

# Endogenous social distancing and its underappreciated impact on the epidemic curve

Sebastian Funk<sup>a</sup>, Marko Gosak<sup>b,c</sup>, Moritz U. G. Kraemer<sup>d,e</sup>, Heinrich H. Nax<sup>f,g,\*</sup>, Matjaž Perc<sup>b,h,i</sup>, and Bary S. R. Pradelski<sup>j</sup>

<sup>a</sup>London School of Hygiene and Tropical Medicine, Keppel St, WC1E 7HT London, UK; <sup>b</sup>Faculty of Natural Sciences and Mathematics, University of Maribor, Koroška cesta 160, 2000 Maribor, Slovenia; <sup>c</sup>Faculty of Medicine, University of Maribor, Taborska ulica 8, 2000 Maribor, Slovenia; <sup>d</sup>Department of Zoology, Mansfield Road, University of Oxford, OX1 3SZ Oxford, UK; <sup>e</sup>Harvard Medical School, 25 Shattuck St, 02115 Boston, USA; <sup>f</sup>Behavioral Game Theory, Clausiusstrasse 37, ETH Zurich, 8092 Zurich, CH; <sup>g</sup>Institute of Sociology, Andreasstrasse 15, University of Zurich, 8050 Zurich, CH; <sup>h</sup>Department of Medical Research, China Medical University Hospital, China Medical University, Taichung, Taiwan; <sup>i</sup>Complexity Science Hub Vienna, Josefstädterstraße 39, A-1080 Vienna, Austria; <sup>j</sup>Univ. Grenoble Alpes, CNRS, Inria, Grenoble INP, LIG, 38000 Grenoble, France

This manuscript was compiled on May 9, 2020

1 **Social distancing is an effective strategy to mitigate the impact of infectious diseases. If either sick or healthy individuals, or both, socially**  
2 **distance, the epidemic curve flattens. Substantial amounts of contact reductions occur endogenously during a disease outbreak: Some are**  
3 **due to health-related mobility loss (severity of symptoms), duty of care for an infected person in the same household, and forced quarantine.**  
4 **Other changes are due to voluntary social distancing. In particular, sick people reduce contacts to avoid infecting others, and healthy**  
5 **individuals do so in order to stay healthy. We use game theory to formalize the interaction involving voluntary social distancing in a partially**  
6 **infected population. This improves the behavioral micro-foundations of epidemiological models and predicts differential social distancing**  
7 **dependent on health status. The model's key predictions in terms of comparative statics are derived, which concern changes and interactions**  
8 **of endogenous differential social distancing behaviors. We fit the relevant parameters for endogenous social distancing in an epidemiological**  
9 **model with evidence from influenza waves and the current COVID-19 pandemic, and use these fits to provide a benchmark for an epidemic**  
10 **curve with endogenous social distancing. Our results suggest that a curve similar in peak and case numbers to what resulted from a**  
11 **lockdown, yet quicker to pass, could have occurred endogenously. Going forward, eventual social distancing orders and policies should be**  
12 **benchmarked against more realistic curves that take endogenous social distancing into account, rather than be driven by unrealistic horror**  
13 **scenarios that are based on static estimates for social mixing.**

Social distancing | Game theory | Disease spreading | Contact rates

1 **T**he contact rates of infectious and non-infectious agents play a key role in determining the epidemic  
2 curve. For example, when individuals infected with the SARS-CoV-2 virus have limited contact with  
3 susceptible individuals because of rapid case-isolation policies, transmission can be reduced effectively (1).  
4 How effective less stringently enforced policies and recommendations of social distancing or isolation are  
5 depends in general on the individual-level decisions regarding whether or not to adhere to social distancing  
6 voluntarily and/or in response to social distancing policies.

7 Governments seem to disagree strongly regarding how much freedom of choice their citizens are ideally  
8 entrusted with in order to achieve social distancing, resulting in less (e.g. Sweden) and more (e.g. China)  
9 stringent policies. To identify adequate policy responses, it is important to understand *behavioral change*  
10 in epidemiological models (see reviews by (2–4)). However, the interactive nature of the behavioural  
11 change in social distancing during infectious disease outbreaks (in contrast to the contexts of decisions for  
12 vaccination (5–7) and antiviral prophylaxis (8)) have not been explored in much detail, in particular not  
13 regarding the individual-level game-theoretic foundations of social distancing, and how these compare with  
14 real-world evidence. Progress in this direction ought to be made, because game-theoretic analyses have  
15 shown that interactions can crucially shape the epidemic curve (9–11), and modeling increasingly rests on  
16 rich assumptions regarding how individual behavior changes dynamically with the disease outbreak.

17 In the context of policy-relevant COVID-19 modelling, some assumptions made regarding behavioral  
18 change had to be made without adequate empirical foundations. The early simulations “driving the world’s  
19 response to COVID-19” (12), for example, were based on static estimates of social mixing. These simulations  
20 painted horrific scenarios in terms peak and case numbers of the outbreak, which led to the introduction of  
21 social distancing policies across (most of) the globe. To evaluate which policies should be used, modelers  
22 again made assumptions regarding how social distancing policies would be adopted in terms of reductions  
23 of the population—as this example on how a population would respond to a recommendation of voluntary  
24 home quarantine from (13) illustrates: “Following identification of a symptomatic case in the household, all  
25 household members remain at home for 14 days. Household contact rates double during this quarantine

26 period, contacts in the community reduce by 75%. Assume 50% of household comply with the policy.”

27 What numbers are chosen precisely is important to justify policies, yet it is unclear what these particular  
28 ones were based on—theory, evidence or introspection. What is clear is that understanding the exact nature  
29 of individual-level incentives and responses underlying decision-making better is important as it helps  
30 to elucidate when and whether there are conflicts of interest between individual and collective interests  
31 (5), or not (14). This is an important factor when governments choose between health and mobility  
32 recommendations and forced quarantine and lockdown measures. Hence modeling must move from making  
33 mobility assumptions to theoretically and empirically validated mobility ingredients. Here, making such a  
34 step toward establishing a suitable framework for integrating behavioral micro-foundations of mobility, we  
35 draw on game theory to embed interactive decision-making of social distancing in an epidemiological model.

36 Recently, many countries enforced strict restrictions on movements and social interactions, because the  
37 general impression was that voluntary social distancing recommendations would not be sufficient (15).  
38 Quite plausibly, due to the large economic and social impact of country-wide lockdowns, governments  
39 increasingly consider restricting human movements and social contact dependent on health and risk status  
40 so as not to lock down the entire population (16). The issue is that research has not yet provided empirical  
41 benchmarks for differential mobility in disease scenarios, so it is unclear how such policies can be evaluated:  
42 Any policies aimed at reducing mobility should be benchmarked against what mobility would have been  
43 without such policies in light of the disease, not against what mobility was like before the disease. We here  
44 propose a game-theoretic model of social distancing behavior in order to provide avenues for formulating  
45 such benchmarks, and compare its predictions to observations of contact rates during two influenza seasons  
46 in the United Kingdom where human contact was not affected by specific government restrictions. The  
47 observed restrictions might be viewed as lower bounds on the counterfactual endogenous levels of social  
48 distancing that ought to be expected in the current COVID-19 situation if no explicit policies had been  
49 imposed. We show that levels of endogenous social distancing as would be expected from an influenza  
50 season would already flatten the epidemic curve substantially, and that social distancing orders would really  
51 have to be quite effective to warrant their introduction given such counterfactuals.

