RESEARCH ARTICLE

COVID-19 inequalities in England: a mathematical modelling study of transmission risk and clinical vulnerability by socioeconomic status

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Abstract

Background The COVID-19 pandemic resulted in major inequalities in infection and disease burden between areas of varying socioeconomic deprivation in many countries, including England. Areas of higher deprivation tend to have a different population structure—generally younger—which can increase viral transmission due to higher contact rates in school-going children and working-age adults. Higher deprivation is also associated with a higher presence of chronic comorbidities, which were convincingly demonstrated to be risk factors for severe COVID-19 disease. These two major factors need to be combined to better understand and quantify their relative importance in the observed COVID-19 inequalities.

Methods We used UK Census data on health status and demography stratified by decile of the Index of Multiple Deprivation (IMD), which is a measure of socioeconomic deprivation. We calculated epidemiological impact using an age-stratified COVID-19 transmission model, which incorporated different contact patterns and clinical health profiles by decile. To separate the contribution of each factor, we considered a scenario where the clinical health profile of all deciles was at the level of the least deprived. We also considered the effectiveness of school closures and vaccination of over 65-year-olds in each decile.

Results In the modelled epidemics in urban areas, the most deprived decile experienced 9% more infections, 13% more clinical cases, and a 97% larger peak clinical size than the least deprived; we found similar inequalities in rural areas. Twenty-one per cent of clinical cases and 16% of deaths in England observed under the model assumptions would not occur if all deciles experienced the clinical health profile of the least deprived decile. We found that more deaths were prevented in more affluent areas during school closures and vaccination rollouts.

Conclusions This study demonstrates that both clinical and demographic factors synergise to generate health inequalities in COVID-19, that improving the clinical health profile of populations would increase health equity, and that some interventions can increase health inequalities.

Keywords COVID-19, Mathematical model, Socioeconomic inequality, Comorbidities, England, Demography

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Goodfellow *et al. BMC Medicine*   (2024) 22:162

Page 2 of 13

Background

The COVID-19 pandemic disproportionately affected people in lower socioeconomic groups around the world [1, 2]. In England, there were large disparities in COVID-19 burden between areas of different relative deprivation, measured by the Index of Multiple Deprivation (IMD). Initial reports by the UK Office for National Statistics (ONS) found that from April to July 2020, the most deprived 10% of areas in England experienced an age-standardised COVID-19-related mortality rate more than twice as high as the least deprived 10% [3]. These disparities were repeatedly observed throughout the pandemic: between June 2020 and January 2021, the age-standardised mortality rate in laboratory-confirmed cases of COVID-19 was 371.0 per 100,000 (95% confidence interval (CI) 334.2–410.7) compared to 118.0 (95% CI 97.7–141.3) in the most vs least deprived quintiles [4]. The inequality in mortality rates seen in the early pandemic exceeded that observed in previous years, indicating that there were further factors exacerbating the 'expected' effects of relative deprivation [5]. Even after adjusting for age, sex, region, and ethnicity, this report found worse outcomes in more deprived areas but did not adjust for the prevalence of comorbidities. Other studies have consistently confirmed an association between comorbidities and more severe COVID-19 outcomes [6, 7].

Morbidity and the presence of underlying health conditions tend to vary greatly by socioeconomic status (SES) and are a significant risk factor for severe infection [8]. Vulnerability to more severe infection has both direct effects, including a greater risk of consequential long-term health complications and greater mortality risk, and indirect population-level effects, such as potentially increased infectiousness of symptomatic cases. In England, before the COVID-19 pandemic, life expectancy was 9.4 years longer for men in the least deprived decile than the most and 7.7 years longer for women [9]. These gaps continue to widen: female life expectancy in the most deprived decile fell by 4 weeks between 2014–2016 and 2017–2019 but rose by 11 weeks in the least deprived [9, 10]. The prevalence of underlying health conditions that affect the quality of life is also consistently correlated with local deprivation levels: men in the most deprived decile could expect to live 18.4 years fewer in good health than those in the least deprived decile; the corresponding gap for women was 19.8 years [9].

It is well established that infectious disease burden is associated with SES [11–13]. This is linked to a multitude of complex and interwoven factors including, but not limited to, lack of access to healthcare, poor housing conditions, inability to avoid high-exposure settings such as crowded public places, differences in occupation type, and avoiding restrictions or testing due to mistrust of authorities [14, 15].

Here, we use a novel transmission model to combine the differences in risk of infection and risk of severe disease infection between areas of different relative deprivation, as measured by the IMD, to quantify their relative importance in the observed COVID-19 inequalities in England. We consider underlying health conditions as a key determinant of an individual's risk of developing a clinical case of COVID-19 and focus on the impact of IMD-specific health and age structure on infectious disease burden at the population level. To represent the health status of each decile, we used the UK Census 2021 self-reported health responses. This variable is also used by the ONS to calculate healthy life expectancy and to compare health-related well-being in subpopulations of England [9]. By making simplifying assumptions and modelling a synthetic population, we aim to produce a conceptual exploration of the interaction between underlying health and demographic structure.

Methods

We developed an age-stratified dynamic transmission model for SARS-CoV-2, which was further stratified by IMD decile, and by urban or rural classification in England. Here, we detail how the model was modified to incorporate the characteristics of each decile and geography.

IMD-specific age structure

Each epidemic was simulated on the population of a given IMD decile in either an urban or rural area, to account for the distinct underlying age structures in these areas. We used 17 age groups (0–1, 1–5, every 5 years to 75, and over 75). The mid-2020 (30 June) age-specific population of each lower layer super output area (LSOA), which is on average 1500 people, was linked via LSOA codes to their IMD decile and urban/rural classification (where urban is defined as a settlement with over 10,000 residents) [16–19]. We calculated the size of each age group, specific to each IMD decile and geography, and used this to determine the average age structure of each IMD- and geography-specific population, $n = (n_1, \ldots, n_{17})$, where $\sum_{a=1}^{17} n_a = 1$ in each population. We also calculated the median age for each urban and rural IMD decile and the proportion of each IMD decile residing in urban or rural LSOAs (Additional file 1: Section 1).

