



One Health: Connecting Humans, Animals and the Environment Video Transcript

Modelling animal-human disease transmission

[Jakob Zinsstag]: For any given disease, the interplay of susceptible, infectious, and immune hosts determines the way how an infectious disease establishes, spreads, or dies out. This includes the transmission of diseases between animals and humans. Putting this together, we create dynamic One Health approaches to solve problems.

The transmission of many infectious diseases can be expressed as a set of compartments-- the number of susceptible individuals S , the infectious I , and the recovered or immune individuals R . This has led to the term 'SIR model'. Such models have been initially developed in 1760 by Daniel Bernoulli in Basel, and later by Kendall and McCormick in 1925. For now, we develop just the basic principles of such a transmission model to introduce how we simulate the transmission of infectious diseases between animals and humans. The arrows directed to a compartment is an increase of the amount and the arrows heading away a decrease in individuals. We apply here a deterministic approach. The compartment of the susceptible S , increases by the birth rate λ .

We consider that offspring from the infected or recovered compartment are susceptible, assuming no maternal transmission happens. The arrows with μ pointing away from the compartment is the mortality rate. It is considered equal in all compartments, as we assume that there are no fatalities due to the disease. For simplicity, we assume that we have a constant population size, which means birth rate equals mortality rate. Susceptible individuals become infected by a contact rate β . β is a product of the probability of transmission and contact. This is proportional to the size of the susceptible population S and the infectious population I . The size of the contact rate depends on the biological characteristics, like the mode of transmission, reproduction, and composition of the agent.

The infectious population I increases by the product βIS . Infectious animals recover with the rate δ , which is the inverse of the duration of the disease. Further, we assume a short disease duration in relation to the lifespan of the host and that recovered individuals have a permanent immunity to the infection. Now we add the human population below, in a similar way. Typically, humans get infected from contact with animals-- for example, for rabies by dog bites or for brucellosis by direct contact with sheep. The infected humans, I_H , are now calculated by α multiplied by I_A multiplied with S_H . This is the dynamic animal-human interface, allowing us to quantify the zoonotic potential of a given zoonosis.

To calculate this process, you can formulate each compartment with a differential equation, like this one. But this is a far more complex step. Important is to understand that we deal with nonlinear dynamics. For this, we can calculate the transmission between animals alone and between animals to humans. This shows that if you vaccinate a certain amount of animals you can interrupt the transmission to humans. By dividing the transmission constant β of the animals and the transmission constant α of animal-human transmission, we can estimate the zoonotic potential of a given disease. For example, for *Brucella melitensis* one in 15 infectious contacts with animals leads to a human infection. In *Brucella abortus*, only one in 150 infectious animal contacts lead to a human infection. For dog rabies this is one in 400, and for bovine tuberculosis this is lower than one in 1,000. With such models, we can thus estimate the comparative zoonotic potential in a given context. These equations have no analytical solution, but parameters can be estimated by iterative approximation.



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Here you see a simple example of the introduction of one infectious host into a population of susceptible hosts. After an initial epidemic peak, the transmission reaches a steady endemic state. We assume no extra mortality among the diseased, a relatively short course of the infectiousness, and lifelong immunity. Many zoonotic diseases are in a state of endemic stable transmission if they are uncontrolled. This means there is no epidemic wave but ongoing transmission. You can see this in the following example. We did a weekly recording of rabid dogs in N'Djamena, the capital city of Chad, over a duration of six years. The red spikes, left are the rabid dogs, and right you see the exposed humans. The blue lines are the best deterministic fit.