## 52 **Modelling infectious disease dynamics**

53 The close monitoring and detailed modeling of outbreaks of infectious diseases has become an increasingly  
54 active research focus in epidemiology since the seminal works by (17–19) and (20, 21). Over the past  
55 decades, the emergent body of epidemiological research has substantially improved our understanding of  
56 the dynamics of infectious diseases as well as how to control and prevent them (e.g. vaccination, quarantine,  
57 social distancing policies, etc. (22–24)), which together with the increasing availability of relevant data has

### **Significance Statement**

Infectious disease transmission in human populations crucially depends on contact patterns during outbreaks: *Who makes which contacts when?* An underappreciated element of contact behaviors is their interactive nature, and the cost-benefit analyses driving them. Endogenizing interactive cost-benefit analyses that factor in both infection risk and health status crucially changes predictions for the epidemic curve in ways that state-of-the-art epidemiological modeling does not capture. We look at data and find empirical evidence for health status dependent social distancing, as well as for other behaviors predicted by our theory. We run empirically informed simulations based on our model, and show that levels of curve flattening ought to be expected that match rather draconic lockdown policies, but they are endogenous and not imposed.

All authors contributed to all aspects of the paper, with M.U.G.K. and H.H.N. designing the study, and B.S.R.P. and H.H.N. modeling, M.G. and M.P. simulating, and S.F. working with Flusurvey.

There are no conflicting interests to declare.

<sup>2</sup>To whom correspondence should be addressed. E-mail: heinrich.nax@uzh.ch

58 allowed to apply some of these models to real-world epidemics.

59 One key modeling aspect concerns the transmission of infectious pathogens via individual contacts  
60 between infectious and susceptible individuals (25, 26), which have been shown to differ dependent on  
61 demographic factors such as age and sex (27–29). While a lot of previous work focuses on reconstructing  
62 the transmission trees of observed epidemics (30), or on their final size and geographic spread (31, 32), less  
63 attention has been paid to the role that individual decision-making regarding social distancing –weighing  
64 the risks of infecting and being infected– plays in shaping behavioral contact patterns that underlie these  
65 dynamics.

66 Mobility or, more generally, contact-seeking/avoiding decisions are key drivers of disease dynamics.  
67 Descriptive analyses of individual human mobility have revealed remarkable consistency at multiple  
68 temporal and spatial scales in the absence of exogenous factors (33–37), but also revealed that contact  
69 patterns change as a result of disease severity (38). One roadblock for making progress has been that contact  
70 and movement data are typically collected independently of health status.\* While this is currently changing  
71 with emerging health-tracking applications, there is no robust data that has been made available as of now.  
72 As a consequence, there is little empirical evidence on how human contact rates change depending on health  
73 status and as a function of disease incidence overall –often due to the lack of available real-time contact  
74 information (40).† Precisely this kind of insight, however, would be important to advance the understanding  
75 of the interactive nature of contact rate decisions, because the incentives to practice social distancing or not  
76 are different for healthy and for sick people. To improve predictions concerning the dynamics of diseases at  
77 the population level (43), and to understand what kinds of policies are actually appropriate, uncovering the  
78 behavioral determinants of contact patterns is therefore an important next step as applied work until now  
79 has to work largely by making crude assumptions, which may be sensitive, especially during the key (early)  
80 periods of an epidemic.

81 A rational-choice foundation of individual contact-seeking/avoiding behaviour in response to an infectious  
82 disease in epidemiological models is a framework proposed by (44). The result is a model where the contact  
83 rates of the resulting epidemiological model are no longer exogenous variables, but instead are determined  
84 co-evolutionarily with the dynamics of the disease itself. (44)’s framework presents an individual risk  
85 assessment, presuming that individuals’ propensities to stay home (more social distancing) increase with  
86 intensity and awareness of the disease due to the increased risks for contracting the disease. The simulations  
87 presented in (44) show that incorporating this type of individual decision-making changes predictions  
88 concerning the epidemic curve: (much) flatter curves are the result, particularly if sick individuals also  
89 reduce their contacts.

90 The decision-theoretic framework by (44) is a primer toward integrating human behavior into disease  
91 modeling, especially as regards understanding the role of infection fear in shaping contact patterns. To  
92 improve the behavioral micro-foundations of disease modeling, the aim of which is to better predict epidemic  
93 dynamics and to deliver more effective intervention policies, we generalize the existing decision-theoretic  
94 framework in two ways. First, the contact-reduction results are checked against some data on contact  
95 patterns during the 2012 and 2013 flu epidemics in the United Kingdom. Second, going beyond the  
96 single-player decision-theoretic approach, which does not account for the interactive nature of contact  
97 decisions, the underlying theoretical framework is extended to a game-theoretic model. By using game  
98 theory we can model not just the trajectory of the disease as a function of the underlying contact data,  
99 but more generally endogenize contact patterns by an interactive decision model and as determined by  
100 the dynamics of the disease (e.g., incidence rates). The framework proposed improves the rational-choice  
101 foundations of epidemiological models towards an integrated co-evolutionary view on contact rates and  
102 disease dynamics, which may substantially advance its predictive potential.

103 The core argument of this paper is that the kinds of risk assessments underlying contact decisions are  
104 interactive, which we model using game theory by formulating what we shall call “the social distancing

\* See (39) for one of the first studies to do so via a telephone survey conducted during an influenza season.

† Some related empirical work has been done to capture the change in human movements in response to environmental disaster (41), and travel restrictions (42), but not in response to disease outbreaks except for some recent work we shall discuss separately in the concluding remarks.

105 game<sup>†</sup>. The model permits us to produce testable individual-level comparative statics regarding how  
106 individuals will react during the outbreak of a disease and in response to others' contact patterns (checking  
107 against data from two influenza seasons in the UK for some empirics). Looking forward, the advantage  
108 of our game-theoretic modeling approach is that it becomes feasible to identify tipping points in the  
109 underlying dynamics,<sup>‡</sup> whose transitions may be explosive and differ fundamentally for marginally different  
110 starting conditions as compared to those predicted by a non-game-theoretic model (47). The next level of  
111 epidemic modeling should therefore consider game-theoretic modelling so as to leverage possible dynamics  
112 of equilibrium transitions to policy advantage, as is argued in policy making related to social dynamics  
113 (48, 49).

114 Indeed, there have been two very recent concurrent papers making progress in this direction, and future  
115 work could merge our lines of analyses with theirs. The first is by (50) who considers a theoretical model  
116 with endogenous contact rates where the two types of agents, sick (and infectious but not yet symptomatic)  
117 and healthy, who choose contact rates are in the same information set. Infected individuals stay at home  
118 with probability one. Their model generates the same contact rates for both types, and does not make  
119 predictions regarding interaction effects of the two. Our data indicates that health status leads to different  
120 contact patterns, and that symptomatic individuals also vary contact rates as a function of incidence. This  
121 is also an important feature of our simulations. A very nice feature of their model is an explicit treatment  
122 of the path dependency of equilibrium, which would be nice to extend to a framework like ours too in  
123 future work. The second paper is a related theoretical framework by (51) who do not consider endogenous  
124 contact reductions by infected individuals at all because they have no private benefit from it. In that sense  
125 their model is more similar to (44) than ours, but adds a Nash equilibrium analysis to it. Again, our data  
126 indicates that infected and infectious individuals do also reduce contact rates with incidence levels, and  
127 that there are interactions between contact rates of sick and healthy individuals. Pro-social concerns for  
128 the health of others, not just concerns for one's own health, clearly play a very important role.

129 In sum, the ambition of this paper is to integrate behavioral responses from a game-theoretic framework  
130 into classical epidemiological models that accounts for health status and includes self-protective and pro-  
131 social concerns. By doing so, we propose a new model, spell out its behavioral predictions, in particular  
132 regarding differential rates of social distancing. We compare theoretical results with empirical observations  
133 from the 2012 and 2013 influenza epidemic in the United Kingdom, and discuss implications for policy  
134 recommendations in light of the simulated epidemic curves our model generates. We compare the endogenous  
135 curve with curves that would result from interventions such as immobilizing fractions of the population.

