



Social deprivation and burden of influenza: Testing hypotheses and gaining insights from a simulation model for the spread of influenza



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ABSTRACT

Factors associated with the burden of influenza among vulnerable populations have mainly been identified using statistical methodologies. Complex simulation models provide mechanistic explanations, in terms of spatial heterogeneity and contact rates, while controlling other factors and may be used to better understand statistical patterns and, ultimately, design optimal population-level interventions. We extended a sophisticated simulation model, which was applied to forecast epidemics and validated for predictive ability, to identify mechanisms for the empirical relationship between social deprivation and the burden of influenza. Our modeled scenarios and associated epidemic metrics systematically assessed whether neighborhood composition and/or spatial arrangement could qualitatively replicate this empirical relationship. We further used the model to determine consequences of local-scale heterogeneities on larger scale disease spread. Our findings indicated that both neighborhood composition and spatial arrangement were critical to qualitatively match the empirical relationship of interest. Also, when social deprivation was fully included in the model, we observed lower age-based attack rates and greater delay in epidemic peak week in the most socially deprived neighborhoods. Insights from simulation models complement current understandings from statistical-based association studies. Additional insights from our study are: (1) heterogeneous spatial arrangement of neighborhoods is a necessary condition for simulating observed disparities in the burden of influenza and (2) unmeasured factors may lead to a better quantitative match between simulated and observed rate ratio in the burden of influenza between the most and least socially deprived populations.

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1. Introduction

Seasonal influenza epidemics pose several challenges for society in terms of social, economic and health impacts (Molinari et al., 2007). These impacts are not always proportional among different populations. Lower vaccination coverage may increase susceptibility to infection in some racial/ethnic groups (Lu et al., 2013). Heterogeneous contact patterns due to social and demographic factors may affect disease transmission between populations (Charland et al., 2011; Laskowski et al., 2011; Mossong et al., 2008). Also, material/social deprivation, neighborhood socioeconomic status, distrust of authority, and access to health care services may be driving disparities in the burden of influenza

(Charland et al., 2011; Crighton et al., 2007; Loeb, 2003; Principi et al., 2003).

Social determinants of health, such as social deprivation, are inherently constructs of individual- and neighborhood-level factors. In Charland et al. (2011), social deprivation, which represented social support, cohesion and cooperation at the neighborhood scale, was negatively associated with the burden of influenza. Specifically, social deprivation reflected family-type composition within households (e.g., living alone and single-parent families). Household characteristics, such as size, structure, and presence of school-aged children, affect disease transmission and severity via differences in susceptibility and contact patterns (Cauchemez et al., 2009; House and Keeling, 2009; Longini et al., 1982; Marathe et al., 2011; Stroud et al., 2007). Despite these past findings, the combined role of family-type composition and spatial heterogeneity of neighborhoods with dissimilar distribution of family-type households has not been studied as a possible explanation for the relationship between social deprivation and burden of influenza, as reported in Charland et al. (2011).

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While current statistical models have identified interesting patterns between social deprivation and the burden of influenza (Charland et al., 2011), complementary methodologies, which explicitly consider the outcome of putative mechanisms, may be useful to explore (Auchincloss and Diez Roux, 2008). Particularly, individual-based models (IBMs), which are also called agent-based models, offer a unique way to model real-world processes and have extensively been used to model the spatial spread of influenza (degli Atti et al., 2008; Eubank et al., 2004; Ferguson et al., 2005; Germann et al., 2006). In an IBM, interactions are modeled between agents (e.g., people), their characteristics (e.g., age, pre-existing immunity, place membership at school or work) and processes (e.g., infectiousness over time, place-based contact probabilities, staying home from work/school). IBMs offer flexibility in modeling nonlinear, dynamic, and feedback processes over multiple spatial and temporal scales (Auchincloss and Diez Roux, 2008; Galea et al., 2010; Mabry et al., 2008). Importantly, these IBMs will allow us to ask whether we can understand the observed effects of social deprivation mechanistically in terms of spatial heterogeneity, contact rates, etc. Conversely, we can examine whether the rich theoretical developments on IBMs of the spread of influenza is consistent with and sufficient to explain observed patterns of social deprivation and burden of influenza. Ultimately, the IBM is a virtual platform that leverages detailed information on individual-, household- and neighborhood-level factors to design, test and predict the impact of novel mitigation strategies.

