Using Agent Based Modeling to Understand the Emergence and Reproduction of Social Inequalities in Health†

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Abstract: Studies on social inequalities in health present contradictory findings when they attempt to describe and identify the complex societal mechanisms that give rise to poor health outcomes and health inequalities. This work aims to study the mechanism of reproduction of health inequalities among different population groups using agent-based modeling. We combine evidence-based knowledge and survey data to set the simulation model. Our initial findings show that the combination of the most adverse contextual conditions (i.e. negative environmental exposure and absence of healthcare provision) combined with extreme social inequalities in health might increase mortality drastically. The model suggests that although poor health outcomes may emerge through the action of individual determinants, social inequalities generally emerge and reproduce through non-linear associations and complex multivariate data structures.

Keywords: social inequalities; socio-economic status; health inequalities; agent-based models; emergence; complex systems.

1. Introduction

Despite the present acknowledgment by the scientific community on the relevance of social determinants of health (SDoH) to explain health inequalities, there is persistent uncertainty to explain how contextual and individual level factors are interrelated [1]. Studies have revealed that the higher the socioeconomic status (SES), the lower the prevalence of health problems, illness, disease and death [2-4]. However, current works present contradictory findings when they attempt to describe and identify the complex societal mechanisms that give rise to poor health outcomes and health inequalities [5]. A wide attention has been dedicated to analyze the direct effect of SES indicators on health (e.g. education, income, occupational status, neighborhood, etc.), but the indirect effects with other determinants of health are not commonly described and/or nor totally captured. Determinants of health such as genetic or environmental ones are rarely considered as interdependent elements at different levels within a connected whole [6]. Due to the partial study of factors and mechanisms that are dynamic, multidimensional and context-dependent, the association between the SDoH and health inequalities remains empirically inconclusive and theoretically puzzling [7].

The fundamental problem to evaluate the effect of environmental and social factors on human health is how to trace the (direct/indirect) causative path and the hidden mechanisms that might

describe the process of influence between ecosystems and population health [6]. Research evidence in social and life sciences face the same analytical problems. The general research approach is too simple to account for the complex interplay between environmental and human systems [1]. Studies on social-environmental determinants of health has mainly been generated by research techniques and methods that were developed to answer specific questions about concrete cause-effect relationships (e.g. to study the impact of education or air pollution on cardiovascular health). Existing empirical evidence commonly assumes a certain kind of bivariate directionality between single predictors (e.g. specific behaviors, environmental exposures, etc.) and specific health outcomes that, theoretically, could be separately explained and understood. Therefore, from this analytical point of view, we cannot describe how a wide set of interdependent social and environmental determinants are connected and related with health outcomes and health inequalities in a single whole [7].

Considering the complex nature of SDoH and the impossibility to understand the emergence and reproduction of social inequalities in health from the independent study of single components, recent studies have recognized the need for a complex systems approach to provide new evidence in public health [1,8]. This work aims to study the mechanism of reproduction of health inequalities among different population groups using agent-based modeling.

2. Material and methods

2.1. Data

In this study, we combine evidence-based knowledge and survey data, from the health inequalities module of the European Social Survey (ESS, 2014)[9], to set an agent-based model (ABM) in Netlogo 6.0.4 that is aimed to study the mechanism of reproduction of health inequalities among different population groups under different simulated scenarios. An ABM is an object-oriented computational model aimed at simulating the behavior of autonomous agents and also their interactions under specific contextual conditions. Like in object-oriented programing we have to define the properties of some objects (i.e. agents or simulated individuals) and their methods or behaviors (i.e. a specific set of rules which defines their interactions). Inside the simulation model, agents are self-directed individual entities, though they can also represent collective or clustered units like organizations such as hospitals, schools, or for example companies, and even aggregation or groups of individuals such as healthcare units in a hospital or departments in a particular company[10-14].

In our ABM, according to scientific literature and evidence-based health data, four basic determinants of health were considered (Table 1): (a) genetic and biological factors; (b) environmental factors; (c) medical care factors; and (d) social circumstances and behavioral factors (i.e. SDoH).

Table 1. Determinants of health.