Contact matrices

To define contact between the age groups, we used age-specific social contact data for the United Kingdom (UK) for physical and conversational contacts, accessed via the *socialmixr* R package [20, 21]. The contact matrices are

highly age-assortative, with the highest daily contact patterns occurring between individuals in the same age group for those aged 5–19. We projected the contact patterns onto the age structure of each IMD- and geography-specific population in 2020, using the density correction method, by constructing an intrinsic connectivity matrix and scaling this matrix to match the population's age structure [22].

The intrinsic connectivity matrix was calculated from the 2006 UK contact matrix $M^{2006} = \left(M_{ij}^{2006} \right)_{i,j=1,\dots,17}$ and age structure $N^{2006} = \left(N_1^{2006}, \dots, N_{17}^{2006} \right)$ as follows:

$$\Gamma = \left(\Gamma_{i,j} \right)_{i,j=1,\dots,17}$$

$$\Gamma_{i,j} = M_{ij}^{2006} \frac{\sum_{a=1}^{17} N_a^{2006}}{N_j^{2006}}$$

The new contact matrix for a population with age group sizes $N = (N_1, \dots, N_{17})$ and proportions $n = (n_1, \dots, n_{17})$ had entries:

$$M_{ij} = \frac{\Gamma_{ij} N_j}{\sum_{a=1}^{17} N_a} = \Gamma_{ij} n_j$$

Age-specific fraction of COVID-19 cases causing clinical symptoms

We separated infections of SARS-CoV-2 as in [23], into clinical or subclinical cases. Clinical cases of COVID-19 are infections that lead to noticeable symptoms such that an individual may seek clinical care. Subclinical infections do not seek care and are assumed to be less infectious than clinical cases. We defined a population's clinical fraction as the probability of an individual in the population developing a clinical case of COVID-19 upon infection. Here, we related an individual's probability of being a clinical case of COVID-19 to the self-reported health status of their IMD- and age-specific population in England, as a proxy for the relative presence of comorbidities in each population, and then examined how differences in self-reported health status by IMD decile, coupled with differences in age distribution, affect the burden in each IMD decile.

To define health status, we used data from the 2021 Census, specifically the question 'How is your health in general?', with response options of 'very good', 'good', 'fair', 'bad', and 'very bad' [24]. This is provided by the Census stratified by IMD and by age. We then defined 'health prevalence' as the proportion of individuals reporting 'very good' or 'good' general health, stratified by the same age groups and the deciles of IMD:

To map a population's health prevalence to clinical fraction, we used locally weighted regression (LOESS), which fits a smooth curve without any assumptions about the underlying distribution of the data, trained on age-specific health prevalence data from Census 2021 and age-specific clinical fraction values from Davies et al. [23, 24]. Any populations with health prevalences outside of the training dataset's range were assigned the most extreme clinical fractions found by Davies et al. [23], to avoid extrapolation outside of observed values. Health prevalence was highest in children, but children have separate risk factors for severe disease (such as smaller airways), and children under 10 have been found to be subject to a higher risk of clinical COVID-19 cases and a greater infection fatality ratio (IFR) [23, 25] (as observed for other infections such as influenza [26]). Therefore, we fixed the clinical fraction of the 0–9 age group at 0.29, matching that found by [23].

COVID-19 transmission model

The transmission model includes a single SARS-CoV-2 variant, no existing immunity in the population, and natural history parameters drawn from the first wave of the pandemic. We considered the non-pharmaceutical intervention (NPI) of school closures and also explored the effect of vaccinating adults over the age of 65. We developed an age- and IMD-stratified deterministic compartmental model in R (version 4.3.1) (Fig. 1c). There is no mixing between IMD deciles in the model. The aim is to demonstrate the importance of health prevalence and differences in age and social mixing in epidemic impact, rather than to reproduce the COVID-19 epidemic in England.

Individuals are first assumed to be susceptible (*S*) and become exposed (*E*) but not yet infectious after effective contact with an infected individual (Fig. 1c). Each exposed individual then progresses to one of two infected states: subclinical infection (*Is*) and clinical infection, which is represented by a pre-symptomatic (but infectious) compartment (*Ip*) followed by a symptomatic compartment (*Ic*). Each individual then moves into the recovered (*R*) or dead (*D*) compartment, at which point they are assumed to no longer be infectious and to be immune to infection. This susceptible-exposed-infectious-recovered-dead (SEIRD) is an extension of [23], with the addition of a *D* compartment. We ran the epidemic for 365 days, which allowed

