



### Commentary

## Network multipliers and public health

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#### Introduction

The term 'spillover effect' is sometimes used in the social and biomedical sciences to describe one person's exposure affecting the outcome of another.<sup>1-6</sup> The term 'contagion' is sometimes used to describe one person's outcome affecting another's outcome.<sup>6–9</sup> One classic setting for such spillover and contagion is that of vaccines and infectious diseases. The vaccination status of one person may affect the infection status of a family member (spillover) and infection status of one family member may affect the infection status of another (contagion).<sup>6,9-11</sup> Indeed, much of the methodological work in describing these phenomena has arisen from infectious disease epidemiology, wherein 'spillover' is also sometimes referred to as an 'indirect effect' or 'interference'.<sup>2,6,12-16</sup> However, the phenomena themselves are much more widespread than infectious disease contexts and may pertain to health behaviours, affective states, learning outcomes, judicial and voting behaviours and to many other settings, exposures and outcomes.<sup>5,7-9,17-25</sup> Indeed, arising in part from the recognition of the importance of spillover in a variety of contexts, the development of formal methodology to analyse spillover and contagion has begun to rapidly advance in a number of fields beyond the infectious disease setting.<sup>1</sup>

In two recently published articles in the *International Journal of Epidemiology*, Benjamin-Chung *et al.*<sup>1,2</sup> have done a tremendous service to epidemiologists in providing an overview of the types of questions and methodologies that may be relevant in understanding spillover effects,<sup>2</sup> and in providing a survey of current applications of these approaches.<sup>1</sup> Other overviews of the methodological literature on spillover, framed in terms of counterfactuals, is provided elsewhere.<sup>26</sup> It is clear that both the infectious disease context (in which such spillover effects are most frequently considered) and also other settings are of interest and relevance. Indeed, here, we argue that spillover needs to be considered far more frequently when thinking about epidemiology and public health; otherwise, we may miss important opportunities for public health impact and evaluation. To that end, we introduce a new relevant metric: the network multipler.

#### **Network multipliers**

Spillover effects and contagion should be taken into account when assessing the public health impact of an intervention and also its cost-effectiveness.<sup>27</sup> For example, with a protective intervention where the baseline risk of the outcome is  $p_0$  and the risk ratio for the outcome for the treatment or intervention under consideration is RR, we could calculate a number needed to treat as  $1/[p_0(1-RR)]$ . For cost-effectiveness, the cost per life saved (or disease cured or prevented) would be (cost per treatment)/[ $p_0(1-RR)$ ].

Now suppose that, in addition to an intervention having an effect on its directly treated recipients, it also had spillover effects on various individuals in a person's family or community or social group. Consider an intervention in which some subset of individuals within each community are treated, and suppose that there are potential spillover effects within communities, but not between communities. Suppose further that the average number of untreated persons for whom the spillover effect was operative, for each treated person, was N, and that the spillover effect of the intervention on untreated persons was RR<sub>s</sub> on the risk ratio scale. It is straightforward to show (see Appendix) that for each person who was directly treated and cured, we would expect an additional N x  $(1-RR_s)/(1-RR)$  individuals cured because of the spillover effect. Because of the spillover effects, we would thus essentially multiply the effectiveness of our treatment by a factor of:

$$NM = 1 + N x (1 - RR_s) / (1 - RR)$$
(1)

to obtain the overall impact on the number cured from administering a single intervention.

We will refer to this quantity above as a network multiplier (NM), defined as the ratio of the total number of changed outcomes due either to someone being directly treated or to spillover, as compared with the number of changed outcomes due only to someone being directly treated. For each person directly cured because of treatment, we actually get a total of  $NM = 1 + N \times (1 - RR_s)/$ (1-RR) cured because of the additional effects of spillover. The network multiplier will be greater than or equal to 1, unless it is the case that the direct effect on the individual who actually received treatment and the spillover effects are in opposite directions (for instance, as might arise if effective treatment also happens to prolong the communicable period). Thus, provided the direct effect and spillover effect are in the same directions, we always 'cure' more people than simply looking just at direct-effect risk ratios would suggest. The calculations are exactly analogous if the outcome is a positive desirable one with risk ratios above 1, or an undesirable one with risk ratios below 1.