ID	Variables	Measure	Effect size
1	Genetic/biological	Initial health	22%
2	Environmental	Exposure	7%
3	Health care	Accessibility	11%
4	Social/behavioral	Survey data	60%

2.2. ABM Scenarios

The effect size for environmental and genetic determinants of health was derived from health science literature, and relative weight of SDoH indicators was established through the use of survey data and machine learning algorithms. Instead of focusing on the traditional cause-effect approach, we used neural networks (https://cran.rproject.org and http://rstudio.com) to set the explanatory

power of SDoH (R2=0.22), but also to study new theoretical hypothesis across the implementation of diverse scenarios where different population groups may dynamically interact and affect each other.

2.3. Model specification

In the model, we have two health areas (Environment A / Environment B) to simulate environmental effects in health. The simulation introduces two types of agents (population group A / population group B) randomly distributed on the map, so that the level of population and gender/ethnic differences might be also defined. All agents have a predefined level of health (0-100), and the same probability to reproduce, get sick and recover (Figure 1). Finally, the effects of health services availability on agents A and B can be defined separately to reflect disparities in health care provision. Health services are characterized as white colored agents (i.e. see doctors in figure 3 of results section) that are randomly distributed at the beginning of the simulation. These specific agents provides health in a predefined radius to reflect territorial disparities in the access to health facilities.
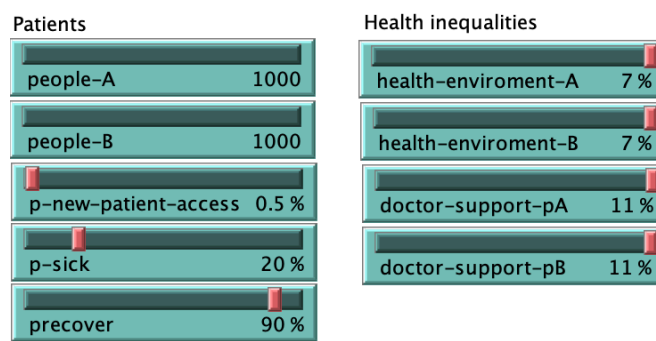
Patients		Health inequalities	
people-A	1000	health-enviroment-A	7 %
people-B	1000	health-enviroment-B	7 %
p-new-patient-access	0.5 %	doctor-support-pA	11 %
p-sick	20 %	doctor-support-pB	11 %
precover	90 %		

Figure 1. Patients and model initialization panel.

SDoH were collated from the ESS data: family net income, smoking frequency, alcohol frequency, unemployed, discrimination, social capital, childhood problems, education level, fresh diet, physical activity, unmet needs, no. people in house, alternative treatments, accommodation problems, and occupational hazards (Figure 2).

Social determinants					
physact	0.0	alttreat	0.0	alcfreq	0.0
soccap	0.0	famincpr	0.0	income	0.0
unmet	0.0	praccom	0.0	occhaz	0.00
phouse	0.0	isced	0.0	etnmin	0.00
freshdiet	0.0	unempl	0.0	cigday	0.000

Figure 2. Social determinants of health panel.

3. Results

3.1. Sick and death rates

Differences in mortality are represented in two basic environments (green environment-A / orange environment-B). Specifically, it can be observed how dead agents (i.e. black colored) and sick agents (i.e. red colored) are more prevalent in the environment-A (green) which presents poor simulated environmental conditions and a lower proportion of health services. The evolution of population (population A[males] vs. population B[females]), number of sick and dead agents, and