$$Health\ prevalence\ = \frac{Number\ in\ 'Very\ good'\ health\ +\ Number\ in\ 'Good'\ health}{Number\ in\ all\ health\ statuses} \tag{1}$$
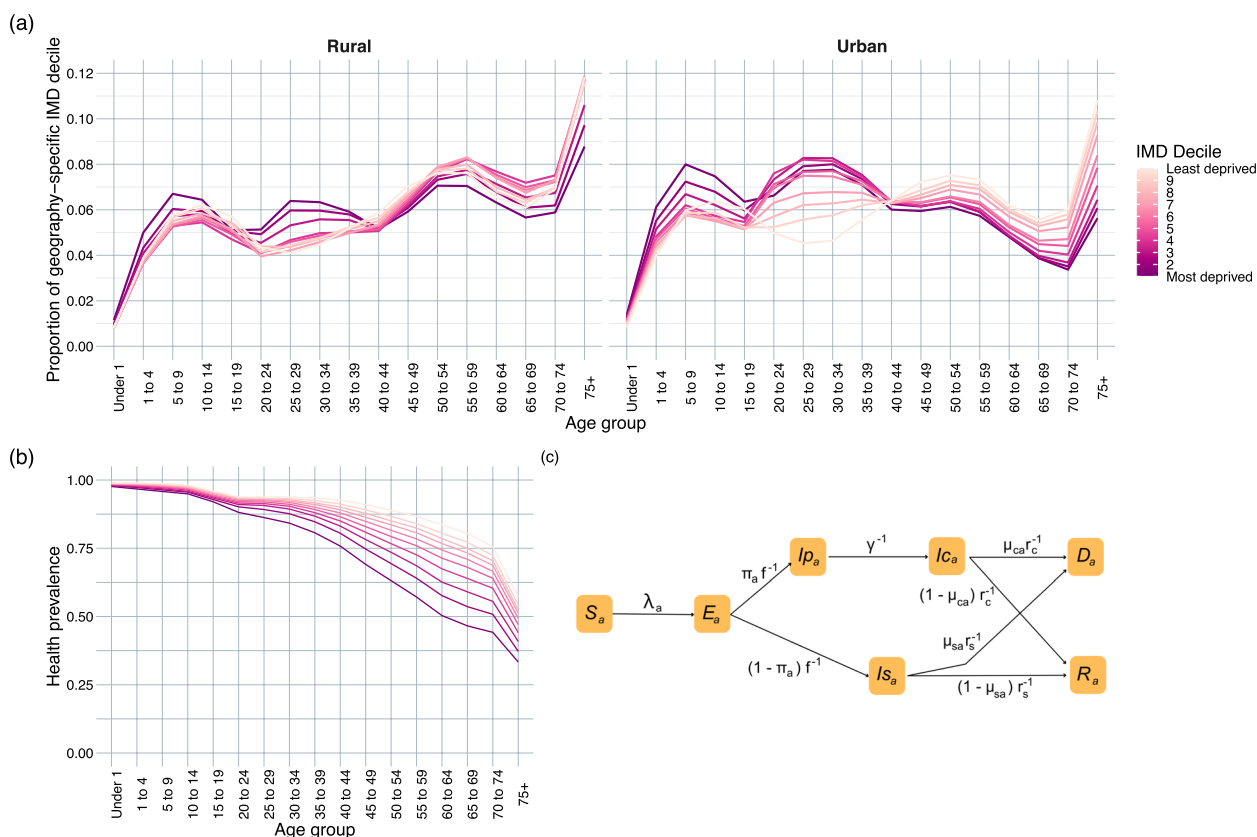
Fig. 1 **a** Proportion of each geography-specific IMD decile in each age group. **b** Age- and IMD-specific health prevalence (1, most deprived decile; 10, least deprived). **c** Age-stratified SEIRD model, specific to IMD decile and geography. Subscript *a* denotes age-specificity, *c* clinical parameters, and *s* subclinical parameters

the completion of each epidemic in each decile and geography. Each epidemic was run on a synthetic population of a fixed IMD decile and urban/rural geography, with no births, non-infection-related deaths, or ageing between the age groups, as the time frame of each epidemic was less than a year. The model also assumed that contact patterns remain constant throughout the epidemic.

The force of infection in age group *k* is given by:

$$\lambda_k = p \sum_{a=1}^{17} M_{ak} \frac{Ip_k + Ic_k + \xi Is_k}{n_k}$$

where p is the probability of a contact between an infected and susceptible individual resulting in transmission of infection, M_{ak} is the mean daily number of contacts that an individual in age group *a* has with individuals in age group *k*, and ξ is the relative infectiousness of subclinical cases. The age-specific clinical fraction is denoted by π_a and depends on the IMD decile. Rates of transition from each disease state are given in Table 1.

We assumed the relative subclinical infectiousness (ξ), to be equal to 0.5, and tested this assumption in a sensitivity analysis (see Additional file 1: Section 12). The transmission probability during a contact was assumed to be $p = 0.06$ as in [23]. The remaining parameter estimates were taken from [23] where possible, to replicate the conditions used to derive the clinical fraction estimates. The mortality probability of subclinical infections was assumed to be 0 for all age groups (*a*). The age-specific probability of mortality of clinical cases was estimated using age-specific IFRs (ϕ_a) found by Verity et al. in 2020 [27] (Additional file 1: Table S4). As the IFR is $\phi_a = \pi_a\mu_{ca} + (1 - \pi_a)\mu_{sa} = \pi_a\mu_{ca}$, since $\mu_{sa} = 0$, the age-specific clinical mortality probabilities were estimated by:

$$\mu_{ca} = \frac{\phi_a}{\pi_a}$$

where π_a is the age-specific clinical fractions for the general population in [23] (Additional file 1: Table S4).

Goodfellow *et al. BMC Medicine*   (2024) 22:162

Page 5 of 13

Table 1 Model parameters

Parameter	Value	Definition	Source
p	0.06	Transmission probability	[23]
M_{ak}	Varies by age and IMD	Daily age-specific contacts	Based on [20]
ξ	0.5	Relative subclinical infectiousness	Assumption
π_a	Varies by age and IMD	Clinical fraction	Based on [23]
f	3 days	Duration of the latent period	[23]
γ	2.1 days	Duration of the preclinical infectious period	[23]
r_c	2.9 days	Duration of the clinical infectious period	[23]
r_s	5 days	Duration of the subclinical infectious period	[23]
μ_{ca}	Varies by age, see Additional file 1: Table S4	Clinical mortality probability	[27]
μ_{sa}	0 for all age groups	Subclinical mortality probability	Assumption

We calculated the total infections, clinical cases, and fatalities per 1000 people, the peak number of clinical cases per 1000 people, the IFR, and the basic reproduction number (R_0) for each IMD decile in urban and rural areas. We also calculated age-standardised measures of total infections, clinical cases, and fatalities within a specific geography for increased comparability. The age-standardised results were of the form:

$$D^{\text{standard}}(365) = \sum_{a=1}^{17} \frac{D_a(365)n_a^u}{n_a}$$

where $n^u = \left(n_1^u, \ldots, n_{17}^u\right)$ is the standard urban population, defined as the proportion of people living in urban LSOAs who are in each age group, similarly $n^r = \left(n_1^r, \ldots, n_{17}^r\right)$ for rural areas.

R_0 in each IMD decile in urban and rural areas was calculated as the absolute value of the largest eigenvalue of the next-generation matrix N:

$$N = \left(N_{ij}\right)_{i,j=1,\ldots,17}$$

$$N_{ij} = pM_{ij}\left(\pi_j(\gamma + r_c) + \xi\left(1 - \pi_j\right)r_s\right)$$

Counterfactual scenarios

To determine the epidemic burden attributable to the difference in underlying health status between IMD deciles, we created the counterfactual health prevalence scenario, where all deciles were assigned the age-specific health prevalence of decile 10 (the least deprived). We calculated the total clinical cases and fatalities in each IMD decile under this assumption. In order to reflect the size of each population (while each IMD decile comprises 10% of the population of England, geography-specific IMD deciles vary widely in size, see Additional file 1:

Table S1), we scaled mortality to mid-year 2020 population sizes and totalled over the 20 populations.