## 136 Methods

137 **Contact rates.** The key ingredients, implicitly behavioral ones, that determine the dynamics of epidemiolog-  
138 ical models are so-called 'contact rates' which govern the frequency and likelihood of human interactions  
139 and therefore transmissions: *Where do you go? Who do you see? How do you make contact?* At the  
140 individual level, a change in contact rates may occur for symptom-specific medical reasons after contracting  
141 a disease that leads to reduced mobility for example. Moreover, a person, whether infected or not, may  
142 consciously decide to social distance, that is, to reduce contacts in light of various evolving risks (i.e. of  
143 spreading the disease and/or of contracting the disease) during an outbreak.<sup>§</sup>

144 To understand the implications of these endogenous phenomena, we need a model for how and why  
145 behavioral change occurs during outbreaks of infectious diseases. To do so, we extend the existing  
146 decision-theoretic model of (44) to allow for interactive decisions and *strategic* considerations as the risks of  
147 contracting and transmitting a disease depend on one's own contact patterns as well as on everyone else's  
148 levels of social distancing. Therefore, we model the individual decision as dependent on others' decisions,  
149 and we identify the rational-choice predictions for these decisions. By combining the human perspective  
150 on decision-making including considerations of risks and interactions in this way –using game theory– we

<sup>†</sup> See, for example, (45, 46).

<sup>§</sup> We referred to social distancing as self-quarantine in earlier versions of the paper, but adopt this jargon in line with (44) as is becoming standard.

151 obtain new and testable predictions for how human contact patterns and mobility decisions interact.

152 To illustrate the interactive nature of the proposed problem, consider the following thought experiments  
153 at the two extremes of the logical spectrum. At one extreme, suppose that everyone (sick and healthy alike)  
154 stays home, i.e. has reduced their contacts to zero (extreme social distancing). In that case, of course, any  
155 given individual (think of Will Smith in "I Am Legend" to lighten the mood) can move freely without fear  
156 of infection (if healthy) or of infecting others (if sick). Thus, in game-theoretic language, this does not  
157 constitute a Nash equilibrium, because every individual prefers to deviate (from staying at home), given  
158 the decision of everyone else (to stay at home). At the other extreme, by contrast, when everyone (sick and  
159 healthy) is moving around all the time resulting in very high contact rates (no social distancing), it is safest  
160 to stay home in order to not become infected (if healthy) or not to infect others (if sick). Again, everyone  
161 moving freely around will not constitute an equilibrium.

162 **Social distancing in a population.** In this section, we propose a formal model that will highlight the main  
163 advantages of choosing a game-theoretic rather than mechanistic approaches (as is done in applied work),  
164 and spell out how it goes beyond a single-player decision-theoretic model.

165 *Population.* Consider a human population  $N = \{1, 2, \dots, n\}$ . Each person  $i \in N$  either belongs to the set  
166  $H \subset N$ , the *healthy* (or non-symptomatic, susceptible, uninfected, etc.), or to the other set  $S = N \setminus H$ , the  
167 *sick* (or symptomatic, non-susceptible, infected, etc.).<sup>¶</sup>

168 *Social-distancing decisions.* Each  $i \in N$  chooses a contact rate  $\beta_i \in [0, 1]$ . Write  $\beta$  for the full vector of  
169 contact rates,  $\beta_H$  for the average contact rate of healthy agents, and  $\beta_S$  for the average contact rate of sick  
170 agents.

171 *Utilities.* Individual utility is generated by reaching places (or people) which is facilitated by being  
172 mobile. Hence, positive mobility is required to generate utility. But increased levels of mobility are also  
173 increasingly costly as they increase the exposure to infection risks for self and others. Hence, both complete  
174 immobility and full mobility generate no utility. Once there are risks of infection due to the presence of a  
175 disease, this mobility will be reduced to mitigate these risks.

176 Let us consider two scenarios distinguished by whether *i.*) everyone is healthy, or *ii.*) there are infected  
177 individuals.

178 *i.) No-disease scenario.* Suppose there is no disease, that is,  $|S| = 0$ . In that case, we assume utility for  
179 any player  $i$  is described by a twice-differentiable, continuous utility function

180 **Base utility.**  $u_i(\beta) = u(\beta_i)$  [1]

181 such that  $u(0) = u(1) = 0$ ,  $u(\beta) > 0$  for all  $\beta \in (0, 1)$ ,  $u'(\beta) > 0$  ( $< 0$ ) for  $\beta < \beta^*$  ( $> \beta^*$ ) given some  
182  $\beta^* \in (0, 1)$ , and  $u''(\beta) < 0$ . These assumptions ensure that  $\beta^*$  represents the unique utility-maximizing  
183 level of mobility in the no-disease scenario. We shall refer to levels chosen below  $\beta^*$  as ‘social distancing’.  
184 Of course, the optimal level will be heterogeneous within a population, but we abstract from this level of  
185 detail for the moment.<sup>||</sup>

186 *ii.) Disease scenario.* Once some individuals are infected, that is, if  $|S| > 0$ , then the ‘base utility’  $u(\beta_i)$   
187 that corresponds to a healthy individual, for an infected individual, is reduced directly by some disease  
188 factor  $\delta$  (with  $\delta \in [0, 1]$ ) representing a proportional disutility from being sick) resulting in ‘sick utility’  
189  $\delta u(\beta_i)$ . Moreover, depending on health status, all individuals suffer additional disutility from the risk of  
190 becoming infected (for healthy), or from the risk of infecting others (for sick), both of which increase with  
191 mobility, thus adding further costs to being mobile. Hence, for a **healthy individual**,  $i \in H$  the utility is

192 **Healthy H-utility.**  $u_i(\beta) =$  [2]  
193  $(1 - f \cdot \underbrace{(1 - [(1 - \beta_S^i)^{n-|H|} \cdot \beta_i + 1 - \beta_i])}_{\text{infection risk}}) \cdot \underbrace{u(\beta_i)}_{\text{base utility}}$

<sup>¶</sup> Here, we work with a basic epidemic model setting without recovery in mind, which naturally ought to be generalized in future work.

<sup>||</sup> Recent empirical work by (36, 49) identifies heterogeneous levels of mobility in the absence of a disease.



194 where  $f \in [0, 1]$  measures the fear of a healthy individual of getting infected, which would also express how  
 195 severe the disease is. Similarly, for a **sick individual**,  $i \in S$ , the utility is

$$196 \quad \textbf{Sick } S\text{-utility.} \quad u_i(\beta) = \tag{3}$$

$$197 \quad (1 - c \cdot \underbrace{(1 - [(1 - \beta_H^i)^{|H|} \cdot \beta_i + 1 - \beta_i])}_{\text{spreading risk}}) \cdot \underbrace{\delta u(\beta_i)}_{\text{sick utility}}$$

198 where  $c \in [0, 1]$  measures the pro-social concern an infected individual has for another individual's life,  
 199 that is, the expected reduction in utility from exposing other healthy humans to the risk of infection,  
 200 which would naturally increase with the severity of the disease too. Note that the introduction of this  
 201 parameter expressing this type of motivation, which is central to most policies aimed at reducing mobility of  
 202 symptomatic humans, is absent in (44), but will generate the kinds of mobility reductions that characterize  
 203 several of his simulations resulting in the flattest epidemic curves.

204 The underlying contact scenario we thus express is one where  $\beta_i$  represents agent  $i$ 's probability of  
 205 exposing him/herself to an infection-risk encounter, and  $1 - (1 - \beta_S^i)^{n-|H|}$  and  $1 - (1 - \beta_H^i)^{|H|}$  respectively represent  
 206 the probabilities of at least one infected / susceptible making the same encounter. Thus we model the  
 207 probability of two parties meeting at a given location, or all parties spending some time at a central  
 208 locations. W.l.o.g., when two individuals with different health status enter the location, we assume an  
 209 infection takes place with probability one.

