbiome-resident bacteria can also provide host protection by thwarting the establishment of harmful bacteria.

The host's defence against infectious pathogens must include both innate and adaptive immune responses. Each of these reactions is carried out by cells from a different hematopoietic stem cell lineage: the lymphoid lineage provides rise to adaptive immunity cells, while the myeloid lineage gives rise to innate immune cells (such as neutrophils, macrophages, and dendritic cells) (e.g., T cells, B cells) [4]. The innate immune response is a quick, general reaction to many different infections. The adaptive immune response, in contrast, is first produced over a period of 3–4 days, it detects certain pathogens, and it is divided into two main branches: (1) T cell-mediated immunity, and (2) B cell-mediated immunity.

There are fundamentally two different forms of immunisation: active and passive. The term "active immunisation" describes the process through which a host's own immune response produces immunological protection [5]. On the other hand, passive immunisation is given when immune effectors, most frequently antibodies, are transferred from a donor animal or person. For instance, a person who seeks medical attention after being bitten by a dog will get both active and passive postexposure immune prophylaxis, which includes rabies vaccination (to trigger the host immune response) and rabies immune globulin (to provide immediate passive protection against rabies). The transmission of immunity from a woman to her child during nursing is an illustration of natural passive immunisation.

Conclusion

Many prevalent infectious diseases, such as STIs (such as HIV, syphilis), and respiratory illnesses are carried by humans (e.g., influenza). For several neglected tropical diseases (NTDs), such as dracunculiasis, humans also act as a reservoir, but not usually the principal reservoir. Human reservoirs have a crucial role to play in public health since they may not exhibit symptoms of sickness and may operate as unnoticed disease carriers in their communities.

*Correspondence to: Joseph Kate, The Department of Non-communicable Disease Epidemiology, London School of Hygiene and Tropical Medicine, London, UK, E-mail: katej@lshhtm.ac.uk

Received: 01-Sep-2022, Manuscript No. AABID-22-80165; Editor assigned: 03-Sep-2022, PreQC No. AABID-22-80165 (PQ); Reviewed: 16-Sep-2022, QC No. AABID-22-80165; Revised: 19-Sep-2022, Manuscript No. ABID-22-80165 (R); Published: 26-Sep-2022, DOI:10.35841/aabid-6.5.122

References

1. Yu IT, Li Y, Wong TW, et al. Evidence of airborne transmission of the severe acute respiratory syndrome virus. *N Engl J Med.* 2004;350(17):1731-39.
2. Steinmann P, Keiser J, Bos R, et al. Schistosomiasis and water resources development: systematic review, meta-analysis, and estimates of people at risk. *Lancet Infect Dis.* 2006;6:411-25.
3. Snieszko SF. The effects of environmental stress on outbreaks of infectious diseases of fishes. *J Fish Biol.* 1974;6:197-208.
4. Screaton G, Mongkolsapaya J, Yacoub S, et al. New insights into the immunopathology and control of dengue virus infection. *Nat Rev Immunol.* 2015;15:745-59.
5. Rasmussen SA, Jamieson DJ, Honein MA, et al. Zika virus and birth defects-reviewing the evidence for causality. *N Engl J Med.* 2016;374(20):1981-87.