their respective rates are dynamically represented in the ABM model through the following panels (Figure 3):
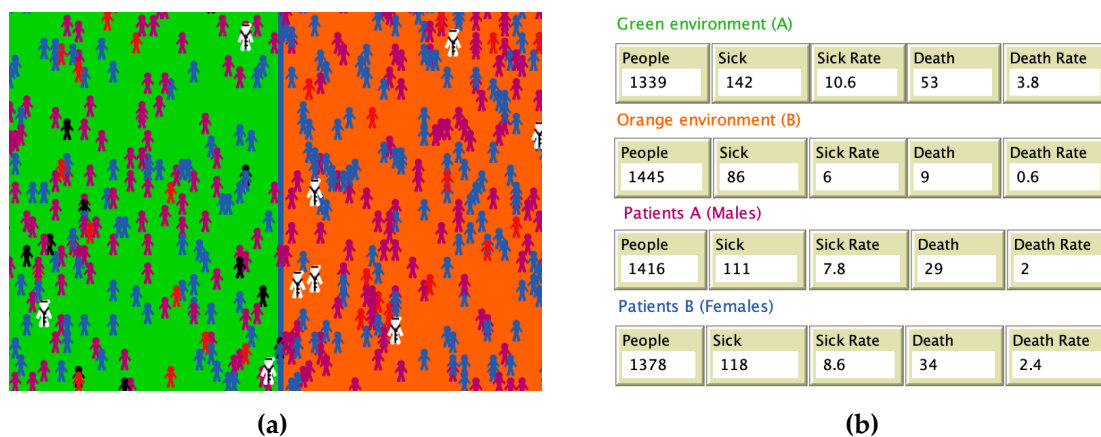

Green environment (A)

People	Sick	Sick Rate	Death	Death Rate
1339	142	10.6	53	3.8

Orange environment (B)

People	Sick	Sick Rate	Death	Death Rate
1445	86	6	9	0.6

Patients A (Males)

People	Sick	Sick Rate	Death	Death Rate
1416	111	7.8	29	2

Patients B (Females)

People	Sick	Sick Rate	Death	Death Rate
1378	118	8.6	34	2.4

(a)   **(b)**

Figure 3. Model visualization: **(a)** sick and dead agents' in two simulated environments; **(b)** population, sick and death rates in environments A and B, and in two different population groups (males/females).

As we can observe, the model calculates the sick and mortality rates for every agent class and for every environment. In this particular simulation result focused on environmental effects, we can observe that predefining a negative environmental effect of 7% in the green environment significantly increases differences in morbidity (sick rate 10.6 per 1000 inhabitants in environment A; death rate 6.0 per 1000 inhabitants in environment B) and mortality (sick rate 3.8 per 1000 inhabitants in environment A; death rate 0.6 per 1000 inhabitants in environment B).

3.2. Mortality patterns in four simulated scenarios

Figure 5 presents the results in four basic scenarios: S1 is the baseline model (no effects); S2 simulates environmental differences (i.e. green environment reduces agents' health up to a maximum value of 7%); S3 adds differences in healthcare provision (i.e. doctor-agents provide additional health to population A up to a maximum of 11%); and S4 adds social inequalities (i.e. the effect of social determinants is set to 0, so that agents' health is reduced 22%).

