

3 The Infectious Disease Process

Epidemics never arise from a single cause, but from the interaction of several, at time numerous causes; their strength depending on various influences.--G. Francke and V. Görtler (1930)

3.1 Reasons to Study the Infectious Disease Process

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3.2 Herd Immunity • Herd Immunity • Stemming an Outbreak Through Herd Immunity • Epidemic Modeling

3.1 REASONS TO STUDY THE INFECTIOUS DISEASE PROCESS

Studying infectious disease epidemiology is important for two different reasons. First, infectious disease epidemiology provided the original model for the study of disease on a population basis. Many general epidemiologic principals emerged when studying infectious agents and have since been adopted by other fields of epidemiology. For example, the interaction of agent, host, and environmental factors in determining levels of disease in the population was first recognized in an infectious disease context, as was the importance of ordering multiple causal components into causal pathways. The insufficiency of agent-only theories of disease was initially an infectious disease concept. There are many conceptual similarities between infectious disease epidemiology and chronic disease epidemiology. In fact, many prominent epidemiologists believe division of epidemiology into subspecialites of infectious disease epidemiology and chronic disease epidemiology is arbitrary and detrimental to the discipline (Barrett-Connor, 1979; Stallones, 1980; Susser, 1985).

Second, infectious and parasitic diseases remain a leading cause of morbidity and mortality worldwide (World Health Organization, 1992; National Institute of Allergy and Infectious Disease, 1992). Many infectious diseases have recently emerged (e.g., HIV, hantavirus) or reemerged in virulent forms (e.g., tuberculosis, Yellow fever) to imperil the public's health, and the risk of bioterrorism has increased. Therefore, studying infectious disease epidemiology in its own right has taken on added relevance.

As an introduction to infectious disease epidemiology, we consider the following components of the infectious disease process:

- Agents
- Reservoirs
- Portals of entry and exit

{Text continues on p. 39 of the first edition with some markup.}

3.3 HERD IMMUNITY

The small-pox would be . . . sometimes arrested, by vaccination which protected a part of the population.—William Farr, 1885, p. 320

What is Herd Immunity?

An individual has an immune status that governs their susceptibility and, hence, the likelihood of infection once exposed. Similarly, a group has an immune status that governs the susceptibility of the group, and hence the incidence of disease in the “herd.” This property of **herd immunity** is defined as the proportion of resistant individuals in the population. As with individual immunity, we speak of innate herd immunity and acquired herd immunity.

Innate herd immunity is the proportion of individuals in the population that are resistant to infection for reason other than prior exposure or immunization. Several examples of innate herd immunity are known. For instance, people with sickle cell trait have relatively low parasite levels in the blood when infected with *P. falciparum* and are thus relatively protected from severe disease (Benenson, 1995). The relative resistance of falciparum malaria in populations with a high prevalence of sickle cell trait is due to a genetically determined metabolic polymorphism of red blood cells. While this polymorphism is fatal when seen in the homozygous form, and while heterozygosity renders some physiological disadvantages, its overall benefit to survival in endemic *P. falciparum* areas outweighs any such disadvantage. Thus, presence of the agent in the environment selects for the sickle-cell trait over successive generations. When the selection pressures of falciparum malaria are removed from the population, the frequency of this otherwise disadvantageous trait begins to decline.

Acquired herd immunity is the proportion of individuals in the population resistant to infection as the result of earlier exposure or immunization. The ultimate goal of a vaccination program is to reach an effective levels of coverage so that the disease is stopped in its tracks.

Stemming an Outbreak Through Herd Immunity

Herd immunity need not be absolute in order to halt the spread of infection through the “herd.” This is because when a high percentage of individuals in a population are resistant, transmission may dead-end before reaching remaining susceptibles. **Figure 3.4** illustrates how this might work. This figure assumes we are dealing with an infection that is transmitted by direct contact, and a single case is introduced into the population (cross-hatched). In scenario 1 there is no herd immunity and the agents spreads to all susceptibles (infection risk = 100%). In scenario 2 the herd immunity level is 65% (13 of 20) and the agent is blocked after infecting only 2 of susceptibles (infection risk = $2/7 = 29\%$). In scenario 3 the herd immunity level is 25% (5 of 20) and the agent spreads to all remaining susceptibles (infection risk = 100%). Thus, in scenario 2, a **herd immunity threshold** protects even those who might otherwise be susceptible.

The herd immunity stemming threshold depends on the infectivity of the agent and the population’s rate of effective contacts. To confer protection to susceptibles for a disease which is highly contagious (e.g., measles), herd immunity must high (e.g., about 95%) in order to prevent outbreaks. In populations where the “social distance” between individuals is less (e.g., India), herd immunity rates must be higher still (e.g., 99%) to stem infection (Berger, 1999). With a less contagious agent, such as mumps, a lower frequency

of immunity is necessary to prevent outbreaks.

Moderate levels of herd immunity can slow the spread of infection without completely halting its spread. This can have the negative consequence of delaying infection to older ages in those who eventually contract the disease. Since some infectious diseases are more severe or cause negative consequences when contracted in adulthood (e.g., mumps, chickenpox, hepatitis A, and rubella), a semi-effective level vaccination that affords incomplete herd immunity may decrease the number of infections, but the disease in those who do become ill may have more disastrous effects (Panagiotopoulos et al., 1999; Edmunds & Gay, 2000).

Epidemic Modeling

Epidemic modeling uses mathematical systems to predict the dynamics of infection, estimate or test parameters concerning incubation and infection rates, and educate decision-makers about vaccination programs. The **Reed-Frost model** is a simple, educational epidemic model in which:

$$C_{t+1} = S_t(1 - q^{C_t})$$

where C_{t+1} is the number of incident cases in time period $t+1$,

S_t is the number of susceptibles in time period t , and

$1 - q^{C_t}$ is the probability of having *at least* one effective contact during the interval.

An “effective contact” is defined as an exposure that would result in infection if one of the individuals was infectious and the other was susceptible; when a susceptible has an effective contact with a transmitter, the susceptible will develop into a case. Thus, the Reed-Frost equation models the course of an epidemic based on the number of susceptibles in the population and probability of having an effective contact (“mass action principle”).

Many important assumptions are made in applying the Reed-Frost model (e.g., random mixing of the population, the population is closed to outside contact, conditions of transmission remain constant over time, the infection is transmitted by direct contact only, infectious cases are immune in subsequent time periods). The Reed-Frost model can be modified to simulate more realistic assumptions (e.g., two or more open populations with different within- and between- population contacts and random mixing of contagious, immune, and susceptible individuals). The main value of the Reed-Frost model today is as a teaching tool, where it can be used to demonstrate mass action principles of contagion. More sophisticated mathematical models of transmission have since been developed which incorporate realistic contact structures and social patterns of behaviors (e.g., Longini et al., 1988).

{REFERENCES THAT MUST BE ADDED TO THE EXISTING LIST ON PAGE 52 - 53}

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