We also created the counterfactual scenario of constant age structure, where we held the age structure constant at the average of each geography-specific England population, independent of the IMD decile. This allowed us to determine the impact of clinical vulnerability separately from the differences in age distribution in each IMD decile. The health prevalence by age remained at the IMD-specific value.

School closures

School closures were a major NPI implemented in the UK during the pandemic, and were implemented evenly across all IMD deciles, unlike some other contact-reducing interventions. We therefore modelled school closures to determine the impact of this intervention across IMD deciles. To quantify the potential differences in the impact of school closures in different IMD deciles, we calculated the effect of school closures on R_0 and total fatalities. The social contact data used is a combination of location-specific contact matrices, defined by home, work, school, and other locations. We removed the school-specific contacts from the contact matrix (retaining contacts in home, work, and other locations), re-projected onto the 2020 age structure, and recalculated the next-generation matrix, N, and its largest eigenvalue, R_0. While assuming that the closure of schools results in a complete subtraction of school-specific contacts may not be realistic (as some contacts would likely be replaced by social interactions in other locations [28]), the results demonstrate the maximum potential impact of school closures.

We simulated the closure of schools after a certain cumulative proportion, P, of the population developed clinical COVID-19 cases. The use of cumulative

Goodfellow *et al. BMC Medicine*   (2024) 22:162

Page 6 of 13

clinical cases as a threshold for implementation is reflective of using total confirmed cases as a measure of the size of an early epidemic. We assumed a value of $P = 0.05$ but tested different values in sensitivity analyses (Additional file 1: Section 11).

Vaccinations

To quantify the relative impact of vaccination rollouts on populations of different levels of deprivation, we calculated the change in mortality rates in each population after vaccinating all adults over the age of 65. This correlates with the earliest vaccination programmes in England, where the first target populations were individuals of older ages. We assumed that vaccination reduced the likelihood of an individual developing a clinical case of COVID-19 upon infection but did not prevent infection. We assumed 76.5% vaccine efficacy against symptomatic infection [29] and reduced the clinical fraction of vaccinated individuals in line with this estimate. To estimate the maximum impact of vaccination, we assumed coverage in over 65s of 100%. We then calculated the change in mortality rates and the number of deaths prevented in each population. We also calculated how many vaccine doses would be given to each population.

Results

Self-reported health prevalence is lower in more deprived areas

There was an older age structure in rural areas compared to urban and a generally younger age structure in more deprived areas (Fig. 1a). The relationship between IMD decile and age structure was confirmed by the median age in each population (Additional file 1: Fig. S1); rural areas have consistently higher median ages than urban areas of the same IMD decile. The median age monotonously increased with affluence in urban areas but peaked in the fourth decile for those living in rural areas.

Age-specific health prevalence was consistently lower in more deprived areas (Fig. 1b). Forty-seven per cent of those aged 65–69 living in the most deprived decile reported living in 'very good' or 'good' health, compared to 80% of those in the least deprived decile. Those living in the most deprived decile experienced the same health prevalence (76%) at ages 40–44 as those in the least deprived decile did at ages 70–74.

Health prevalence was mapped to a clinical fraction in the age groups used in [23] as described in the 'Methods' section (Fig. 2a). Under this assumption, all those over the age of 10 in more deprived areas had a greater likelihood of developing a clinical case of COVID-19 than in other deciles (Fig. 2b).

Epidemic burden increases with relative deprivation

We found that total infections and clinical cases increased with deprivation (Fig. 3a, b). In rural settings, the most deprived decile experienced 72 more crude infections per 1000 population than the least deprived decile; this inequality increased to 90 infections in urban settings. The inequalities in clinical cases were even larger: in rural areas, the most deprived decile experienced 147 more clinical cases per 1000 than the least and 130 more clinical cases in urban areas. The peak clinical epidemic size was 97% larger in urban areas of the most deprived decile than the least deprived decile under these model assumptions and 91% larger in rural areas (Fig. 3c).
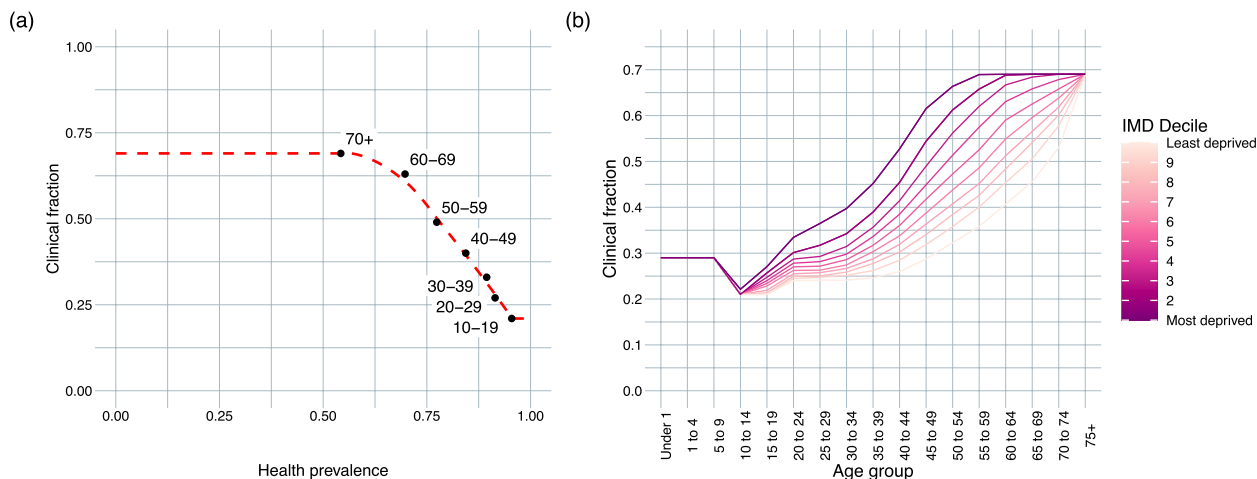