Once we have calculated these network multipliers, we would also want to adjust our numbers-needed-to-treat calculations, and our cost-effectiveness calculations, accordingly. For the numbers-needed-to-treat and for cost-effectiveness, we would divide the typical expressions and calculations by the network multiplier. The number-needed-to-treat would thus be  $1/[p_0(1-RR)NM]$  and the cost-effectiveness would be (cost per treatment)/ $[p_0(1-RR)NM]$ .

As an example of the calculation of a network multiplier outside the infectious disease context, Nickerson<sup>28</sup> examined the effect of a get-out-the-vote message in a randomized experiment in Minneapolis, Minnesota. Households with two registered voters were randomized to receive either face-to-face encouragement to vote, or faceto-face encouragement to recycle. The message was delivered to whichever registered voter answered the door, and then it was later recorded whether or not each registered household member subsequently voted. The effect of the intervention on the recipient of the face-to-face message was an increase in the probability of voting with RR = 1.67; the effect of the intervention on the other member of the household who did not directly receive the face-to-face message was an increase in the probability of voting with  $RR_s = 1.36$ . Here, because there is only one additional person per household who may benefit from the spillover effect, we have N = 1 and the network multiplier is thus:

$$\begin{split} NM &= 1 + N\,x\,(1 - RR_s)/(1 - RR) \\ &= 1 + 1\,x\,(1 - 1.36)/(1 - 1.67) = 1.54 \end{split}$$

For every person for whom the face-to-face message alters their voting behavior, there are a total of 1.54 persons who in fact vote because of the intervention, who would not have otherwise done so (i.e. an additional 0.54 person due to spillover).

The network multiplier is in some ways analogous to the reproduction number,  $R_0$ , in the infectious disease context,<sup>29,30</sup> defined as the number of cases that one case of infection generates on average over the course of its infectious period, in an otherwise uninfected population. In the infectious disease context, when  $R_0 < 1$ , the infection will in general die out relatively quickly; but if  $R_0 > 1$ , the infection will spread through the population. There are arguably some differences across contexts. Often, when assessing spillover effects outside of infectious diseases, analyses are not carried out on effects across an entire population but are restricted to the spillover effects in one's own more local and immediate social group or cluster (e.g. household, circle of friends etc.). Sometimes it is assumed that there are only spillover effects within clusters of individuals, not between clusters, an assumption sometimes referred to as 'partial interference'.<sup>5,14</sup> It is this assumption that underlies the network multiplier formula given above in equation (1). The network multiplier formula given in (1) is thus not immediately applicable to sociocentric settings in which there are not distinct clusters, but only a single social network.

However, in many cases, proceeding with the 'partial interference' assumption of no spillover between clusters may lead to conservative estimates of the network multiplier if the treatment for the treated individual has effects on the treated individual's social group, and then the effects on the members of this social group spread further to individuals who may be part of other social groups not otherwise socially connected to the original treatment recipient. Such issues about multistage transmission of treatment and outcomes throughout a network and population may seem more likely to occur in the context of infectious disease than with health behaviours such as smoking or diet. However, recent social network analyses involving both observational and experimental approaches do suggest some evidence for non-negligible spread of certain health behaviours and other outcomes up to three degrees of separation from the individual that was initially treated.<sup>7,23,24,31–33</sup> Thus, if one used risk ratio estimates from a randomized trial with outcome data also collected on each individual's social group, the network multiplier calculation might represent a conservative estimate of the actual network multiplier for the entire population.

#### Conservative estimates of network multipliers using contagion

Even in the absence of data on spillover effects, we might be able to get conservative estimates of such network multipliers using estimates from contagion effects (one person's outcome affecting another's). Suppose, for example, that we were considering a smoking cessation support intervention, and that the intervention's risk ratio for ceasing to smoke at the end of follow-up was RR = 1.5. We might use this risk ratio and also information on the rate of smoking cessation in the control group to estimate the number needed to treat (NNT) or the cost-effectiveness of the intervention. However, suppose all participants in the programme were in fact married. Evidence from observational studies of contagion effects for smoking cessation<sup>19</sup> suggests that when someone quits smoking, their spouse is then 67% [95% confidence interval (CI): 59%-73%] less likely to smoke subsequently. For every person for whom the smoking cessation support intervention successfully led to smoking cessation, we might then expect this to lead to, say, an additional 67% x (prevalence of smoking among spouses) individuals who cease smoking because their spouse did as a result of the smoking cessation intervention. In cost-effectiveness and number-needed-to-treat calculations, we would likely want to take that into account as well. We might use as the 'network multiplier' the quantity [1+67% x (prevalence of smoking among spouses)]since, for every person 'cured' by the intervention, we have an additional 0.67 x (prevalence of smoking among spouses) of the spouses 'cured' as a result as well.

