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Reply to comment

Spatial coupled disease–behavior framework as a dynamic and adaptive system
Reply to comments on "Coupled disease–behavior dynamics on complex networks: A review"

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We would like to begin this response by recognizing all the insightful and thought-provoking comments to our review "Coupled disease–behavior dynamics on complex networks" [1]. We find that, with their diverse expertise, all the commentators enrich the discussion on this topic, and also identify important, interesting questions [2–13], indicating how much space there still is for the development of the field. To give the readers a systematic understanding, these opinions and suggestions are roughly divided into two classes: (i) whether the coupled models could be closer to realistic observations, yet simpler [2–5,7–10,13]; and (ii) whether the hypothesis of network models could mimic the empirical networks more accurately [5–8,10–13].

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Human behavioral response to disease spreading has been recognized to have great influence on epidemic dynamics. Based on the interplay between individual behavior and epidemic diffusion (*i.e.* the so-called coupled disease–behavior dynamics) in complex networks, many effective prevention measures, such as voluntary vaccination and disinfection, have been proposed and studied, both theoretically and empirically [14–16,16,17]. However, as Ref. [2] pointed out, an individual's behavioral response is subject to a myriad of local and global information in realistic cases; how to integrate such economic, cultural, and political factors into the present framework will be an interesting challenge. One well-known example is to consider subsidy policy as a governmental incentive strategy [18], where the complex interplay among multiple dynamics (including epidemic spreading, governmental policy, and behavioral response) may decrease the potential for infection. Similarly, due to the fact that perceived infection risk is closely related to rumors, media reports, and the economic level of families, its estimation (*i.e.* Eq. (5) of Ref. [1]) should also become more adaptive in future research, depending on the situation at hand. We thus agree with the comment that instilling the present framework with greater psychologically- and socioeconomically-grounded structure (using growing social science data) will yield a deeper and better understanding of public health and corresponding coupled dynamics.

Along the same lines, Holme [9] emphasizes that, though the coupled framework sheds new light on disease prevention research, the impact of irrational aspects of an individual's behavior should be studied in future research. We agree, and we note this brings up an important issue that has often caused confusion in the field. Individual behavioral responses guided by strategic (game theoretical) interactions can easily lead to a social dilemma: if a person can benefit from the actions of others (e.g. others' vaccinating to generate herd immunity), then avoiding the personal cost of taking the action will be the optimal choice for that person (*i.e.* that person can 'free-ride' on the actions of others). Economists–alone out of all fields–describe this behavior as 'rational'. Some individuals who do not vaccinate invoke irrational arguments, as Holme notes [9]. This raises the question: is a person who uses irrational arguments to rationalize their 'rational' free-riding behavior rational, or irrational? The answer depends upon whom you ask and what field they were trained in. A related point is that coupled disease–behavior frameworks derived from evolutionary game theory are often described in the coupled disease–behavior literature as making the 'rational individual' assumption, when in fact those models include social processes such as social learning and social norms, and psychological limits to behavior such as adoption of 'rules of thumb' in decision making [19], which are not really characteristic of the 'rational actor' model of classical economics where individuals are perfect and selfish optimizers. Clearly, the field has language issues that need to be sorted out. In any case, the processes that Holme refers to–the uncertainty of imitation/learning behavior–are ubiquitous and usually exhibit heterogeneous distributions; how to improve the present assumption of imitation/learning behavior is thus a very promising area of further study. Similarly, Small [10] advocates a larger role for behavioral rules, which should likewise exhibit great heterogeneity in real populations (*i.e.* moving beyond the hypothesis of utility maximization). We fully agree and believe that this valuable suggestion may promote interdisciplinary research long into the 21st century. Furthermore, interdisciplinary collaborations with behavioral scientists and epidemiologists will be an important way to achieve these goals and move the field forward, as commented by Wells and colleagues [13], who also discuss the 2014 Disneyland, California measles outbreak and the 2012 MERS outbreaks as motivating examples of how the sociological details of behavior can matter and how they can interact nontrivially with epidemiological considerations.

Others comment that for the models to be more valuable in practice, another aspect deserving great attention is to make the present framework simpler and more adaptive [3,8]. Ideally, one would wish to decrease the dimensionality of the model's parameter space, yet retain or enhance the theoretical accuracy, which is particularly relevant to studies that seek control methods of new emerging diseases [20,21]. This point is beneficial for the setup of multiple strategy models as well. For example, allowing a third, self-protective strategy in a multi-strategy framework results in a counter-intuitive phenomenon analogous to the well-known Braess's Paradox, as well as self-organization patterns in networks [22], but the larger number of parameters makes the exploration of such observations more difficult. Indeed, the larger the number of model parameters, the less universal the predictive ability of such a model becomes. In this sense, we agree that a sensible first step in theoretical modeling of coupled disease–behavior dynamics (or any phenomenon in nature) is to first develop the lower-dimensional parameterizations of coupled dynamics in multiple-strategy campaigns or realistic scenarios, which may also turn out beneficial for policy makers. Similarly, Aguiar notes that parsimony is a requirement of coupled behavior–disease modeling, and the complexity of the model needs to depend on the scientific question being addressed and the temporal scale [7]. Availability of empirical data is a further consideration. Aguiar [7] notes that the current models do not have predictive power, but in fact some relatively