Fig. 2 Results of mapping underlying health to clinical vulnerability. **a** The training dataset of age-specific health prevalence and clinical fraction estimates for the general population of England over age 10, and corresponding predictive model, with linear extensions outside the domain [0.21, 0.69]. **b** Resulting age- and IMD-specific clinical fractions (1, most deprived decile; 10, least deprived)
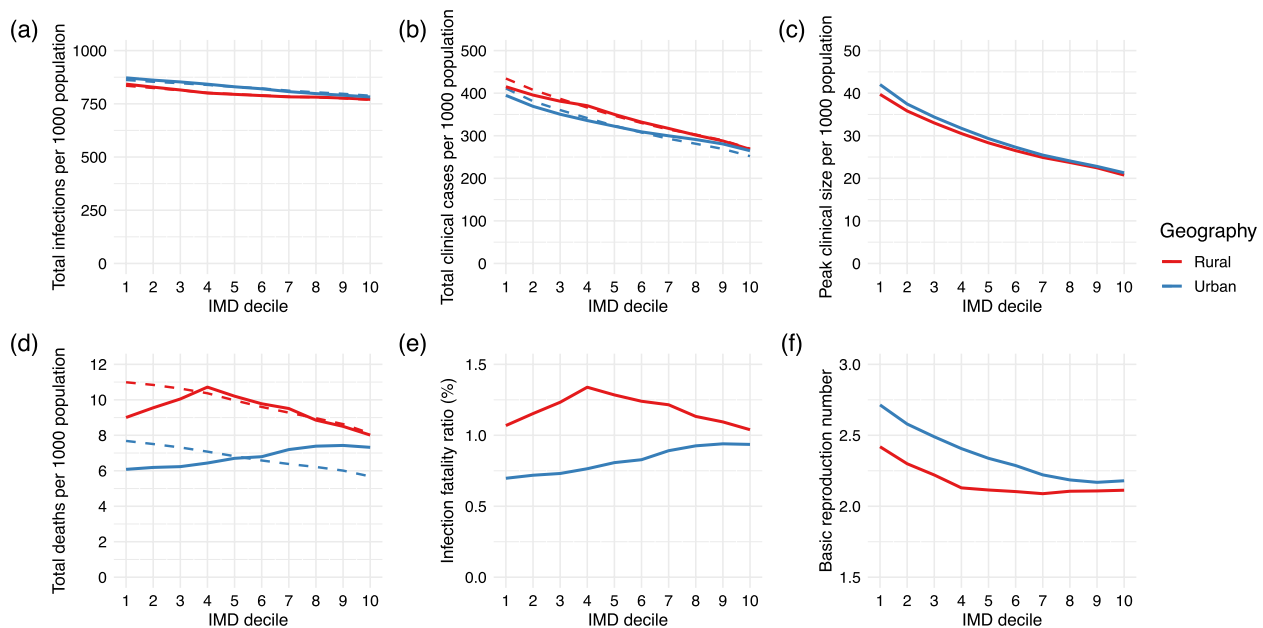
Goodfellow *et al. BMC Medicine* (2024) 22:162

Page 7 of 13

Fig. 3 Measures of the size of a COVID-19 epidemic in each IMD decile and geography. Solid lines represent crude measures, and dashed lines represent those age-standardised by geography. The most deprived decile is decile 1, and the least is decile 10. **a** Total infections per 1000 population. **b** Total clinical cases per 1000 population. **c** Clinical cases per 1000 population at the clinical peak of the epidemic. **d** Total deaths per 1000 population. **e** Infection fatality ratio. **f** Basic reproduction number, R_0

Mortality inequalities differed between the crude and age-standardised results (Fig. 3d). The crude total number of deaths by IMD decile and geography closely followed the median age (Additional file 1: Fig. S1). There was a strong positive association between increasing relative deprivation (decreasing decile) and the age-standardised number of deaths (Fig. 3d). In urban areas, 2.0 more deaths occurred per 1000 age-standardised population in the most deprived decile than the least; this inequality increased to 2.9 deaths per 1000 age-standardised population in rural areas. The IFR followed a very similar pattern to crude mortality (Fig. 3e), likely due to a combination of the relative stability of total infections with deprivation compared to the large variation in mortality rates, and the strong relationship between median age and mortality.

R_0 was generally higher in more deprived areas (Fig. 3f) and ranged from 2.09 in rural areas of the 7th decile to 2.71 in urban areas of the most deprived decile. R_0 was not strongly related to the median age because the lower clinical fractions in younger populations were counteracted by their higher contact rates.

Rural areas experienced fewer total infections, lower peak clinical sizes, and lower R_0 than urban areas, but more clinical cases and deaths, at all levels of deprivation. This is likely due to the older rural age structure, as older individuals had fewer daily contacts than younger individuals and so produced fewer secondary infections

but were more likely to develop clinical COVID-19 if infected.

Further sensitivity analyses considering epidemiological parameters show consistent patterns of age-standardised mortality by deprivation, but a change in the pattern of crude deaths (Additional file 1: Section 12). In particular, if subclinical cases experience a similar level of infectiousness to clinical cases, then crude deaths are more dependent on the age structure and therefore higher in less deprived areas, but in the case that subclinical cases are relatively much less infectious, more crude deaths are consistently observed in more deprived areas (Additional file 1: Fig. S11).

Health-attributable deaths occur at all ages
Under the counterfactual health prevalence scenario, 340,532 deaths occurred, compared to 405,695 under the original assumption. Therefore, 16% of deaths, or over 65,000 fatalities, would have been prevented by achieving health prevalence equity at the level of the least deprived decile. These health-attributable deaths did not only occur in those at older ages: over 29,000 prevented deaths were in individuals aged under 65 (Additional file 1: Fig. S6). At all ages between 30 and 70, over 20% of deaths that occurred under the original model assumptions were attributable to underlying health inequalities (Additional file 1: Fig. S7). We similarly found 21% of clinical cases (3.8 million) to be attributable to inequalities in underlying health under the model assumptions.

Lower clinical infection and mortality rates occurred in the most deprived areas, in both urban and rural geographies in the counterfactual health prevalence scenario (Fig. 4). Age-standardised deaths were consistent across IMD deciles in both geographies when clinical fraction was only dependent on age (Additional file 1: Fig. S10), as is the case in the counterfactual health prevalence scenario. This result contradicts observed mortality rates [3, 4], providing evidence for the existence of a dependency of clinical vulnerability on IMD and more specifically underlying health. The true relationship between IMD and age-specific clinical fraction may be more complex than the assumptions made in this paper; for example, pre-existing immunity may be dependent on previous exposure to coronaviruses [30], which may be associated with SES but is not considered here.