210 **Results.** What interests us are the comparative statics of rational-choice contact rates in equilibrium when  
 211 mobility rates are chosen optimally so as to maximize subjective expected utilities. These we identify by  
 212 inspection of the conditions for optimal behavior for the two utility functions given by Equations 3 and 2,  
 213 which we obtain by maximizing both expressions with respect to  $\beta_i$ , yielding the two first-order conditions  
 214 (FOCs):

$$215 \quad \textbf{H-FOC.} \quad \overbrace{(1 - f \cdot (1 - [(1 - \beta_S^i)^{n-|H|} \cdot \beta_i + 1 - \beta_i]))}_{\text{marginal utility effect}} u'(\beta_i) \tag{4}$$

$$216 \quad = \underbrace{f \cdot (1 - (1 - \beta_S^i)^{n-|H|})}_{\text{marginal infection risk effect}} u(\beta_i)$$

$$217 \quad \textbf{S-FOC.} \quad \overbrace{(1 - c \cdot (1 - [(1 - \beta_H^i)^{|H|} \cdot \beta_i + 1 - \beta_i]))}_{\text{marginal utility effect}} u'(\beta_i) \tag{5}$$

$$218 \quad = \underbrace{c \cdot (1 - (1 - \beta_H^i)^{|H|})}_{\text{marginal spreading risk effect}} u(\beta_i)$$

219 Note that both right-hand sides of the latter equations are positive, indicating that both marginal utilities  
 220  $u'(\beta_i)$ s must also be positive; i.e. that we now must obtain lower contact rates for both sick and healthy  
 221 individuals (compared with the utility-maximizing level of mobility  $\beta^*$  from the no-disease scenario) in  
 222 order for FOCs to be satisfied than in the no-disease benchmark. This means that both sick and health  
 223 individuals will engage in some optimal level of 'social distancing', that is, choosing a lower equilibrium  
 224 utility than  $\beta^*$  from the no-disease scenario.

225 *Comparative statics.* From inspection of above two FOCs, we obtain the comparative statics summarized  
 226 in Table 1.\*\*

227  
 228 Naturally, the optimal contact rates for healthy and sick are different and take intermediate values, the  
 229 exact value depending on factors related to disease incidence, fear, concern, disease severity, risks, etc. Note

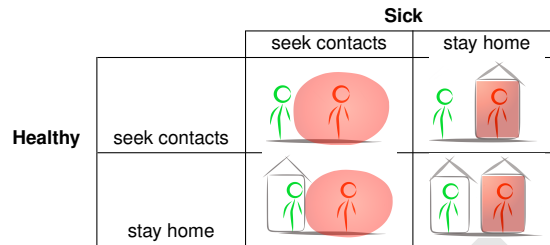
\*\* Comparative statics describe how the optimal contact rate varies with the various other parameters. Here, these are evaluated under the assumption that a symmetric Nash equilibrium exists such that, in equilibrium,  $\beta_i = \beta_H$  for all healthy and  $\beta_i = \beta_S$  for all sick.

**Table 1. Comparative statics of the equilibrium analysis.**

A marginal increase in ...	... leads to ...
social distancing of Healthy	less social distancing of Sick.
social distancing of Sick	less social distancing of Healthy. <sup>†</sup>
size of the Healthy population	more social distancing of Sick. <sup>†</sup>
size of the Sick population	more social distancing of Healthy. <sup>†</sup>
pro-social concern of the Sick	more social distancing of Sick.
fear of disease of the Healthy	more social distancing of Healthy.*

\*: Present in the model by (44). The other effects are new.

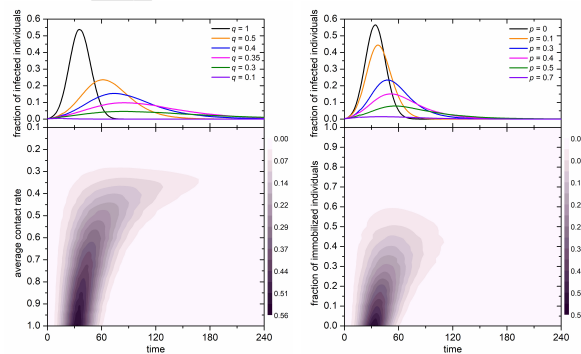
†: Contrary to imitation, herding, etc. as proposed, for example, in (10, 52).



**Fig. 1.** The social distancing interactions simplified.

230 that individuals may also differ in their fears, concerns, etc., hence we can think of the comparative statics  
 231 in Table 1 also as organizing individual heterogeneity. While this is a two-population evolutionary game  
 232 with continuous action space for every player, the strategic essence of this interaction can be represented  
 233 by a simplified game played between Sick and Healthy as is illustrated in Fig. 1. Both health types seek  
 234 contact leads to infection. Both staying home leads to no infection, but also generates zero utility for  
 235 anyone. The two mixed outcomes, where only one party stays home, also do not lead to infection, and have  
 236 the advantage that the population that continues to be mobile generates positive utilities.<sup>††</sup> As governments  
 237 aim to return to higher levels of economic and social activities, such an outcome, with the sick rather than  
 238 the healthy doing most of the staying at home, will likely become the goal.

239 **Concluding remarks**

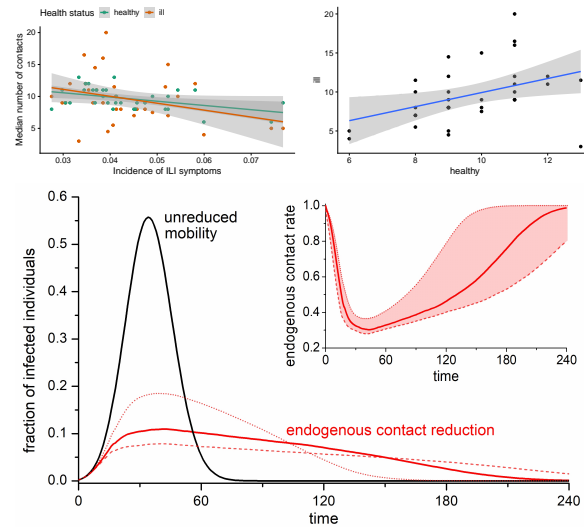


**Fig. 2.** Left: The color map encodes the fraction of infected individuals in dependence on time and the average contact rate. The upper panel shows characteristic cross-sections of the color map, where it can be observed that the endogenous contact reduction effect is matched no sooner than at 60-70% reduction of the contact rate (average mobility). Right: The color map encodes the fraction of infected individuals in dependence on time and the fraction of immobilized individuals. The upper panel shows characteristic cross-sections of the color map, where it can be observed that the endogenous contact reduction effect is matched no sooner than at 40-50% immobilization. Results were obtained by averaging outcomes over 1.5 million nodes in network configurations that are representative for real social networks. See Appendix C for details.

240 Here, we have developed a rational-choice framework for differential (health-dependent) levels of social

<sup>††</sup> Similarly, if the elderly are particularly at risk, either the young or the elderly, or both, should perhaps avoid contacts with one another to avoid infections.

241 distancing that includes interactive incentives related to risks of infection. A major issue regarding health-  
 242 dependent analyses of contact rates in general, and to test the kinds of predictions that our model generates  
 243 in particular, is data availability. To date, very little data is available that records contact rates and health  
 244 status at the same time. This is hopefully going to change as health-tracking applications are becoming  
 245 increasingly popular during the ongoing COVID-19 pandemic. Early work indicates that human contacts  
 246 have reduced markedly in China (53), and that contact rates are crucially important for disease transmission  
 247 as countries move to lift the lockdowns (16). However, many obstacles remain, especially considering the  
 248 adherence to recommended behaviours in different societies. We simulated different interpretations of social  
 249 distancing policies in Fig. 2, highlighting what kinds of epidemic curves ought to be expected from either  
 250 reducing mobility of everyone in the population or from immobilizing a certain fraction.



**Fig. 3.** Top left: Social distancing for sick and healthy. Median number of contacts in any different weeks as a function of incidence of ILI symptoms among Flusurvey participants in that particular week. The lines show linear fits, and shades 95% confidence intervals. Slopes: healthy -70 (95% CI: -120-(-20)), ill -110 (95% CI: -220, 10); p-value testing null hypothesis of slope 0: healthy 0.01, ill 0.07. Top right: No negative correlation between social distancing rates. Median number of contacts in participants with ILI symptoms as a function of the median number of contacts in participants without ILI symptoms. The lines shows a linear fit, and shades 95% confidence intervals. Slope: 0.9 (95% CI: 0.2-1.6); p-value testing null hypothesis of slope 0: 0.02. Bottom: Comparison of the infected curve, as obtained with unrestrained mobility (black) and endogenous contact reduction based on the Flusurvey data (black). The inset shows how fast the contact rate decreases as the fraction of infected individuals peaks, and then increases comparatively slowly as the incidence of infections decreases. Dashed and dotted lines were obtained with three-fold reductions at 5% and 15%, respectively, as the lower and upper bound on the error from the data (which suggest a three-fold decrease at about 10%).

