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CHAPTER 1

CIRCLES OF DISEASE TRANSMISSION

Magdalena Dunowska

Institute of Veterinary, Animal and Biomedical Sciences Massey University, University of New Zealand

1 Introduction

A variety of transmissible agents can cause disease in animals. These include viruses, bacteria, fungi and parasites. Within each of these main groups there are hundreds of potential pathogens with unique biological characteristics and life cycles. Our understanding of the causative relationships between infectious agents and disease has evolved over the years. While the canonical Koch's postulates presumed a simple direct relationship between a pathogen and disease, even Koch himself predicted the inadequacy of such an assumption in all situations (Fredericks & Relman, 1996). Today, we have a much better appreciation of the multifactorial nature of many diseases, and the complexity of events that determine not only the outcome of infection for each individual, but also the ability of pathogens to spread within populations. Understanding these complex interactions is necessary for the implementation of effective infection control programs.

New technologies have facilitated advanced molecular research and a sharp increase in our knowledge of the microbial world over the past 20-30 years. At the same time, rapid technological development has also created new challenges. The ever-increasing density of people has led to intensification of agriculture and change of land use through activities such as irrigation, deforestation or urbanisation. These changes have been accompanied by increased international travel, as well as increased international trade of animals and animal-derived products. The association between such anthropogenic activities and the emergence of diseases is fairly well recognised (Mackenzie & Jeggo, 2013). Human encroachment on wildlife habitats has created increased opportunities for cross-species transfer and potential for emergence of new zoonotic diseases, as we have seen with cases of Hendra virus in Australia, Nipah virus in Malaysia or severe acute respiratory syndrome (SARS) virus in China (Plowright et al., 2015). What once may have been confined to a few localised cases of disease now has a potential to spread throughout the world within days. As an example, about 8,000 cases and 900 deaths in 30 countries were traced back to a single SARS-infected person staying overnight at

the Metropole hotel in Hong Kong (Mackenzie & Jeggo, 2013). In 2013-2016 we also witnessed an epidemic of Ebola virus infections in West Africa at the unprecedented scale compared to several previous geographically restricted outbreaks (Shultz, Espinel, Espinola, & Rechkemmer, 2016).

Transmission of infectious agents from one host to another is a complex and multi-step process that requires a number of conditions to be fulfilled. Understanding how the pathogens are maintained within individual hosts and within populations enables us to intervene in this process. In a sense, the old saying 'know your enemy' is as applicable to people as it is to microbes. The better we understand the 'offenders' and the methods they adopt in order to survive and perpetuate themselves, the better we are able to effectively target the most relevant stages in their transmission circles.

This chapter provides a short overview of the steps necessary for transmission of microscopic pathogens from one host to another. It is not meant to be a comprehensive review of the topic. Instead, it introduces some basic concept, with an emphasis on common principles that can be exploited for the purpose of infection control.

2 Common steps in transmission of infectious agents

It is important to consider all potential pathogens that may be encountered in a given situation when designing an effective infection control programme (Morley, 2002; Traub-Dargatz, Dargatz, Morley, & Dunowska, 2004). Many of these pathogens have unique biological properties and life cycles. While this needs to be taken into account, it is often practical to focus on commonalities, as opposed to differences. Route of transmission is one of such characteristics that is often shared by several infectious agents and can therefore be exploited in the design of control measures that are effective against a number of different pathogens. For example, the use of disinfectant foot mats or footbaths helps to minimise the spread of pathogens that transmit via the faecal-oral route (Dunowska, Morley, Patterson, Hyatt, & Van Metre, 2006). Similarly, the use of disposable gloves and/or appropriate hand hygiene is an effective control measure against pathogens that can be transmitted through fomites (Neo, Sagha-Zadeh, Vielemeyer, & Franklin, 2016). In contrast, neither hand hygiene nor disinfectant foot mats/ footbaths would be particularly effective in controlling arboviral infections such as Bluetongue or African horse sickness. In order to prevent infections with these arboviruses, measures to control vector population (Culicoides midges) and to minimise contact between midges and susceptible animals would need to be implemented (Maclachlan & Mayo, 2013). These may include elimination of stagnant water (breeding environment for midges), application of insect repellents, use of screens in stables/barns, or keeping the animals indoors at times when the activity of midges is the greatest (dusk/dawn). Concurrently, minimising the density of susceptible animals, for example via vaccination, could also be implemented.

In general terms, most pathogens have to follow some common steps in order to spread and maintain themselves in populations. These include entry, replication and spread within the host (either locally or systemically), which may or may not be accompanied by disease, and exit to enable infection of the new host (Fig. 1.1). Each of these steps can be targeted by infection control strategies.



Table 1.1: Routes of entry

Res	piratory	Tract
ILCO,	pulatory	LINCI

neepinatory maet	
Description	Occurs through inhalation of infectious agents.
Protective Mechanisms	Entrapment of pathogens in a blanket of mucous produced by Goblet cells; removal of pathogens by coordinated movement of cilia on epithelial cells; removal of pathogens by specific (secretory antibodies, mainly IgA) or non-specific (phago- cytic cells such as neutrophils and macrophages) immunological defences (Antunes & Cohen, 2007; Baskerville, 1981; Fokkens & Scheeren, 2000).
Factors facili- tating entry	Concurrent infections e.g. viral infections that destroy ciliary epithelium often predispose subjects to secondary bacterial infections (shipping fever complex); presence of infectious aerosols; poor ventilation combined with crowded environment.
Examples ¹	foot-and-mouth disease, infectious bovine rhi- notracheitis, malignant catarrhal fever, anthrax, <i>Aspergillus</i> spp, Q fever, tuberculosis
Gastrointestinal T	ract
Description	Transmission via ingestion of pathogens. Typically occurs via contamination of feed with faecal mate- rial or via contamination of pastures/water supply.
Protective Mechanisms	Peristaltic activity of intestines, low pH of the stomach, presence of digestive enzymes, bile, pancreatic secretions, presence of mucous in the stomach and intestines, specific (mainly IgA) and non-specific (e.g. defensins) immuno- logical defences (Elson & Alexander, 2015)
Factors facilitat- ing spread	Heavy environmental contamination with pathogens that survive outside of their hosts for a prolonged period of time; poor clean- ing and disinfection; high density of animals.
Examples ¹	rotaviruses, coronaviruses, parvoviruses, morbilliviruses, bacteria e.g. <i>Salmonella</i> or <i>Campylobacter</i> species, <i>Brucella</i> species, tuberculosis, anthrax, many internal para- sites such as coccidia (<i>e.g. Eimeria or Crypto-</i> <i>sporidium species</i>) or <i>Giardia</i> species.
Skin	
Description	Pathogens enter the body through the skin barrier.