In the counterfactual scenario of constant age structure, we observed more clinical cases and deaths in more deprived areas (Fig. 4). We also considered an underlying age structure independent of IMD decile or geography and found that the most deprived decile experiences 40% higher mortality and a clinical peak 1.88 times larger than the least deprived decile (Additional file 1: Fig. S5), demonstrating the inequality resulting from health prevalence separately from demographic differences. These results indicate that observed inequalities in clinical case numbers and mortality are the result of a complex
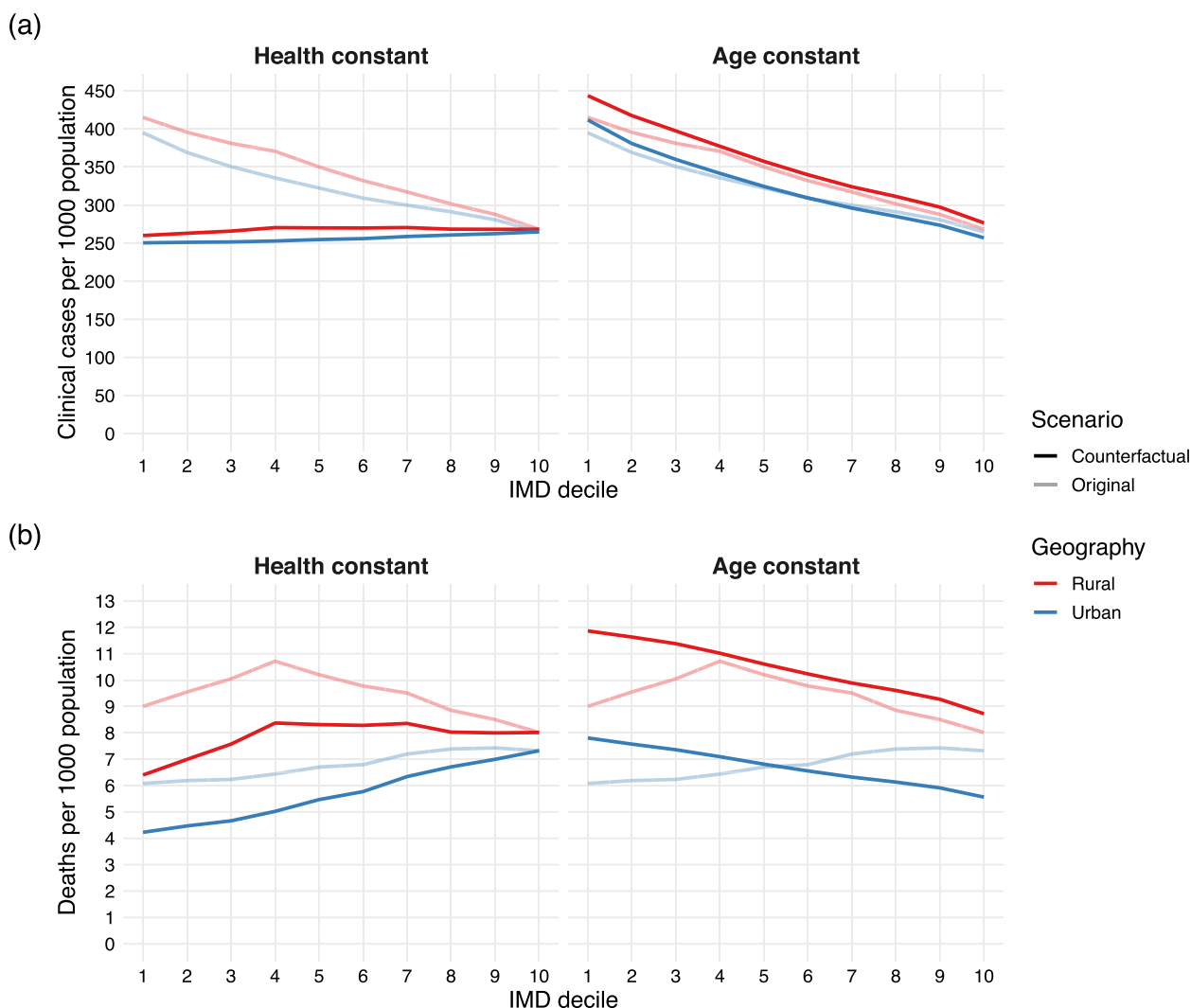

Fig. 4 Epidemiological burden in counterfactual scenarios. **a** Total clinical cases per 1000 population, in geography-specific areas of each IMD decile (1, most deprived decile; 10, least deprived), in the counterfactual health prevalence scenario, and in the counterfactual constant age structure scenario. The original model is shown for comparison in pale lines. **b** Total deaths per 1000 population, in geography-specific areas of each IMD decile, under the same scenarios

Goodfellow *et al. BMC Medicine* (2024) 22:162

Page 9 of 13

interaction between comorbidity-related clinical vulnerability and a population's demographic structure, the outcome of which is not necessarily consistently related to deprivation.

School closures and vaccinations prevent more deaths in less deprived areas

With school closures in place in the model, R_0 decreased for all geographies and IMD deciles but remained larger in urban than rural areas and was consistently higher in more deprived areas in both geographies (Fig. 5a). In urban areas, R_0 was 0.38 higher in the most deprived decile than in the least; the equivalent inequality was 0.29 in rural areas. The largest reductions in R_0 occurred in the most and least deprived deciles, with the least impact in the median deciles (Fig. 5b). This U-shaped result is likely a product of the age structure of each population, as R_0 is driven by both high daily contact patterns in young individuals and greater clinical vulnerability in older individuals (more detail in Additional file 1: Section 11). In all IMD deciles, greater reductions in R_0 occurred in urban than rural areas, likely due to the greater proportion of school-aged children and hence larger reduction in contacts. In no scenario was R_0 reduced below 1 (Fig. 5a), meaning that school closures were not able to halt COVID-19 transmission in any rural or urban IMD decile and could only reduce the epidemic burden under our model assumptions.