With this in mind, we propose to use and extend a sophisticated simulation model, which has been developed and applied to forecast epidemics as well as validated for predictive ability (Hyder et al., 2013), to examine mechanistically the observed relationship between social deprivation and the burden of influenza. We hypothesize that the influence of social deprivation on the burden of influenza is mediated through the distribution of household size and contact patterns. In the context of social deprivation, our conceptualization of household size includes data on family type or structure, such as living alone, as a couple with no kids or parent(s) with one or more child. Due to spatial heterogeneity in social deprivation, we also hypothesize that the spatial arrangement of households of different size (due to family type) may affect epidemic dynamics at larger (city) and local (neighborhood) spatial scales.

2. Methods

2.1. Simulation model

Our model was a spatially-explicit stochastic representation of influenza epidemics in the Census Metropolitan Area of Montreal (CMA). We used census data to recreate key demographic and contact patterns, such as age and household size distribution, place membership (e.g. number of employees in a workplace and number of students in each grade level), age-based contact within households, schools and workplaces and random contact in the community. Disease natural history parameters, such as latent period, infectiousness profile and recovery, were modeled based on probabilistic functions using literature-based parameters. Disease transmission was modeled by calculating a force of infection due to infectious contact with infected individuals from three sources: household, place (school or workplace) and community. We calibrated transmission coefficients for each of these three sources using observed data on the: (i) laboratory surveillance data and (ii) age-based clinical attack rates. Details on model formulation, mathematical equations, and data sources schools and workplaces are found in the Supplementary material while details about households are given below. Further details of model fitting,

calibration and validation may be found elsewhere (Hyder et al., 2013).

We used the Public Use Microdata File (Households) from Statistics Canada (Statistics Canada, 2005) to model household characteristics (e.g., size, family-type) and the age distribution of individuals within households. We considered four family types: two parents with children, one parent with children, couples without kids, and individuals living alone. Note that these households may contain other individuals than just parents, couples and kids. Related to social deprivation, neighborhoods in our model were defined by census tract boundaries based on the 2001 Census conducted by Statistics Canada.

2.2. Model scenarios

We used this IBM to simulate epidemics under scenarios that differed in family-type composition and spatial arrangement. We hypothesized the interaction of these two mechanisms as the primary drivers of the observed relationship between social deprivation and burden of influenza. In our three scenarios (see below), family-type composition within neighborhoods was set to random or heterogeneous and spatial arrangement between neighborhoods' social deprivation level was set to random or "empirical" heterogeneous "Empirical" here implied that we used the observed distribution of social deprivation level in the study area (Fig. 1) (Institut National de Sante Publique du Quebec, 2001).

Scenario 1: random neighborhood composition and spatial location.

Scenario 2: heterogeneous neighborhood composition and random spatial arrangement of neighborhoods.

Scenario 3: heterogeneous neighborhood composition and "empirical" heterogeneous spatial arrangement of neighborhoods.

To model scenario 1, we randomly distributed households across neighborhoods while matching the observed number of individuals within each neighborhood. Thus, we controlled for neighborhood size, but composition and spatial arrangement were random. We labeled this the "Null model" since it did not consider any information of social deprivation, and was analogous to the general formulations of influenza IBMs.

To model scenario 2, we collected data on social deprivation index (SDI) (Institut National de Sante Publique du Quebec, 2001) and the proportion of different family types for each neighborhood. SDI values of 1 (lowest) to 5 (highest) represented levels of social deprivation. For neighborhoods with missing SDI values, we assigned average values from adjacent neighborhoods. In combination, these data provided a list of neighborhoods indicating their SDI value and the proportion of households of each family-type. Using this list, we randomly assigned each neighborhood a SDI value and the corresponding family-type distribution. We used the assigned family-type distribution to distribute households within neighborhoods. In this way, we removed any spatial heterogeneity in SDI values but retained the realistic (and heterogeneous) distribution of household size and family type according to the assigned SDI value. We labeled this the "Composition model" because it modeled the realistic family-type composition within neighborhoods.

In scenario 3, we used the observed SDI value for each neighborhood and then distributed households. This allowed us to match the observed and simulated data within each neighborhood in terms of their: (i) composition, as measured by the proportion of households of each family type, and (ii) spatial location, as measured by the spatial arrangement of social deprivation levels. We labeled this the "full SDI model".

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