

Modelling the evolution of influenza

Annette Gerritsen - Epidemiologist at Epi Result.

On 18 April 2018, Venetia Karamitsou, PhD student in the Disease Dynamics group at the University of Cambridge, held a talk at SACEMA on modelling the evolution of influenza.

According to the WHO, there are 3 to 5 million cases of severe influenza infections per year, and 250-500,000 deaths per year, primarily among high-risk groups, which in industrialized countries are mainly the people over 65 and people with chronic illnesses. There is no treatment for influenza; there are antivirals, but these have risks associated with their use, so are mainly reserved for life-threatening cases. Thus, vaccination remains the most effective way of dealing with influenza. Given the importance of vaccination, it is worrisome that influenza mutates often, making reinfection possible even for vaccinated individuals.

Existing models regarding the evolution of influenza focus on either changes within hosts or between hosts. Between-host or population level models study how influenza spreads among many different individuals in a population. The prototypical example of such a model is the SIR model, which keeps track of the susceptible, infected and recovered individuals from the onset of an epidemic until its end. The within-host models study how influenza behaves inside one particular host. They keep track of target cells, which the virus can enter and turn into infected cells. These infected cells then start releasing more virus until they die, either due to damage from the virus within them or due to the actions of the immune system. So within-host models track, at their most basic level, the number

of target cells, infected cells and virions inside an individual. Finally, influenza is a rapidly mutating virus, which means that as it keeps accumulating mutations in its genome, it becomes so different that our immune system barely recognizes it anymore even if we have already been infected with it in the past. This evolution is referred to as the antigenic drift of influenza, and it is one mechanism by which the virus can keep re-infecting individuals.

The main motivation behind Venetia's research is to find out how we can combine both types of models, the within and between hosts models, into one. From her simulations it became clear that when you have one strong and one weak strain spreading inside a host, it could be (although counterintuitively) that the weak strain ends up becoming more prevalent in the whole population. The reason being that if you introduce a vaccine that targets specifically the strong strain, then the weak one may end up dominating the host. In that case the weak strain will be transmitted to someone else. If the same happens with other individuals in the population, it will be the weak strain that ends up more prevalent in the population.

Considering that vaccination is the main control strategy against influenza outbreaks, these results can be useful in reassessing vaccination policies to ensure both a low disease incidence rate as well as a decrease in antigenic drift speed of influenza.

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