By implementing school closures after 5% of the population experienced a clinical case of COVID-19 ($P = 0.05$), 0.113 more crude deaths were prevented per 1000 people in the least deprived urban areas than the most deprived, with a corresponding difference of 0.073 deaths per 1000 people in rural areas (Fig. 5c). This is likely due to a combination of more crude deaths

occurring in more affluent deciles without intervention, improved health conditions, and older population structures. The deaths prevented when age-standardised by geography were approximately consistent with IMD. We also investigated the pattern of prevented mortality when changing the school closure implementation threshold (Additional file 1: Section 11) and found that the effectiveness of school closures in less deprived areas decreased dramatically as P increased.

The reductions in crude mortality rates associated with vaccinating all over 65s were higher in less deprived urban populations, and peaked in the central deciles of rural populations, due to the age distribution of those deciles (Fig. 6a). However, reductions in age-standardised mortality rates were consistently higher in more deprived areas; this is likely to be due to higher clinical vulnerability and therefore a greater absolute reduction in clinical fractions in vaccinated individuals.

When these mortality reductions are considered across the whole population, more deaths were generally prevented in more affluent areas, with over 25,000 deaths prevented in the least deprived deciles, compared to less than 18,000 in the most deprived decile (Fig. 6b). Similarly, more infections were prevented in less deprived areas on a population level, with over 106,000 infections prevented in the least deprived decile compared to just over 36,000 in the most deprived (Additional file 1: Fig. S13b). Fewer vaccination doses were given in more deprived deciles (Fig. 6c), as a smaller proportion of individuals were over the age of 65.

Discussion

We have shown that, under the assumption that vulnerability to clinical COVID-19 infection is a direct result of a population's health prevalence, total COVID-19 infections, clinical cases, and age-standardised deaths
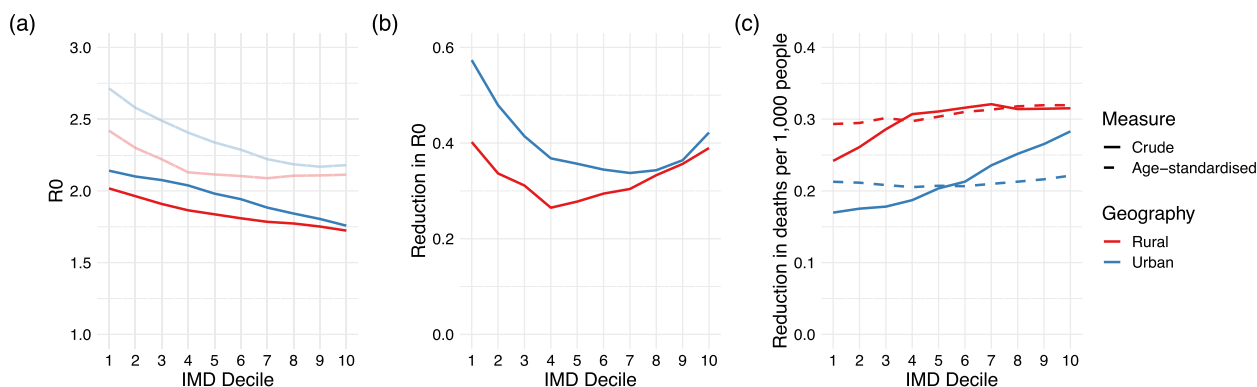

Fig. 5 Results of implementing school closures. **a** R_0 in each IMD- and geography-specific population (1, most deprived decile; 10, least deprived), before (pale lines) and after school closures. **b** Reductions in R_0 due to school closures. **c** Crude (solid lines) and age-standardised by geography (dashed lines) reductions in deaths observed per 1000 population after implementing school closures at $P = 0.05$
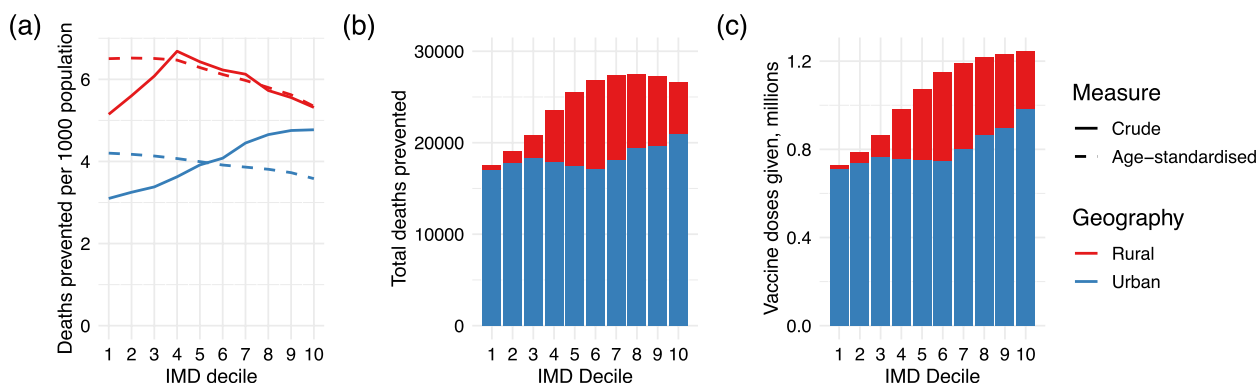
Fig. 6 Results of vaccinating the over 65-year-olds. **a** Deaths prevented per 1000 population, after vaccinating all adults over 65. **b** Total number of deaths prevented by vaccination in each decile (stratified by urban and rural areas). **c** Total number of vaccine doses given in each decile (stratified by urban and rural areas) when vaccinating all over 65s

consistently increased with relative deprivation, therefore exposing those living in the most deprived areas to a greater risk of mortality, as well as more non-fatal consequences such as hospitalisation and long COVID. The peak clinical size of the modelled COVID-19 epidemics, which describes the worst-case scenario hospitals would have to withstand, was approximately twice as large in the most deprived decile than the least deprived. We have found that 16% of the deaths observed under the assumptions of this model, or over 65,000 deaths, would be prevented if every IMD decile experienced the same age-specific health as the most affluent 10% of the country. We have also shown that school closures, which disproportionately negatively affect children's education and well-being in more deprived areas, may also disproportionately benefit the most affluent in society in terms of epidemiological burden [31, 32]. Vaccination programmes targeting over 65s disproportionately target and benefit the least deprived areas of the country.

