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Comment

The importance of accurately modelling human interactions Comment on "Coupled disease–behavior dynamics on complex networks: A review" by Z. Wang et al.

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Human behaviour and disease dynamics can greatly influence each other. In particular, people often engage in self-protective behaviours that affect epidemic patterns (*e.g.*, vaccination, use of barrier precautions, isolation, *etc.*). Self-protective measures usually have a mitigating effect on an epidemic [16], but can in principle have negative impacts at the population level [12,18,15]. The structure of underlying social and biological contact networks can significantly influence the specific ways in which population-level effects are manifested. Using a different contact network in a disease dynamics model—keeping all else equal—can yield very different epidemic patterns. For example, it has been shown that when individuals imitate their neighbours' vaccination decisions with some probability, this can lead to herd immunity in some networks [9], yet for other networks it can preserve clusters of susceptible individuals that can drive further outbreaks of infectious disease [12].

Wang et al. [16] give an overview of these and other recent developments, covering a wide range of networks that have been applied in the study of disease–behaviour dynamics and the varying epidemiological and behavioural results that arise when different networks are applied to similar systems. They argue convincingly that results are sensitive to network structure and highlight studies that exemplify the sometimes surprising ways in which behaviour can influence an epidemic depending on the network structure being considered. The authors also emphasize the subtleties that must be considered when making public health recommendations and suggest how research in this field would be best served in the future, including the sensible recommendation for better collaboration between scientific disciplines such as physics, behavioural sciences, mathematical modelling and epidemiology.

Wang et al. discuss two distinct methodologies that have been used to construct coupled disease–behaviour models. One approach is to combine an epidemiological model with a game-theoretical framework to *explicitly* incorporate individual decisions, the utility of which depend on the behaviour of others (*e.g.,* [3,2,1,13]). Another approach is to modify an epidemiological model to account for behaviour *implicitly* by making transmission rates or other parameters depend, for example, on disease prevalence or mortality (*e.g.*, [11]). The effects of non-homogeneous contact networks [14] can be investigated with either approach.

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There is also a growing body of work, not mentioned in Wang et al.'s review [16], that focusses on attempting to fit behavioural disease models to epidemic data and use statistical inference methods to explain observed patterns of incidence and mortality [6,4,5,11]. For example, it has been shown that individuals in the United States likely adjusted their contact rates in response to public health measures taken during the 1918 influenza pandemic [4], and that social distancing may have influenced the course of the influenza epidemic in Sydney, Australia during the same year [5]. Other work has found that the three waves of infection seen in the United Kingdom during the 1918 pandemic can been explained successfully only with a behavioural model [10,11]. Modelling also indicates that school closures during the 2009 influenza pandemic had significant effects on disease dynamics [17,8]. The inferences drawn based on such work offer valuable insight into the interplay between human behaviour, contact network structure and epidemic progression.

While Wang et al. state that "network topology will directly determine the threshold of epidemic outbreak" [16], and review work that shows how sensitive other epidemic characteristics are to network topology, they do not indicate which networks may be most realistic. Comparisons of theoretically constructed and empirically derived networks (*e.g.*, [7]) would help to distinguish which of the many possible results should be most seriously considered.

It is evident that the interactions between disease dynamics and human behaviour are complex, and all the more so if realistic contact networks are considered. The research that Wang et al. review may become more and more relevant over time, as the level of detail at which real contact networks are understood expands (*e.g.*, as a result of cell phone usage and social media). It will be interesting to see the extent to which disease–behaviour modelling research is taken up by those who are in a position to make public health decisions in the future.

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