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The effects of demographic change on disease transmission and vaccine impact in a household structured population

Nicholas Geard[a,b,*], Kathryn Glass[c], James M. McCaw[a,d], Emma S. McBryde[e,f], Kevin B. Korb[b], Matt J. Keeling[g], Jodie McVernon[a,d]

[a] Centre for Epidemiology and Biostatistics, Melbourne School of Population and Global Health, 207 Bouverie Street, The University of Melbourne, VIC 3010, Australia
[b] Clayton School of Information Technology, Building 63, Monash University Clayton Campus, Wellington Road, Clayton, VIC 3800, Australia
[c] National Centre for Epidemiology and Population Health, College of Medicine, Biology and Environment, Building 62, Cnr of Eggleston and Mills Roads, The Australian National University, Canberra, ACT 0200, Australia
[d] Murdoch Childrens Research Institute, Royal Children's Hospital, Flemington Road, Parkville, VIC 3052, Australia
[e] Department of Medicine, The University of Melbourne, 4th Floor, Clinical Sciences Building, The Royal Melbourne Hospital, Royal Parade, Parkville, VIC 3050, Australia
[f] Australian Institute of Tropical Health and Medicine, James Cook University, 1 James Cook Drive, Townsville, QLD 4811, Australia
[g] Biological Sciences and Mathematics Institute, University of Warwick, Gibbet Hill Rd, Coventry CV4 7AL, United Kingdom

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ABSTRACT

The demographic structure of populations in both more developed and less developed countries is changing: increases in life expectancy and declining fertility have led to older populations and smaller households. The implications of these demographic changes for the spread and control of infectious diseases are not fully understood. Here we use an individual based model with realistic and dynamic age and household structure to demonstrate the marked effect that demographic change has on disease transmission at the population and household level. The decline in fertility is associated with a decrease in disease incidence and an increase in the age of first infection, even in the absence of vaccination or other control measures. Although large households become rarer as fertility decreases, we show that there is a proportionate increase in incidence of disease in these households as the accumulation of susceptible clusters increases the potential for explosive outbreaks. By modelling vaccination, we provide a direct comparison of the relative importance of demographic change and vaccination on incidence of disease. We highlight the increased risks associated with unvaccinated households in a low fertility setting if vaccine behaviour is correlated with household membership. We suggest that models that do not account for future demographic change, and especially its effect on household structure, may potentially overestimate the impact of vaccination.

1. Introduction

The demographic structure of a population is a key determinant of patterns of contact and hence of infectious disease spread, with implications for the design of effective control measures. Households in particular are recognised as an important focus of disease transmission, due to the duration and intensity of contacts occurring within them (Hope-Simpson, 1970). Over time, demographic processes such as birth, death, aging, marriage and divorce modify age and household structure. During the 20th century, the populations of more developed countries experienced demographic changes—increases in life expectancy and decreases in fertility—that have led to older populations living in smaller households. Drivers of these demographic changes include improvements to public health, and social and economic transformation associated with the growth of urban industrial societies (Livi-Bacci, 1997). Similar trends are occurring, at differing rates, among less developed countries. Understanding how changes in the demographic structure of a population affect disease transmission is a necessary step towards the design of more effective strategies for disease control (John, 1990; Manfredi and Williams, 2004).

* Corresponding author at: Centre for Epidemiology and Biostatistics, Melbourne School of Population and Global Health, 207 Bouverie Street, The University of Melbourne, VIC 3010, Australia. Tel.: +61 390356216.
E-mail address: ngeard@unimelb.edu.au (N. Geard).

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Mathematical models can help improve our understanding of how infectious diseases spread and inform decision making about how they can be controlled (Anderson and May, 1992; Keeling and Rohani, 2007). To capture the full impact of changes in demography on disease spread, a model must represent age and household structure, as well as how these evolve over an extended period of time. Compartmental models of disease transmission that include either age *or* household structure are well established (Hethcote, 2000; Hall and Becker, 1996; House and Keeling, 2009). However, combining both age *and* household structure in a single model is challenging due to the combinatoric growth in the number of compartments required to capture variations in household composition and disease status. An assumption of many existing models is that population structure exhibits an age distribution that does not change over time (*i.e.*, it is demographically stable). While reasonable over short time frames, for example a single influenza season, this assumption is clearly unrealistic when considering the long term dynamics of an endemic disease, or the long term impacts of vaccination programs. Models that incorporate demographic processes have been proposed, but typically assume either stationary or exponentially growing populations (John, 1990), and only rarely include household structure (Glass et al., 2011). Models that do incorporate non-stationary age structure have demonstrated significant implications for both patterns of disease and the effectiveness of vaccine programs (Manfredi and Williams, 2004; Finkenstädt and Grenfell, 2000; Williams and Manfredi, 2004; Gao and Hethcote, 2006; Iannelli and Manfredi, 2007; Cummings et al., 2009; McDonald, 2012; Merler and Ajelli, 2014; Liu et al., 2014; Marziano et al., 2015).

