Examining the role of genotype in socioeconomic disparities in health: test of a theoretical model of social selection

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background

Health disparities by social class in the United States are a central health concern that is not only important from an equity perspective, but also because of its impact on overall population health (1). Compared to other developed economies, the United States has had a stagnating trajectory of life expectancy improvement that has coincided with widening socioeconomic differences in health (2). Models have estimated that in the US per year 245,000 deaths were attributable to low levels of education and 133,000 deaths to poverty (3).

Despite increasing attention to the origins of *socioeconomic* disparities in health, empirical studies of the role played by genotype in these disparities are virtually non-existent. Examples of the genetic contribution to disparities have almost exclusively focused on one of the *other* primary domains of population health disparities – racial/ethnic. Despite little *empirical* attention to the role of genotype in *socioeconomic* disparities – there *is* substantial *speculation*– with analyses frequently referring to the possibility of genetic confounding as an alternative explanation for findings – even as this is not empirically examined. Some of this gap of attention is due to a lack of available data where participants are genotyped and there are detailed socioeconomic measures with which to examine correlations. To be sure, most current theory and evidence suggests that it is differences in the physical and social environment (e.g. factors related to material deprivation itself) that are the primary drivers of socioeconomic differences in health, but absent empirical tests of alternative explanations (e.g. genetic) we will not fully understand where our best efforts should be focused to decrease socioeconomic disparities in health (1). *We propose a theoretical model whereby social processes would result in genetic differences by socioeconomic position if the following are true*: 1) disease genotype results in illness in working adults, 2) illness in adults leads to less work and thus less earnings, 3) the children of these individuals will grow up in lower socioeconomic position households, and 4) the children will be more likely to be lower socioeconomic position (and with a higher probability of disease related genotype). *To our knowledge, the validity of this model of a social process with potentially substantial impacts on population health has not been empirically examined.*

In order to examine the empirical basis for a genetic role in explaining socioeconomic disparities in health I will present basic descriptive data from the Health and Retirement Study consistent with the four premises of our model. Second, I will present a qualitative causal loop analysis based on these correlations to show that these associations could produce socioeconomic differences in disease genotypes. Finally I plan to examine genetic data from the Health and Retirement Study to test our theoretical model.

methods

Analysis of Health and Retirement Study Data

HRS began in 1992 with a nationally representative sample of non-institutionalized US residents born 1931-1941, and their spouses. The Survey Research Center at the University of Michigan provided detailed documentation on HRS sampling design and the selection and validation of health measures. For my analysis I used the HRS Rand Files Version K that were cleaned and organized to facilitate longitudinal analysis across waves of data from 1992-2008. I used a random effect model to account for the multiple observations of individuals across waves.

I will examine whether genotypes associated with health outcomes that explain the greatest amount of socioeconomic disparities (ischemic heart disease, cerebrovascular disease, hypertension, diabetes) differ by four key measures of socioeconomic position (income, education, occupation and wealth). I will secondarily examine whether these same genotypes are associated with reduced work hours or decreased wages, and whether the strength of association is proportional to differential distribution by socioeconomic position, consistent with my theoretical model.

Loop Analysis

As a first step toward a more formal presentation of the model as well as qualitative assessment of consistency of observed pathways with our hypothesis, we performed loop analysis as described by Puccia and Levins (4), implemented with software developed by Dinno (5).This method of modeling systems with feedback based on user input of positive, negative or null relationships between variables has had limited use in ecology, and has had recent application in health sciences. It is well suited as an initial exploration of our hypothesis given that we do not have accurate quantitative estimates of the relationships between covariates in our system of

interest, and feedback and reinforcing systems are key features of our proposed model. While loop analysis is sometimes characterized as overly simplistic, it is a realistic first step for examining our process and as a basis for ongoing work to more quantitatively model expected genetic distributions based on observed social patterns of illness, socioeconomic position and work.

results

Analysis of Health and Retirement Study Data

In regression models predicting work in individuals age 50 to 65, we find that level of health has a significant impact on work, and subsequent income. Odds of work are lower ($p<0.001$) for individuals with diabetes, cancer, heart disease, stroke and high blood pressure, controlling for level of education, parental level of education, and race/ethnicity.