251 As a step towards some empirical foundations, we considered Flusurvey data from the United Kingdom  
 252 (see Fig. 3, and Appendix for details), where we found evidence of social distancing amongst healthy  
 253 individuals as a function of disease incidences in their neighborhoods, as was predicted by our model and  
 254 by earlier work.<sup>‡‡</sup> The baseline levels of mobility in the UK as recorded per Flusurvey (resulting in medians  
 255 of circa 12 to 14 contacts per week outside the flu season) are in line with prior estimates from other  
 256 countries than the UK (39, 54). Indeed, we found that, even in the context of seasonal influenza, some  
 257 sizeable degree of social distancing took place amongst both sick (mobility reduction of ca. 50-55%) and  
 258 healthy individuals (mobility reduction of ca. 30-35%), fitting closely phenomena of endogenous social  
 259 distancing at the population level in other countries (55). Predicted negative correlations between the levels  
 260 of the two health types were rejected, suggesting presence of behavioral elements beyond individual utility  
 261 maximization such as social influence, norms, imitation, herding, etc.

262 The influenza comparison is useful, as seasonal influenza viruses tend to cause less mortal diseases than  
 263 SARS-COV-2, so any endogenous mobility reductions observed for an influenza according to our model  
 264 would provide lower bounds on the reductions that we would expect in the current situation (without policy)  
 265 of a more serious pandemic. In Sweden, for example, where the government decided against the kinds of  
 266 lockdowns that other European countries implemented, the aggregate population mobility in transit and

<sup>‡‡</sup>Note we pre-registered this type of analysis even though we did not know what kind of data would be available exactly at <https://osf.io/zc5b8> and <https://osf.io/q3m2p>.



267 workplace decreased by 31% and 11% respectively (as per Google’s COVID-19 Community Mobility Report  
268 Sweden). This is comparable to the decrease we recorded in the Flusurvey. Our simulations in **Fig. 3**  
269 which use the fit for contact reductions as observed in Flusurvey and extrapolate further reductions in  
270 case of incidence levels beyond those observed in Flusurvey indicate that with these kinds of endogenous  
271 mobility reductions (as are estimated from real-world behavior in **Fig. 3**) the epidemic curve would have  
272 flattened to levels that are comparable in terms of height of the peak and total case numbers as would have  
273 been obtained from immobilizing 40-50% of the total population or bringing the average mobility down by  
274 60-70%. These are candidate benchmarks we should be evaluating policy success against.

275 We are hopeful that future research and applied modeling will make use of game-theoretic modeling to  
276 endogenize contact rates in line with a modeling framework we proposed. Further, we encourage future  
277 efforts to test our model’s hypotheses with more data, as there are potential confounding factors in our  
278 data related to the seasonality of contacts because of factors unrelated to disease (especially temperature,  
279 but also school holidays etc.), which we cannot account for sufficiently due to data availability. Such  
280 analyses are important, as policymakers will likely move to new, perhaps health-status dependent, mobility  
281 restrictions and relaxations thereof. Ideally, to evaluate the effectiveness of policies aimed at increasing  
282 social distancing there ought to be at least some benchmarking concerning what levels might be expected  
283 endogenously during the pandemic, as well as monitoring of individual behaviours in response to changes  
284 in government recommendations or restrictions. In particular, the UK COVID-19 lockdown is currently  
285 estimated to reduce contacts by 75% (56), which is roughly double the reduction we recorded for healthy  
286 individuals for the 2012 and 2013 influenza seasons (see **Fig. 3**), but not substantially above the levels our  
287 simulations indicated would justify such policies (see **Fig. 2**). This work suggests scope for future studies  
288 in this directions and provides some first measurements.

289 Governments should factor in endogenous social distancing when weighing the pros and cons of policies  
290 as diverse as those ranging from China to Sweden. Epidemic modeling could improve its behavioral  
291 micro-foundations more generally.

## 292 **Materials and Methods**

293  
294 **Influenza season contact data.** Data for **Fig. 3** comes from the UK Flusurvey ([www.flusurvey.org.uk](http://www.flusurvey.org.uk)), an internet  
295 platform launched in 2009 to augment existing influenza surveillance (57, 58). The data underlying our analyses is  
296 available upon request from S.F.. Its focus is on recording healthcare usage by individuals with influenza-like-illness  
297 (ILI) symptoms (59, 60). During an influenza season, participants receive a weekly reminder to report presence or  
298 absence of ILI-related symptoms. When reported, followup questions are asked regarding health-care seeking and  
299 other behaviors. Flusurvey data has previously been used to estimate incidence trends (61), to identify risk factors  
300 (62), to estimate the effectiveness of vaccination (63), and to quantify health-care seeking behavior (64).

301 During the four influenza seasons 2009–13, social contact data were also collected, some of which is analyzed here.  
302 Participants were asked to report conversational and physical contacts by age group in three types of setting (home,  
303 work/school and other), as previously used to model H1N1v influenza (65). Here, we use the total of conversational  
304 contacts reported as a proxy for overall contacts, and assessed whether the date at which the contacts were submitted  
305 were within the start and end dates of an episode of illness with ILI symptoms (one general symptom out of fever,  
306 tiredness, weakness and headache, and one respiratory symptom out of sore throat, cough and shortness of breath).  
307 The end date of an episode was considered to be a healthy date. We cleaned the data in the following ways. We  
308 removed bad symptom dates (end date before start date, dates after the date at which a response was submitted) in 85  
309 out of 8800 symptom reports. We further removed all participants with fewer than three symptom reports (whether  
310 reporting healthy or ill), and removed the first submitted survey report of every participant in order to remove any  
311 potential bias from participants signing up only because they were researching influenza-related information. Where  
312 the end date of an episode was not reported, the date of the report which stated that the illness had ended was taken  
313 as the end date of the episode. Incidence was calculated as number of episodes of illness with ILI symptoms starting  
314 in any particular week divided by the number participants submitting a report in that week.

315 Data presented here are based on results from the UK flusurvey ([www.flusurvey.org.uk](http://www.flusurvey.org.uk)), which was launched in  
316 2009 as a platform for an internet-based cohort to augment existing influenza surveillance (57, 58), most of which

317 depends on recording healthcare usage by symptomatic individuals (59, 60) and therefore misses individuals with  
 318 influenza-like-illness (ILI) who do not seek medical attention. During the influenza season, every participants receives  
 319 a weekly reminder via email, asking to report presence or absence of ILI-related symptoms. If such symptoms are  
 320 reported, a number of followup questions are asked regarding health-care seeking and other behaviour. See Figure 4  
 321 for the key questions of the Flusurvey relevant for this study.

322 As well as estimating incidence trends (61), flusurvey data have been used to identify risk factors to ILI (62), to  
 323 estimate the effectiveness of influenza vaccination (63) and to quantify health-care seeking behaviour (64). During  
 324 the four influenza seasons 2009–13, social contact data were collected in addition to the ILI-related data. Participants  
 325 were asked to report conversational and physical contacts by age group in three types of setting (home, work/school  
 326 and other). These data have previously been used to explain the spread of H1N1v influenza (65).

327 We used the total of conversational contacts reported as measure of overall contact, and assessed whether the date  
 328 at which the contacts were submitted were within the start end end date of an episode of illness with ILI symptoms  
 329 (one general symptom out of fever, tiredness, weakness and headache, and one respiratory symptom out of sore throat,  
 330 cough and shortness of breath). The end date of an episode was considered to be a healthy date.