This study used publicly available data and relied on simplified models of infectious disease transmission; there are hence several limitations to the study. The self-reported nature of the Census data means that there may be systematic differences in how health is reported between age groups and levels of deprivation, due to social desirability and the acceptability of self-reporting ill-health varying by demographic, cultural, and socioeconomic factors [9]. Census data and the IMD may exclude mobile communities and the over 270,000 homeless individuals in England, who are often among the most vulnerable members of society [33, 34]. Self-reported health in 2021 may include the effects of the COVID-19 pandemic, and so preemptively confirm the inequalities that this model aims to investigate. However, the IMD-specific health prevalence in 2021 (Additional file 1: Table S2) is very similar to that found in the 2011 UK Census (75.0%

health prevalence in the most deprived decile and 86.9% in the least deprived decile) [35].

Much of the data used in calculating the IMD relate to 2015–2016 [16]. Any changes that have occurred since are therefore not accounted for in the IMD rankings, such as the wider roll-out of Universal Credit, which has been shown to have exacerbated existing inequalities and negatively impacted claimants' well-being [36, 37]. Health is itself a component of the IMD, potentially limiting the IMD as an exposure for studies with health outcomes; a brief analysis confirms that there are associations between domains of deprivation other than health (Additional file 1: Fig. S14). Other studies have also confirmed the relationship between local deprivation and health outcomes when factoring out the health component of IMD [38].

The assumption of a closed population is unrealistic: apart from during the most stringent lockdowns, which are not represented by the contact patterns used in the above work, individuals will interact and transmit infection between LSOAs as well as within them. A limitation of the contact patterns used is that intrinsic contact patterns are unlikely to be consistent across all IMD deciles and urban and rural geographies. Contact patterns also drastically change in an epidemic, to an extent which depends on SES. The more affluent can more readily reduce their mobility and exposures, while many in the most deprived deciles have less control over their mobility and exposure patterns and are more likely to be in public-facing employment [39]. The ability to self-isolate may also depend on SES, for instance, through the conditions of sick pay. The assumptions of constant contact patterns were necessary due to a lack of readily available data on IMD- and age-specific contact patterns, both under NPIs and in daily life, and as a consequence this study is likely to have underestimated

Goodfellow *et al. BMC Medicine*   (2024) 22:162

Page 11 of 13

the socioeconomic inequalities in epidemic burden. SES-specific contact patterns should be incorporated into epidemic models to include the different contacts that for example arise from different occupational prevalences, ability to reduce mobility, household size, and classroom size. To this end, further data should be collected and made accessible for future research.

By restricting clinical fractions between 0.21 and 0.69, clinical fractions converged at the upper bound in deprived deciles over age 60 while health prevalences were still diverging, meaning that the assigned clinical fractions may underestimate the potential difference in vulnerability, and therefore epidemiological burden, between these IMD deciles. The parameters used for the model, taken from [23] and [27], contain some uncertainty which is included in the original papers but not considered in this study. We assumed that all vaccinations given in our model were given before the epidemic, instead of during, as this made mortality rates easily comparable between the scenarios. While this is unrealistic, this study does not attempt to recreate the exact COVID-19 pandemic but instead provides insight into the interaction between IMD and vaccine impact. This study has not considered vaccination of clinical risk groups, which would likely be larger in areas with lower health prevalence, or taken into account confirmed deprivation-related disparities in vaccine uptake, which are likely to exacerbate existing inequalities [40–42]. Further research into the impact of vaccination on these socioeconomic inequalities would improve the understanding of the interaction between comorbidities, vaccination uptake, age structure, and COVID-19 burden.

Conclusions

The presence of drastically worse underlying health conditions in more deprived areas of England has caused, and will continue to cause, dramatic inequalities in the burden of infectious disease. This study has quantified the potential inequalities in epidemic burden under the assumption that vulnerability to severe infection is a direct result of existing comorbidities. The most effective way to reduce the inequalities in epidemic burden caused by socioeconomic health disparities is to improve socioeconomic equity in health in England. The recommendations made by Health Equity in England: The Marmot Review 10 Years On [10], including maximising empowerment for all, improving standards of living, creating fair employment, and developing healthy communities, would reduce avoidable inequalities in health and by extension avoidable inequalities in epidemic burden.

Abbreviations
CI   Confidence interval
IFR   Infection fatality ratio
IMD   Index of Multiple Deprivation
LSOA   Lower layer super output area
NPI   Non-pharmaceutical intervention
ONS   Office for National Statistics
SEIRD Susceptible-exposed-infectious-recovered-dead
SES   Socioeconomic status

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12916-024-03387-y.

Additional file 1: Fig. S1. Median ages. **Table S1.** Proportion of population residing in urban LSOAs. **Fig. S2.** Projected contact matrices and age-specific total daily contacts. **Fig. S3.** Age-specific health statuses. **Table S2.** Overall health prevalence in each IMD decile. **Table S3.** Age-specific health prevalence and clinical fraction estimates for the general population of England. **Table S4.** Age-specific infection fatality ratios, clinical fractions, and corresponding clinical mortality probabilities. **Fig. S4.** Infections and cumulative clinical cases over the epidemic. **Fig. S5.** Measures of size of a COVID-19 epidemic in each IMD decile, assuming a constant age structure. **Fig. S6.** Deaths occurring in each age group which are prevented in the case of underlying health equity. **Fig. S8.** Mortality under varying school closure implementation thresholds. **Fig. S9.** The reduction in R_0 after school closures, with health prevalence or age structure held constant. **Fig. S10.** Sensitivity analysis of the dependence of clinical fraction on underlying health varies. **Fig. S11.** Sensitivity analysis of relative subclinical infectiousness. **Fig. S12.** Sensitivity analysis of the length of the epidemiological periods. **Fig. S13.** Infection-related effects of vaccinating over 65 year olds. **Fig. S14.** Relationship between the ranks of LSOAs in each IMD domain and health prevalence.

Availability of data and materials
All analysis code and data are available at https://github.com/1035825/imd-covid.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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Goodfellow *et al. BMC Medicine*   (2024) 22:162

Page 12 of 13

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