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SHORT REPORT
Unhealthy behaviour is contagious: an invitation to exploit models for infectious diseases

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SUMMARY

We argue that the spread of unhealthy behaviour shows marked similarities with infectious diseases. It is therefore interesting and challenging to use infectious disease methodologies for studying the spread and control of unhealthy behaviour. This would be a great addition to current methods, because it allows taking into account the dynamics of individual interactions and the social environment at large. In particular, the application of individual-based modelling holds great promise to address some major public health questions.

Key words: Infectious disease epidemiology, modelling, public health.

Over the years many theories have been developed to explain why people engage in certain unhealthy behaviours and how these spread in populations [1]. These theories share the idea that behaviour is in some way influenced by social contacts. Yet, empirical studies of unhealthy behaviours generally investigate behavioural change processes from an individual perspective and until recently paid little attention to social environmental influences on behaviour. An intriguing exception is work from Christakis & Fowler [2, 3], who showed that both smoking and obesity spread from person-to-person, that the type of contact matters, and that groups can be distinguished within a social network. This lead to the idea that unhealthy behaviour is contagious and that it spreads in populations like an infectious disease. This has been suggested before conceptually [4], but there is a need to further operationalize this concept in ways that can be tested scientifically. Basically, adopting unhealthy behaviour is analogous to acquiring, say, influenza from a family member. Moreover, influenza tends to cluster in schools, which can also be observed for unhealthy behaviours. Tuberculosis and leprosy are even better examples of infectious diseases that show similarities with unhealthy behaviours: they cluster in households and communities, only a minority of those exposed eventually develop disease, and clinical signs may not be visible until several years after infection. Although there is considerable evidence that the spread of behaviours is explained by social influence, it is also true that similarity of behaviours observed in social networks may to some extent be the result of the tendency of people to select others with similar behaviours (homophily). Yet, it is difficult to disentangle homophily from social influence [5, 6].

Apart from contagiousness, other concepts and underlying mechanisms can be identified that are comparable for unhealthy behaviour and infectious diseases. First, an important concept in infectious

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diseases is heterogeneity, which can concern individual susceptibility to infection, infectiousness of a patient, and mixing patterns in the population [7]. Heterogeneity in susceptibility resembles variation in adopting unhealthy behaviours, such as stated in the theory of *Diffusion of Innovations* [8], which indicates that some people are more susceptible to adopt a behaviour than others. The rate of adoption further depends on the number of people in the social network that engage in a certain behaviour. Each individual has his/her own adoption threshold. For instance, some people are more self-efficacious than others, resulting in different levels of resilience. Heterogeneity in infectiousness can be compared with variation in social influence: position within networks, closeness of relationships, and number of contacts may explain why some people are more influential than others [9]. Heterogeneity concerning mixing patterns reflects that individuals tend to cluster within populations, e.g. according to age group or socioeconomic position. Second, a mechanism strongly related to heterogeneity is the presence of so-called super-spreaders. These are individuals that accelerate dissemination of an infection in a population, because of a prominent role in the contact network (i.e. many contacts) and/or high infectiousness. This greatly resembles opinion leaders or peer-role models, which are early adopters and can easily spread behaviours to others, due to their persuasiveness and high number of social contacts [8]. Third, vaccination is another concept that both fields share. Vaccination induces immunity, reduces the number of susceptible people, and reduces the risk of infectious diseases. In a similar way, social inoculation provides resistance to unhealthy behaviour by emphasizing refusal skills, and thus reducing the risk of adopting a behaviour [10]. Although vaccination and social inoculation are not exactly the same, they serve the same purpose. A fourth comparable mechanism is the influence of physical environmental factors. The physical environment promotes or discourages the spread of infections and behaviours in social networks through, e.g. climate and availability of fast-food, respectively. However, the availability of fast-food can also trigger a person to start unhealthy eating without any influence from the social environment.

The fact that the principles of infectious diseases and unhealthy behaviour show a remarkable resemblance challenges us to study unhealthy behaviour as an infectious disease. Infectious disease epidemiology has been studied for decades using sophisticated methods, in particular mathematical modelling, to analyse spread within populations, to predict the course of epidemics, and to evaluate interventions. As a major innovative step, Hill *et al*. [11] recently modelled the obesity epidemic as an infectious disease, using data from the Framingham Heart Study cohort [12]. The model mimics transitions between two compartments, i.e. susceptible (non-obese) and infected (obese) individuals. It also allows for possible spontaneous infections not resulting from contacts. The study concludes that the obesity epidemic is driven by both contagious and spontaneous infection and will stabilize at 42% of the population being obese within the next 50 years. However, as the authors indicate in an earlier paper [13], the proposed compartmental model is rather simplistic and does not take into account possible heterogeneities.