As we can observe in figure 4, mortality differences between population groups A/B and environments A/B are low in scenario 1 and simulated life expectancy of population (i.e. agents) is about 100 years old. Scenario 2 adds environmental differences between A/B environments (7%) that considerably reduces life expectancy in 25 years in population living in the green environment-A. Scenario 3 includes differences in the provision of health care (i.e. patients A receive less health), so that inequalities in mortality between population A and B progressively grow. Finally, scenario 4 consider the effect of SDoH data collated from the ESS: family net income, smoking frequency, alcohol frequency, unemployed, discrimination, social capital, childhood problems, education level, fresh diet, physical activity, unmet needs, no. of people living in the house, alternative treatments, accommodation problems, and occupational hazards. In this initial model, we study the global explanatory effect of SDoH with a maximum explanatory power of 22% over the final population health of agents in the models. In particular, considering a reduction of these predictors to their joint minimum value and with the objective to simulate the most negative socio-economic conditions, we can observe that life expectancy decreases under the limit of 50 years on average.
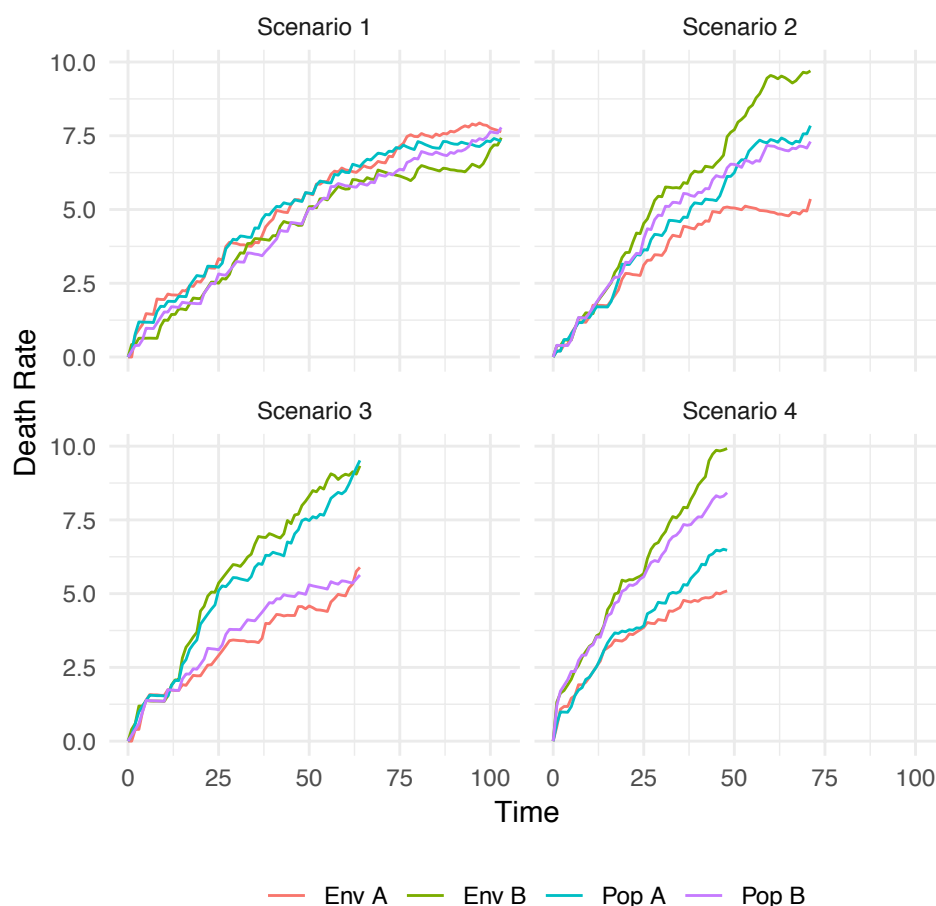
Figure 4. Evolution of death rates in four scenarios.

4. Discussion

Although additional models are needed to capture the mechanism of interactions between structural and intermediary determinants of health, our initial findings show that the combination of the most adverse contextual conditions (i.e. poor environmental circumstances and low healthcare provision) and social inequalities in health might increase mortality drastically (i.e. life expectancy decrease 50 years on average). Therefore, considering that we have used a conservative model grounded on evidence-based knowledge and survey data from European societies, it is plausible that health disparities could be even higher in societies were social and economic conditions could be more unfavorable.

The present study shows ABM as a suitable tool for dynamically studying health inequalities in ecosystems. These models can identify the mechanism and high-order interactions between multiple interfering factors that are hardly observable using traditional epidemiological approaches. In addition, ABM may provide a bridge between qualitative and quantitative methods in health and environmental sciences [1,6,15]. These models are able to integrate the benefits from different theoretical and methodological approaches, and particular tools such as ABM can give response to the micro-processes that are linked with macro-level dynamics in ecosystems. Using ABM we can describe processes that are not dynamically observable through isolated methodologies [14]. Thus, the usage of simulated entities (i.e. agents) is fundamental to change our focus from variables / individuals to a new system thinking approach in health and environmental sciences, but also to understand the underlying mechanism of social ecosystems such as self-organization patterns, emergent social phenomena, processes of human decision making, social norms or collective action, while in parallel we have the opportunity to integrate both qualitative and quantitative evidence-based knowledge in a virtual social laboratory.

5. Conclusion

The present study shows that ABM can be useful to understand the inherent complexity of social inequalities in health. These initial findings suggest that the use of ABM may enhance our understanding of interactive mechanisms between social and environmental determinants of health. The model suggests that although poor health outcomes may directly occur through the action of individual determinants, social inequalities generally emerge and reproduce by non-linear associations and complex multivariate data structures.

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