331 We cleaned the data in the following ways: We removed bad symptom dates (end date before start date, dates after  
 332 the date at which a response was submitted) in 85 out of 8800 symptom reports. We further removed all participants  
 333 with fewer than three symptom reports (whether reporting healthy or ill), and removed the first submitted survey  
 334 report of every participant in order to remove any potential bias from participants signing up only because they were  
 335 researching influenza-related information. Where the end date of an episode was not reported, the date of the report  
 336 which stated that the illness had ended was taken as the end date of the episode.

337 Incidence was calculated as number of episodes of illness with ILI symptoms starting in any particular week divided  
 338 by the number participants submitting a report in that week.

339 **Simulation details.** We use random geometric graphs in hyperbolic spaces to generate networks that have heterogeneous  
 340 degree distributions, strong clustering, and short average path lengths, which are all inherent properties of real social  
 341 networks (66, 67). By increasing the curvature  $\zeta$  of the hyperbolic space, we move from networks having exponential  
 342 to networks having scale-free degree distributions, and from longer to shorter average path lengths, and from weaker to  
 343 stronger clustering. We thus cover the whole family of networks that are representative for real social networks (68).

344 On top of these networks, we consider the susceptible-exposed-infectious-recovered (SEIR) model (69, 70), as  
 345 recently declared suitable for describing the spreading of the COVID-19 disease (71). Initially, we select 0.2% of the  
 346 nodes uniformly at random and designate them as infected (I). The remaining 99.8% of the nodes are designated as  
 347 susceptible (S). Moreover, every node  $i$  is assigned a contact rate  $q_i$ , where  $q_i = 0$  means the node is not exposed at  
 348 all and thus has no way of becoming infected, while  $q_i = 1$  means the node is fully exposed to potentially become  
 349 infected by all the other nodes to which it is connected. We note that  $q_i$  can also be interpreted as social distancing  
 350 or mobility, such that  $q_i = 0$  means that node  $i$  is not traveling to any of the other nodes to which it is connected and  
 351 is thus fully isolated, while  $q_i = 0.5$  means there is only a 50% chance node  $i$  will travel to any of the other nodes to  
 352 which it is connected. We consider the model without social distancing, such that  $q_i = 1$  for all nodes, as well as  
 353 with uniform social distancing, such that we decrease  $q_i$  below one for all nodes. We also consider random social  
 354 distancing, such that a fraction  $p$  of nodes is selected at random and assigned  $q_i = 0.1$  instead of  $q_i = 1$ , and with  
 355 endogenous social distancing, where we fit the Flusurvey data to account for decreasing  $q_i$  as the fraction of infected  
 356 individuals  $\rho$  in the population increases. The function we use is  $q_i = 3^{(-10\rho)}$ , which yields a three-fold decrease in  $q_i$   
 357 at 10% of infected in the population.

358 We perform Monte Carlo simulations of this SEIR model (72), which corresponds to a random sequential update,  
 359 such that during a full Monte Carlo step (MCS) each node gets a chance once on average to become infected. Each  
 360 full MCS consist of repeating the following elementary step  $n$  times. Firstly, select a node  $i$  uniformly at random  
 361 from the whole network. Secondly, (i) If node  $i$  is in state  $S$ , choose one neighbor  $j$  uniformly at random and visit it  
 362 with probability  $q_i$ . If the neighbor is visited and is in state  $I$ , node  $i$  becomes infected with probability  $w = 0.7$ .  
 363 If, however, the neighbor  $j$  is in states  $S$  or  $R$  nothing happens. (ii) If node  $i$  is in state  $I$ , then verify if at least  
 364  $t_r = 15$  full MCS have passed since it became infected. If yes, node  $i$  becomes recovered ( $R$ ), and if no, node  $i$  remains  
 365 infected. (iii) If node  $i$  is in state  $R$ , nothing happens.

366 **Derivations of comparative statics.** We first rearrange H-FOC from Equation 2:

$$\begin{aligned}
 & (1-f \cdot (1-[(1-\beta_S^i)^{n-|H|} \cdot \beta_i + 1-\beta_i]))u'(\beta_i) & [6] \\
 & = & -f \cdot ((1-\beta_S^i)^{n-|H|}-1)u(\beta_i) \\
 \iff & (1+f \cdot (-1+[(1-\beta_S^i)^{n-|H|}-1] \cdot \beta_i + 1))u'(\beta_i) \\
 & = & -f \cdot ((1-\beta_S^i)^{n-|H|}-1)u(\beta_i)
 \end{aligned}$$

371 We are interested in the partial derivative of Equation 7 along  $d/df$ ,  $d/dc$ ,  $d/d\beta_S$ , and  $d/d\beta_H$ . As the calculations  
 372 for the former two and the latter two are very similar we only detail them for  $d/df$  and  $d/d\beta_S$ . The other predictions  
 373 also follow from similar arguments.

374 Partial derivative of Equation 7 along  $d/df$ :

$$\begin{aligned}
 & (1 + f \cdot (-1 + [(1 - \beta_S^i)^{n-|H|} - 1] \cdot \beta_i + 1)) \cdot u''(\beta_i) \cdot \beta'_i \\
 & + u'(\beta_i) \left[ -1 + [(1 - \beta_S^i)^{n-|H|} - 1] \cdot \beta_i + 1 \right] \\
 & - u'(\beta_i) f \cdot [(1 - \beta_S^i)^{n-|H|} - 1] \cdot \beta'_i \\
 & = -((1 - \beta_S^i)^{n-|H|} - 1) \cdot (f \cdot u'(\beta_i) \cdot \beta'_i + u(\beta_i)) \\
 \Leftrightarrow & \beta'_i \cdot \left[ \overbrace{(1 + f \cdot (-1 + [(1 - \beta_S^i)^{n-|H|} - 1] \cdot \beta_i + 1))}^{>0} \cdot \overbrace{u''(\beta_i)}^{<0} \right. \\
 & \left. - \overbrace{u'(\beta_i) \cdot f \cdot [(1 - \beta_S^i)^{n-|H|} - 1]}{=(*)} + \overbrace{u'(\beta_i) \cdot f \cdot ((1 - \beta_S^i)^{n-|H|} - 1)}{=(*)} \right] \\
 & = -u'(\beta_i) \left[ -1 + [(1 - \beta_S^i)^{n-|H|} - 1] \cdot \beta_i + 1 \right] \\
 & - ((1 - \beta_S^i)^{n-|H|} - 1) \cdot u(\beta_i) \\
 \Leftrightarrow & \beta'_i \cdot \left[ \overbrace{(1 + f \cdot (-1 + [(1 - \beta_S^i)^{n-|H|} - 1] \cdot \beta_i + 1))}^{<0} \cdot u''(\beta_i) \right] \\
 & = \overbrace{u'(\beta_i) \left[ -1 + [(1 - \beta_S^i)^{n-|H|} - 1] \cdot \beta_i + 1 \right]}^{>0} \\
 & + \overbrace{(1 - (1 - \beta_S^i)^{n-|H|}) \cdot u(\beta_i)}^{>0} \\
 \Rightarrow & \beta'_i < 0
 \end{aligned}$$

387 Partial derivative of Equation 7 along  $d/d\beta_S$ :

$$\begin{aligned}
 & (1 + f \cdot (-1 + [(1 - \beta_S^i)^{n-|H|} - 1] \cdot \beta_i + 1)) \cdot u''(\beta_i) \cdot \beta'_i \\
 & + u'(\beta_i) \cdot f \cdot \left[ ((1 - \beta_S^i)^{n-|H|} - 1) \beta'_i - \beta_i (1 - \beta_S^i)^{n-|H|-1} \right] \\
 & = -f \cdot ((1 - \beta_S^i)^{n-|H|} - 1) \cdot u'(\beta_i) \cdot \beta'_i \\
 & + f \cdot (1 - \beta_S^i)^{n-|H|-1} \cdot u(\beta_i) \\
 \Leftrightarrow & \beta'_i \cdot \left[ \overbrace{(1 + f \cdot (-1 + [(1 - \beta_S^i)^{n-|H|} - 1] \cdot \beta_i + 1))}^{<0} \cdot u''(\beta_i) \right. \\
 & + \overbrace{u'(\beta_i) \cdot f \cdot ((1 - \beta_S^i)^{n-|H|} - 1)}^{<0} \\
 & \left. + \overbrace{u'(\beta_i) \cdot f \cdot ((1 - \beta_S^i)^{n-|H|} - 1)}^{<0} \right] \\
 & = \overbrace{u'(\beta_i) \cdot f \cdot \beta_i (1 - \beta_S^i)^{n-|H|-1}}^{>0} \\
 & + \overbrace{f \cdot (1 - \beta_S^i)^{n-|H|-1} \cdot u(\beta_i)}^{>0} \\
 \Rightarrow & \beta'_i < 0
 \end{aligned}$$

400 **Supporting Information Appendix (SI).** The data underlying the analyses of this article is available upon  
 401 request from S.F..