A major enhancement would be to go from compartmental modelling to a more comprehensive and realistic approach. Individual-based modelling is particularly useful to realistically model networks and individual heterogeneities. It simulates life-histories of individuals and specific interactions between individuals over time. Events, such as birth, death, relationship formation, transfer between social/risk groups, and acquisition of infection (behaviour), are modelled through chance processes. Another advantage is that it is more suitable for analysing the impact of interventions aimed at certain groups, such as households or schools. Individual-based modelling has proven to be very useful for practical decision-making in infectious disease control, starting with the ONCHOSIM model for river blindness control in West Africa [14, 15]. A more relevant model for sexually transmitted diseases, STDSIM, explicitly models individual contacts (sexual relationships) and formation of (sexual) networks [16]. Another recent example is the SIMCOLEP model for leprosy [17], in which the formation of and movement between households is modelled.

The application of individual-based modelling holds great promise to address some of the major questions in public health regarding health-related behaviours. Why are some people more open for unhealthy behaviours than other people? What are major determinants causing the adoption of certain behaviours? How can we best prevent unhealthy behaviour or promote behavioural change? These questions can only be answered adequately when taking into account the social context in which behaviours take place. Until now behavioural studies have

mainly focused on the individual in a static environment. The introduction of infectious disease methodology and in particular individual-based modelling would be a great addition, because it takes into account the dynamics of individual interactions and the social environment at large. This may result in new or revised interventions and policies. For instance, community interventions for behavioural change that only show small individual effects may eventually have substantial indirect public health effects. In contrast, some interventions with large individual effects may ultimately have a small impact on the population, due to a limited reach. Individual-based modelling in particular allows translating individual effects to population impact. Moreover, infectious disease modelling provides useful key concepts, such as the basic reproduction number (R_0), i.e. the average number of successful transmissions per infectious person in a fully susceptible population. An outbreak of, e.g. smoking in a non-smoking population will occur if $R_0 > 1$, which indicates that each smoker will on average trigger at least one other individual to start smoking. The goal is to reduce R_0 to below 1, to stop further spreading of smoking.

In conclusion, the spread of unhealthy behaviour shows marked similarities with infectious diseases, and hence embracing existing infectious disease methods is beneficial. A first attempt to apply infectious disease modelling for unhealthy behaviours has now been published, but there is substantial room for improvement by including the dynamics and heterogeneities of social networks. The field of research aimed at studying health-related behaviours and at developing interventions and policies to promote health behaviours may benefit substantially from further exploiting models for infectious diseases, in particular individual-based models.

DECLARATION OF INTEREST

None.

REFERENCES

1. **Glanz K, Bishop DB.** The role of behavioral science theory in development and implementation of public health interventions. *Annual Review of Public Health* 2010; **31**: 399–418.
2. **Christakis NA, Fowler JH.** The spread of obesity in a large social network over 32 years. *New England Journal of Medicine* 2007; **357**: 370–379.
3. **Christakis NA, Fowler JH.** The collective dynamics of smoking in a large social network. *New England Journal of Medicine* 2008; **358**: 2249–2258.
4. **Gladwell M.** *The Tipping Point: How Little Things Can Make a Big Difference*. New York: Little, Brown and Company, 2000.
5. **VanderWeele TJ.** Sensitivity analysis for contagion effects in social networks. *Sociological Methods & Research* 2011; **40**: 240–255.
6. **Shalizi CR, Thomas AC.** Homophily and contagion are generically confounded in observational social network studies. *Sociological Methods & Research* 2011; **40**: 211–239.
7. **Anderson RM, May RM.** *Infectious Diseases of Humans: Dynamics and Control*. Oxford University Press, 1991.
8. **Rogers EM.** *Diffusion of Innovations*, 5th edn. New York: The Free Press, 2003, pp. 576.
9. **Valente TW.** *Social Networks and Health*. Oxford: Oxford University Press, 2010.
10. **Evans RI, Raines BE, Hanselka L.** Developing data-based communications in social psychological research: adolescent smoking prevention. *Journal of Applied Social Psychology* 1984; **14**: 289–295.
11. **Hill AL, *et al.*** Infectious disease modeling of social contagion in networks. *PLoS Computational Biology* 2010; **6**: e1000968.
12. **Dawber TR.** *The Framingham Study: The Epidemiology of Atherosclerotic Disease*. Cambridge, MA: Harvard University Press, 1980.
13. **Hill AL, *et al.*** Emotions as infectious diseases in a large social network: the SISa model. *Proceedings of the Royal Society of London, Series B* 2010; **277**: 3827–3835.
14. **Plaisier AP, *et al.*** ONCHOSIM: a model and computer simulation program for the transmission and control of onchocerciasis. *Computer Methods and Programs in Biomedicine* 1990; **31**: 43–56.
15. **Remme JH.** Research for control: the onchocerciasis experience. *Tropical Medicine & International Health* 2004; **9**: 243–254.
16. **Korenromp EL, *et al.*** HIV spread and partnership reduction for different patterns of sexual behaviour–a study with the microsimulation model STDSIM. *Mathematical Population Studies* 2000; **8**: 135–173.
17. **Fischer E, *et al.*** Different mechanisms for heterogeneity in leprosy susceptibility can explain disease clustering within households. *PLoS ONE* 2010; **5**: e14061.