This would, moreover, likely be an underestimate of the network multiplier since the smoking cessation intervention programme might potentially also affect the smoking behaviouur of the spouse because of the transmission of the information from the programme, even if the original programme recipient did not, in the end, quit smoking. Said another way, the contagion effect of the person ceasing smoking on the spouses' smoking cessation status is only part of the spillover effect of the intervention itself.<sup>6</sup> However, if the effect on the spouse that is independent of the outcome of the treatment recipient were relatively small, then the approximation above (just using the contagion effect and the prevalence of the exposure among the spouses) may not be far off from the actual network multiplier. If smoking cessation of the treatment recipient affected not only the spouse but also the smoking behaviour of other members of the household or the community, then this too could potentially be included in the network multiplier calculation.

#### Implications for public health

What are the implications of such spillover effects and network multipliers for public health? Traditionally, the public health impact of an exposure is often assessed as a function of the prevalence of the exposure and the magnitude of its effect on relevant outcomes, and this is sometimes captured in terms of attributable fractions.<sup>34–37</sup> If an exposure has large effects and is common, then its impact on population health may well be substantial. However, this traditional and important framework for thinking about public health impact does not take into account spillover. We might thus supplement the traditional framework with this notion of the network multiplier. The public health impact of an exposure is shaped by its prevalence, its effect size and its network multiplier.

We could thus expand our understanding of public health impact to include not only prevalence and effect size, but also network multipliers. The network multiplier is not entirely separate from exposure prevalence and effect size, but it instead concerns both of these quantities in relation to individuals who have not been directly treated or exposed.

The magnitude of the network multiplier itself depends on multiple aspects of: (i) the structure of the network; (ii) the attributes of the individuals in the network; and (iii) the attributes of the item that is spreading (e.g. the pathogen). This can be illustrated by the following fanciful example. Imagine that there are two islands, each with 100 people. On one island, every person is paired with another and is sexually monogamous, and on the other, everyone has intercourse with everyone else. On neither island is there initially any sexually transmitted disease (STD). Now suppose a sailor with an STD washes ashore and has intercourse with one person. The epidemic will be larger on the latter island than on the former. Here the structure of the network matters, and the social network structure alters the number of additionally affected individuals, N, in the network multiplier calculation. Now, suppose that we have two islands in which everyone has intercourse with everyone else but, in one island, everyone is immunocompetent and, in the other, everyone is immuncompromised. Once again, suppose a sailor washes ashore and has intercourse with one person. It is clear that the epidemic will be larger on the latter island. The attributes of the individuals in the network, and not just the structure of the network, matter and in this case the attributes of the individuals alter the magnitude of the spillover effect, RR<sub>s</sub>. Now, finally, consider the scenario in which there are two islands in which everyone has intercourse with everyone else, and everyone has intact immune systems. Suppose that on the first island, a sailor with HIV washes ashore and, on the second, a sailor with active syphilis washes ashore. It is known that the probability of transmission from one person to another with these two pathogens based on a single sexual encounter varies by more than an order of magnitude-one is much more likely to contract syphilis than HIV. It is clear, again, that the epidemic of syphilis will be larger. Here an intrinsic attribute of the item that is spreading is also crucial, and this intrinsic attribute again alters the magnitude of the spillover effect, RRs, in the network multiplier calculation. A network multiplier may thus itself be enhanced or dampened by altering: (i) the structure of the network; (ii) the attributes of the individuals in the network; or (iii) the attributes of the item that is spreading.

Ignoring such network multipliers has important implications for cost-effectiveness research and may yield substantial underestimates.<sup>27</sup> Moreover, if different interventions have spillover effects of different magnitudes and hence different network multipliers, this could in fact alter which of two interventions is deemed to be more costeffective. For instance, hip replacement might be formally assessed as more cost-effective than cataract surgery but, if fixing someone's cataract added more quality-adjustedlife-years to the person's spouse than fixing someone's hip, both the cost-effectiveness assessments and the relative standing of the two interventions could be reversed.