402 **ACKNOWLEDGMENTS.** We are thankful for comments from several colleagues, in particular from Petra Klepac  
 403 and Bernhard von Stengel. S.F. was funded by a Wellcome Trust Senior Research Fellowship (210758/Z/18/Z).  
 404 M.G. acknowledges funding from the Slovenian Research Agency (Grant no. P3-0396). We thank the participants



Breakfast    Travel    Work    Lunch    Work    Travel

Flu is spread via social contacts. Measuring how we meet each other helps understand and predict flu epidemics. Think about the all people you met yesterday and where you met them.

**NOTE: If you are filling this in on behalf of someone else, please answer all the questions as if you are that person**

How many people did you have conversational contact with yesterday (talking face to face)? (i)

	0-4 years	5-18 years	19-44 years	45-64 years	65+ years
Home	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>
Work	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>
Other	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>

How many people did you have physical contact with yesterday (skin-to-skin contact, e.g. handshake, kiss)? (i)

	0-4 years	5-18 years	19-44 years	45-64 years	65+ years
Home	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>
Work	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>
Other	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>	<input type="text" value="0"/>

How much time did you spend on public transport (e.g. bus, train, underground) yesterday? (i)

No time at all  
 0-30 minutes  
 30 minutes - 1.5 hours  
 1.5 hours - 4 hours  
 Over 4 hours

How long did you spend in an enclosed indoor space (e.g. office, classroom, bar, cinema) with more than 10 other people yesterday? (Not including public transport) (i)

No time at all  
 0-30 minutes  
 30 minutes - 1.5 hours  
 1.5 hours - 4 hours  
 Over 4 hours

What was the furthest distance from home that you travelled yesterday? (i)

Under 1 mile  
 1-4 miles  
 5-9 miles  
 10-29 miles  
 30-100 miles  
 Over 100 miles

Fig. 4. Key questions from the flu survey: the contact survey as seen by flusurvey participants.

of Flusurvey and the Influenzanet consortium who have enabled this study to be performed. Influenzanet were supported by the EU FP7 Epiwork project (grant number 231807) and Flusurvey received additional support from the i-sense (EPSRC IRC in Early Warning Sensing Systems for Infectious Diseases) Exploratory Project. M.U.G.K. acknowledges funding from the Branco Weiss Fellowship, Oxford Martin School and European Horizon 2020 Programme project MOOD. H.H.N. acknowledges an SNF Eccellenza Grant from the Swiss National Science Foundation. M.P. acknowledges funding from the Slovenian Research Agency (Grant nos. J4-9302, J1-9112, and P1-0403).