Loop Analysis

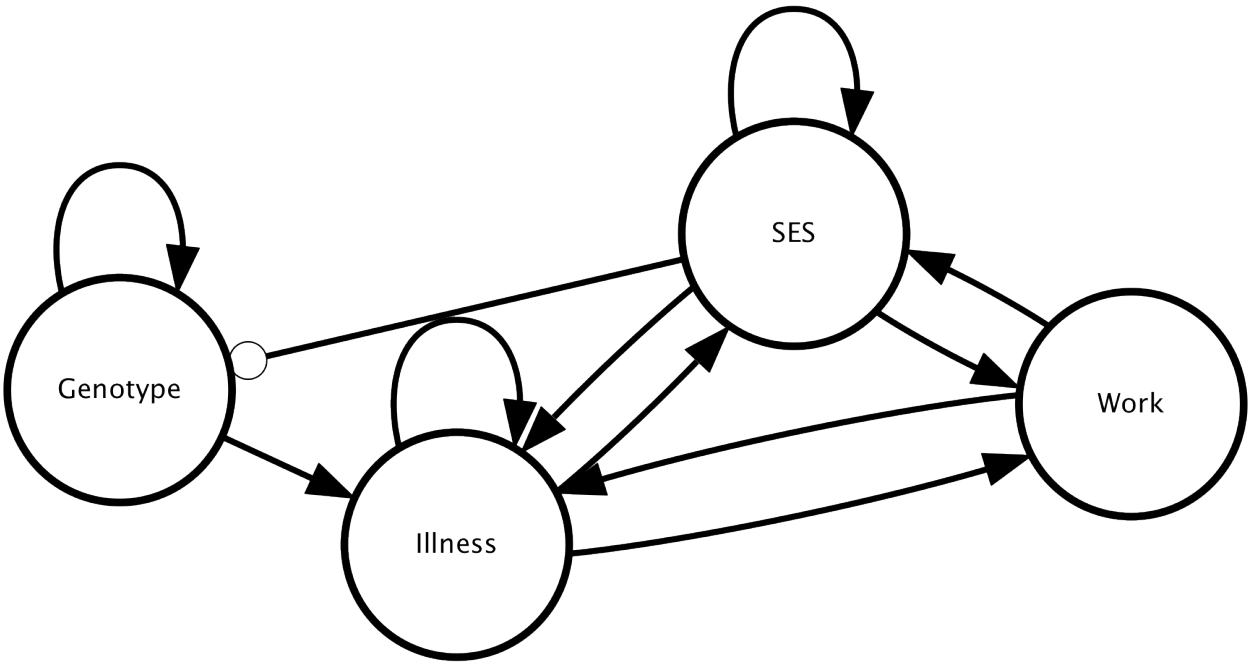
The figure presents a loop diagram of the proposed relationship, where factors in the model (e.g. SES) are examined *across generations*. Arrows indicate positive relationships, with an arrow running into itself indicating a positive feedback across generations. For example the arrow from genotype back to itself indicates the genotypes will be similar across subsequent generations, as is the case with SES and illness as well. Arrows between nodes indicate impacts that are proposed to occur (and for which there is documentation in the literature) either within or between generations. Finally, a connection with an open arrow indicates suppression. The table shows the nature of the relationships implied based on our loop model, with the positive sign indicating that this system produces a positive relationship between SES and health related genotype.

conclusions

Based on our *a priori* proposed relationships between key social system variables, we are likely to observe a positive association between genotype and SES. Future work to be presented will examine how this relationship differs with different model assumptions, including other variables in the model, and other assumptions about the nature of the connections between variables. Most importantly, ongoing work to be presented using Health and Retirement Survey genetic data will

be able to empirically test whether the prediction from our model is consistent with disease related polymorphism distributions.

figure



table

Prediction Matrix

	Genotype	SES	Illness	Work
Genotype	?	+	-	?
SES	+	+	-	?
Illness	-	-	+	?
Work	?	?	?	?

references

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