An alternative approach is individual based models, which explicitly simulate each member of a population together with their demographic characteristics, social contacts and disease status. These models allow much greater flexibility in representing the heterogeneity present in real populations. They have been used for simulating outbreak scenarios in realistically structured (*i.e.*, containing both age and household structure) static and dynamic populations (Eubank et al., 2004; Ferguson et al., 2005; Ajelli and Merler, 2009; Guzzetta et al., 2011; Silhol and Boëlle, 2011). To date, these models do not explicitly capture the long-term impact of demographic changes to both age and household structure that underpin the contact patterns most relevant to disease transmission.

We have previously described a parsimonious individual based model of household structure and dynamics capable of simulating a range of non-stationary demographic scenarios (Geard et al., 2013). Here we use this model to show how demographic processes alter the age and household structure of a population, and the effects this has on patterns of contact, disease transmission and vaccine impact.

2. The model

We model a population of individual people characterised by their age, sex, and the household in which they currently reside. Over time, people are born, age, enter into and leave couples and households, and eventually die. The dynamics of these demographic processes are parameterised using age- and sex-specific mortality and fertility rates, and calibrated against observed patterns of household formation and dissolution (see Supplementary information for detail). By choosing appropriate rates, a variety of demographic scenarios can be simulated, including stable, exponentially growing, and non-stationary populations (Geard et al., 2013). Here we focus on a population moving from a high to a low fertility setting, using current and historical Australian census and survey data to calibrate our model. The key demographic trends included are an increase in life expectancy and a decrease in birth rate, together with social factors such as an increase in the average age of childbearing and an increase in the rate of couple separation.

This demographic model is overlaid with a Susceptible, Infectious, Removed disease transmission model, with contact and transmission simulated in the community and household settings. As our primary focus is the role of household transmission, we aggregate contacts occurring outside of the household—in locations such as schools, workplaces and public spaces—into a matrix of age-specific community contact rates. We assume these contact rates to be age-assortative; that is, people are more likely to come into contact with others of a similar age to themselves (Mossong et al., 2008). These contact rates are derived from the age structure of the population and empirically observed activity levels (Hethcote, 1996; Mossong et al., 2008) (see Supplementary information for detail). Within the community, we make the standard assumption for large populations that transmission is frequency dependent. As the age structure of the population evolves over time, we recalculate the community contact rates at five yearly intervals. Contacts occurring within households are determined directly by the structure of the model population. Here we assume tha all individuals within a household mix equally with one enother, irrespective of age. The degree to which household transmission is frequency or density dependent is not well-established—and most likely varies by disease (van Boven et al., 2010)—and can be varied within the model.

Thus, the probability of a susceptible person in age class i becoming infected in a given time step (here, 1 week) depends on the prevalence of disease in their household and in the broader community, and is given by $1 - e^{-\lambda_{i,N_H}}$, where the force of infection λ_{i,N_H} on an individual in age class i, in a household of size N_H is given by

$$\lambda_{i,N_H} = q_h \frac{I_H}{(N_H - 1)^{\alpha}} + q_c \sum_j \eta_{ij} \frac{I_j}{N_j} \tag{1}$$

where q_h and q_c are transmission coefficients for household and community transmission, I_H and N_H are, respectively, the number of infectious people and the total number of people in the susceptible person's household, α specifies the degree to which household transmission is frequency ($\alpha = 1$) or density ($\alpha = 0$) dependent, η_{ij} is the average number of community contacts between a person in age class i and people in age class j, and I_j and N_j are, respectively, the number of infectious people and the total number of people in age class j. In addition to endemic transmission, we also allowed for the importation of infection from sources external to the population. At each time step, a susceptible individuals could become infected from an external source with a small probability.

In this study, we parameterised the demography of our population model based on historical Australian census and survey data from 1910 to 2010 (Australian Bureau of Statistics, 2008, 2009, 2010a,b; de Vaus, 2004; Wilkins et al., 2011) (see Supplementary information for detail). As data were only available on the average size of households in the Australian population in 1910, initial household size distributions were estimated using a zero-truncated Poisson distribution (Jennings et al., 1999). The model is stochastic, and each scenario was simulated 10 times; unless otherwise noted, results reported represent means and standard deviations across each set of simulations. Starting populations for all simulations were created by running the model for 200 years, using the earliest available demographic rates, to reach an endemic disease equilibrium. Final population sizes in each simulation were approximately 225,000. Importation of cases from an external source (equivalent to 5×10^{-6} cases per person per week on average) was used to prevent epidemic fade-out due to stochasticity. The model is implemented in Python and source code is available from http://bitbucket.org/ngeard/simodd-pub.