1. MU Kraemer, et al., The effect of human mobility and control measures on the COVID-19 epidemic in China. *Science* (2020).
2. S Funk, M Salathé, VA Jansen, Modelling the influence of human behaviour on the spread of infectious diseases: a review. *J. Royal Soc. Interface* **7**, 1247–1256 (2010).
3. P Manfredi, A D'Onofrio, *Modeling the interplay between human behavior and the spread of infectious diseases*. (Springer Science & Business Media), (2013).
4. F Verelst, L Willem, P Beutels, Behavioural change models for infectious disease transmission: a systematic review (2010–2015). *J. The Royal Soc. Interface* **13**, 20160820 (2016).
5. CT Bauch, AP Galvani, DJ Earn, Group interest versus self-interest in smallpox vaccination policy. *Proc. Natl. Acad. Sci.* **100**, 10564–10567 (2003).
6. CT Bauch, DJ Earn, Vaccination and the theory of games. *Proc. Natl. Acad. Sci.* **101**, 13391–13394 (2004).
7. AP Galvani, TC Reluga, GB Chapman, Long-standing influenza vaccination policy is in accord with individual self-interest but not with the utilitarian optimum. *Proc. Natl. Acad. Sci.* **104**, 5692–5697 (2007).
8. M van Boven, D Klinckenberg, I Pen, FJ Weissing, H Heesterbeek, Self-interest versus group-interest in antiviral control. *PLoS One* **3** (2008).
9. TC Reluga, An sis epidemiology game with two subpopulations. *J. Biol. Dyn.* **3**, 515–531 (2009).
10. P Poletti, B Caprile, M Ajelli, A Pugliese, S Merler, Spontaneous behavioural changes in response to epidemics. *J. Theor. Biol.* **260**, 31–40 (2009).
11. TC Reluga, Game theory of social distancing in response to an epidemic. *PLoS Comput. Biol.* **6** (2010).
12. D Adam, Special report: The simulations driving the world's response to COVID-19. *Nature* (2020).
13. N Ferguson, et al., Report 9: Impact of non-pharmaceutical interventions (NPIs) to reduce COVID19 mortality and healthcare demand. *Work. Pap.* (2020).
14. S Zhao, CT Bauch, D He, Strategic decision making about travel during disease outbreaks: a game theoretical approach. *J. The Royal Soc. Interface* **15**, 20180515 (2018).
15. S Flaxman, S Mishra, A Gandy, et al., Estimating the number of infections and the impact of non-pharmaceutical interventions on COVID-19 in 11 European countries. *Imp. Coll. preprint* (2020).
16. L Ferretti, et al., Quantifying SARS-CoV-2 transmission suggests epidemic control with digital contact tracing. *Science* (2020).
17. R Ross, An application of the theory of probabilities to the study of a priori pathometry. Part I. *Proc. Royal Soc. A* **9211184**, 204–230 (1916).
18. R Ross, HP Hudson, An application of the theory of probabilities to the study of a priori pathometry. Part III. *Math. Phys. Character* **93**, 225–240 (1917).
19. R Ross, HP Hudson, An application of the theory of probabilities to the study of a priori pathometry. Part II. *Math. Phys. Character* **93**, 212–225 (1917).
20. WO Kermack, AG McKendrick, Mathematical theory of epidemics. Part II. The problem of endemicity. *Proc. Royal Soc. A* **115**, 55–73 (1927).
21. WO Kermack, AG McKendrick, A contribution to the mathematical theory of epidemics. *Proc. Royal Soc. A* **115**, 700–721 (1927).
22. RM Anderson, R May, *Infectious diseases of humans: dynamics and control*. (Oxford University Press, Oxford, U.K.), (1991).
23. H Heesterbeek, et al., Modeling infectious disease dynamics in the complex landscape of global health. *Science* **347** (2015).
24. M Litvinova, QH Liu, ES Kulikov, M Ajelli, Reactive school closure weakens the network of social interactions and reduces the spread of influenza. *Proc. Natl. Acad. Sci.* (2019).
25. S Cauchemez, et al., Household transmission of 2009 pandemic influenza A (H1N1) virus in the United States. *New Engl. J. Medicine* **361**, 2619–27 (2009).
26. JM Read, WJ Edmunds, S Riley, J Lessler, DA Cummings, Close encounters of the infectious kind: methods to measure social mixing behaviour. *Epidemiol. Infect.* **140**, 2117–2130 (2012).
27. J Wallinga, P Teunis, M Kretzschmar, Using data on social contacts to estimate age-specific transmission parameters for respiratory-spread infectious agents. *Am. J. Epidemiol.* **164**, 936–44 (2006).
28. J Mossong, et al., Social contacts and mixing patterns relevant to the spread of infectious diseases. *PLoS Medicine* **5**, e74 (2008).
29. S Cauchemez, et al., Role of social networks in shaping disease transmission during a community outbreak of 2009 H1N1 pandemic influenza. *Proc. Natl. Acad. Sci.* **108**, 2825–30 (2011).
30. S Cauchemez, F Carrat, C Viboud, AJ Valleron, PY Boëlle, A Bayesian MCMC approach to study transmission of influenza: Application to household longitudinal data. *Stat. Medicine* **23**, 3469–3487 (2004).
31. C Viboud, et al., Synchrony, waves, and spatial hierarchies in the spread of influenza. *Science* **312**, 447–51 (2006).
32. MUG Kraemer, et al., Spread of yellow fever virus outbreak in Angola and the Democratic Republic of the Congo 2015–16: a modelling study. *The Lancet Infect. Dis.* **17**, 330–338 (2017).
33. MC González, Ca Hidalgo, AL Barabási, Understanding individual human mobility patterns. *Nature* **453**, 779–82 (2008).
34. J Candia, et al., Uncovering individual and collective human dynamics from mobile phone records. *J. Phys. A: Math. Theor.* **41**, 224015 (2008).
35. F Simini, MC González, A Maritan, AL Barabási, A universal model for mobility and migration patterns. *Nature* **484**, 96–100 (2012).
36. L Alessandretti, P Sapiezynski, V Sekara, S Lehmann, A Baronchelli, Evidence for a conserved quantity in human mobility. *Nat. Hum. Behav.* **2**, 485 (2018).
37. DL Luh, ZS You, SC Chen, Comparison of the social contact patterns among school-age children in specific seasons, locations, and times. *Epidemics* **14**, 36–44 (2016).
38. KV Kerckhove, N Hens, WJ Edmunds, KTD Eames, The impact of illness on social networks: Implications for transmission and control of influenza. *Am. J. Epidemiol.* **178**, 1655–1662 (2013).
39. F DeStefano, et al., Factors associated with social contacts in four communities during the 2007–2008 influenza season. *Epidemiol. & Infect.* **139**, 1181–1190 (2011).
40. M Kraemer, et al., Progress and challenges in infectious disease cartography. *Trends Parasitol.* **32**, 19–29 (2016).
41. X Lu, L Bengtsson, P Holme, Predictability of population displacement after the 2010 Haiti earthquake. *Proc. Natl. Acad. Sci.* **109**, 11576–11581 (2012).
42. CM Peak, et al., Population mobility reductions associated with travel restrictions during the Ebola epidemic in Sierra Leone: use of mobile phone data. *Int. J. Epidemiol.*, 1–9 (2018).
43. EHY Lam, et al., The feasibility of age-specific travel restrictions during influenza pandemics. *Theor. Biol. Med. Model.* **8**, 1–14 (2011).
44. EP Fenichel, et al., Adaptive human behavior in epidemiological models. *Proc. Natl. Acad. Sci.* **108**, 6306–6311 (2011).
45. D Centola, J Becker, D Brackbill, A Baronchelli, Experimental evidence for tipping points in social convention. *Science* **360**, 1116–1119 (2018).
46. D Centola, A Baronchelli, The spontaneous emergence of conventions: An experimental study of cultural evolution. *Proc. Natl. Acad. Sci.* **112**, 1989–1994 (2015).
47. HP Young, The dynamics of social innovation. *Proc. Natl. Acad. Sci.* **108**, 21285–21291 (2011).
48. K Nyborg, et al., Social norms as solutions. *Science* **354**, 42–43 (2016).
49. H Hu, K Nigmatulina, P Eckhoff, The scaling of contact rates with population density for the infectious disease models. *Math. Biosci.* **244**, 125 – 134 (2013).
50. D McAdams, Nash SIR: An Economic-Epidemiological Model of Strategic Behavior During a Viral Epidemic. *Covid Econ. CEPR (forthcoming)* (2020).
51. F Toxvaerd, Equilibrium Social Distancing. *Covid Econ. CEPR 15* (2020).
52. P Poletti, M Ajelli, S Merler, Risk perception and effectiveness of uncoordinated behavioral responses in an emerging epidemic. *Math. Biosci.* **238**, 80–89 (2012).
53. J Zhang, et al., Age profile of susceptibility, mixing, and social distancing shape the dynamics of the novel coronavirus disease 2019 outbreak in China. *medRxiv* (2020).
54. M Kretzschmar, RT Mikolajczyk, Contact profiles in eight European countries and implications for modelling the spread of airborne infectious diseases. *PLoS one* **4** (2009).
55. P Poletti, M Ajelli, S Merler, The effect of risk perception on the 2009 H1N1 pandemic influenza dynamics. *PLoS one* **6** (2011).
56. CI Jarvis, et al., Quantifying the impact of physical distance measures on the transmission of covid-19 in the uk. *medRxiv* (2020).
57. I Friesema, et al., Internet-based monitoring of influenza-like illness in the general population: experience of five influenza seasons in The Netherlands. *Vaccine* **27**, 6353–6357 (2009).
58. NL Tilston, KT Eames, D Paolotti, T Ealden, WJ Edmunds, Internet-based surveillance of influenza-like-illness in the UK during the 2009 H1N1 influenza pandemic. *BMC Public Heal.* **10**, 650 (2010).
59. D Fleming, A Elliot, Lessons from 40 years' surveillance of influenza in England and Wales. *Epidemiol. & Infect.* **136**, 866–875 (2008).
60. AJ Elliot, et al., Monitoring the emergence of community transmission of influenza A/H1N1 2009 in England: a cross sectional opportunistic survey of self sampled telephone callers to NHS Direct. *BMJ* **339**, b3403 (2009).
61. E Brooks-Pollock, N Tilston, WJ Edmunds, KT Eames, Using an online survey of healthcare-seeking behaviour to estimate the magnitude and severity of the 2009 H1N1v influenza epidemic in England. *BMC Infect. Dis.* **11**, 68 (2011).
62. AJ Adler, KT Eames, S Funk, WJ Edmunds, Incidence and risk factors for influenza-like-illness in the UK: online surveillance using Flusurvey. *BMC Infect. Dis.* **14**, 232 (2014).
63. K Eames, et al., Rapid assessment of influenza vaccine effectiveness: analysis of an internet-based cohort. *Epidemiol. & Infect.* **140**, 1309–1315 (2012).
64. M Peppia, WJ Edmunds, S Funk, Disease severity determines health-seeking behaviour amongst individuals with influenza-like illness in an internet-based cohort. *BMC Infect. Dis.* **17**, 238 (2017).
65. K Eames, NL Tilston, E Brooks-Pollock, WJ Edmunds, Measured dynamic social contact patterns explain the spread of H1N1v influenza. *PLoS Comput. Biol.* **8**, 1–8 (2012).
66. M Boguná, F Papadopoulos, D Krioukov, Sustaining the internet with hyperbolic mapping. *Nat. Commun.* **1**, 62 (2010).
67. D Krioukov, F Papadopoulos, M Kitsak, A Vahdat, M Boguná, Hyperbolic geometry of complex networks. *Phys. Rev. E* **82**, 036106 (2010).
68. K Zuev, M Boguná, G Bianconi, D Krioukov, Emergence of soft communities from geometric preferential attachment. *Sci. Rep.* **5**, 9421 (2015).



- 485 69. R Pastor-Satorras, C Castellano, P Van Mieghem, A Vespignani, Epidemic processes in complex networks. *Rev. Mod. Phys.* **87**, 925 (2015).  
486 70. Z Wang, et al., Statistical physics of vaccination. *Phys. Reports* **664**, 1–113 (2016).  
487 71. K Prem, et al., The effect of control strategies to reduce social mixing on outcomes of the COVID-19 epidemic in Wuhan, China: a modelling study. *Lancet Public Heal.* (2020).  
488 72. MEJ Newman, GT Barkema, *Monte Carlo Methods in Statistical Physics*. (Oxford University Press, Oxford, U.K.), (1999).

DRAFT