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Time to Specify Mechanisms of Community Health Benefit or Harm

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The health effects of “place” have become increasingly clear. Neighborhood characteristics, such as socioeconomic disadvantage, access to clinics or pharmacies, quality of housing and environmental exposures, or even per capita involvement in community life (“collective efficacy”), are all relevant for health. Multi-level analyses show that neighborhood factors may affect health apart from individual expression of the same factors. For example, individual educational attainment affects cognition in late life, but so does the proportion of residents in an individual’s neighborhood who have completed high school or college (Wight, et al., 2006; Aneshensel, et al., 2011). Neighborhood levels of perceived collective efficacy, apart from one’s own perception, affect risk of major depression (Ahern, et al., 2011). Black residents of predominantly white zip codes are more likely to make health care visits than black residents of predominantly black zip codes even with adjustment for relevant individual attributes, such as insurance status or education (Gaskin, et al., 2012).

It is time now to establish mechanisms for neighborhood effects.

Research to date has rarely tested mechanistic hypotheses and often treats neighborhood influence as a black box. For example, the cognitive advantages of living in a more highly educated neighborhood may be due to the presence of libraries, less acceptance of untreated chronic disease that may compromise cognition, greater opportunities for physical activity, greater access to health care, or unspecified gain from social contact with more educated neighbors (Wight, et al, 2006; Aneshensel, et al., 2011). The lower risk of depression in neighborhoods with greater collective efficacy may be due to the greater social support associated with more cohesive communities or better buffering of stressful events (Ahern, et al., 2011). Greater utilization of health care services among blacks living in white neighborhoods may reflect increased availability of health care services in such neighborhoods (Gaskin, et al., 2012).

These are all plausible hypotheses. But few studies have taken the next step of actually specifying a key mechanism in a way that would allow definitive testing. Most research seems content to establish an independent neighborhood effect or significant cross-level interaction and call it a day. Why should this be so?

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Part of the problem is lack of appropriate data. Longitudinal data, necessary for causal analysis, are often unavailable. Also, adequate community-level indicators at an appropriate level of granularity are also in some cases unavailable and may be difficult to combine with panel study data. Adequate data require many geographic areas, with a reasonable number of individuals in each place.

But the analytic problem may be more deep-seated. Inability to assess mechanisms in this case may point to a limitation in the prevailing public health paradigm for investigating community effects. Multilevel models are limited in their ability to test pathways and identify mediators of individual-community associations. We need for more flexible analytic tools that can take into account mediation, multi-directional causation, and nonlinear relations (Galea & Ahern, 2006).

One way to capture these dynamic properties is to use agent-based computational models to “grow” phenomena of interest (Epstein, 2006). Agent-based models are computer representations of heterogeneous microentities (agents) that interact and change over time as they encounter other model agents and features of environments. *In silico* experiments using the models can capture system-level properties as these emerge from the interaction of agents. Agent-based simulations, increasingly important for infectious disease modeling, may be an important new tool for public health scientists who wish to understand the effects of place on health.

An advantage (or challenge) of these simulations is the need to specify microlevel behaviors, such as interaction patterns and daily movement, in particular environments, such as communities, that give rise to the community-level effects evident in epidemiologic data. These offer one approach to plausible mechanisms of community effect. To return to one of the examples mentioned earlier, we can explicitly assess the effect of community levels of education on individual cognitive performance if we simulate the interaction of agents across different neighborhoods and develop rules for incrementing cognitive performance resulting from such interaction. Can we grow differences as large as those observed in epidemiologic studies? How much of a gain in cognitive performance from daily interaction is required in our models? How much contact must individuals in different neighborhoods have? Does it matter if the contact occurs in the low- or high-education neighborhood? By forcing such specificity, we commit to testing mechanisms of community effect.

A number of agent-based models have recently appeared that take this approach (Yang, et al., 2011; Auchincloss, et al., 2011). Their value at this point is largely to open up the black box of neighborhood effects. At this point they are less useful for forecasting population health indicators (El-Sayed, et al., 2012), though this limitation may be less pressing in models that are implemented using realistic synthetic agent populations. We can expect progress in understanding neighborhood health effects from the rigorous causal thinking required by computational simulation.

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