There may of course be a temptation, when making use of such network multipliers, to exaggerate the spillover effects. For decision making concerning a single potential intervention, it might be best to try to mitigate these dangers by using more conservative calculations in the application of network multipliers; and indeed, the calculations proposed above all were intended to err on the conservative side. However, when two different intervention strategies are being compared, erring on the conservative side for both may not be sufficient, since differences in the degree to which the network multiplier is specified under its true value may alter which of two interventions seems most cost-effective. Such issues should be considered more frequently in thinking about population health. Although considerable research has gone into understanding spillover effects and network effects, much of the analysis and thinking in epidemiology remains at the individual level. Outside of the infectious disease context, the consequences of spillover and contagion for public health impact and population health are often neglected.

# Networks multipliers, social media and public health

Another important consequence of thinking about network multipliers in public health is that if an intervention has a particularly large network multiplier, its public health implications may be very large indeed, even if the effect size or even the original prevalence of exposure is modest. This also is perhaps especially relevant given the use of contemporary media, since it has potential both for massive outreach (high prevalence of exposure) and massive spillover/contagion (large network multipliers). Contemporary media, therefore, from news outlets to online social network sites, have the capacity to transform what are in fact relatively small, perhaps even tiny, effects into something with potentially very large public health consequences.

As an example in which public health consequences may be massive even though initial effect sizes are small, consider the increasingly negative content of media communications. There is evidence that the content of media has become increasingly negative and polarizing.<sup>38</sup> The negative content of media is likely in part motivated by the fact that the human brain is more likely to be attracted to, carefully watch and become fixated upon something that is negative than something that is positive, perhaps as an adaptive survival response (a phenomenon often referred to as 'negativity bias'<sup>39</sup>). Media sources with negative material thus end up with more viewers.<sup>40</sup> There is evidence also that the witnessing of a positive event is more likely to result in someone subsequently acting altruistically towards another,<sup>31,41–43</sup> and that the witnessing of a negative event or violence is more likely to result in more negative actions and behaviours towards others.44,45 Finally, evidence continues to emerge that altruistic behaviours are themselves subject to considerable contagion<sup>31,46,47</sup>: the recipient of an action of goodwill is more likely to go on to do the same. The contagion effects of altruistic action may extend so far that a positive interaction between two persons can travel through a social network by contagion, and ultimately positively affect the actions of two other persons, neither of whom know either person in the original pair.<sup>31</sup>

The consequences of this chain of causation from the witnessing of negative or positive events, through negative or positive personal interactions, and the spread by contagion through a social network, may be very powerful indeed. Whereas the effect size of negative, divisive or polarizing media reporting or social media posts may, at the individual level, be very tiny, the capacity to both reach large numbers (e.g. through a large social media following) and spread massively through a social network (e.g. by sharing and re-posting) may result in social and public health consequences that, at the population level, bring about considerable harm in human interaction, and subsequently also health.

Media reporting of negative events does have an important role in bringing awareness of society's problems and ills. However, in light of the very strong spillover and contagion, greater effort should perhaps also be given to balance negative reports with those that comment upon what is good in the community or what individuals or groups are doing to bring about a better world. Given the available empirical evidence, it seems that small changes in reporting practices could have substantial beneficial impacts.

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#### Appendix

Suppose that the baseline risk of an adverse outcome for those untreated is  $p_0$ , and that an intervention for someone directly treated reduces the risk by risk ratio RR so that the risk with the treatment is  $p_0$ RR. Suppose further that for each treated person in a community, there are on average an additional N untreated individuals in the community who also indirectly benefit from the intervention (by spillover or contagion), such that the intervention reduces their risk by risk ratio RRs. For the directly treated individuals, it will be necessary to treat 1/[p<sub>0</sub>(1-RR)] per outcome averted. If  $1/[p_0(1-RR)]$  treatments were directly applied, then one outcome would be averted for these directly treated individuals. For those in the directly treated individuals' communities who were not directly treated, the expected number of outcomes among those not directly treated, for each directly treated individual's outcome that was averted, would be reduced because of spillover from  $N x p_0 x 1/[p_0(1-RR)]$  to  $RR_s x N x p_0 x 1/[p_0(1-RR)]$ . This would result in a further reduction in outcomes of magnitude  $\{N x p_0 x 1/[p_0(1-RR)]\} - \{RR_s x N x p_0 x 1/(P_0(1-RR))\}$  $[p_0(1-RR)] = Nx (1-RR_s)/(1-RR)$ . Thus the ratio of the total number of changed outcomes due either to someone being directly treated or to spillover, as compared with the number of changed outcomes due only to someone being directly treated, is given by  $NM = 1 + N x (1 - RR_s)/$ (1 - RR).