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Incorporating social environment data in infectious disease research

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Research on pathogens such as tuberculosis, human immunodeficiency virus (HIV), and cholera have long considered the importance of the environment—particularly the social environment—for understanding population patterns of infectious diseases and their inequities. However, a growing body of research has begun extending such approaches more broadly in infectious disease research. At a fundamental level, the social environment can impact infectious disease risk in two ways: by affecting a person's frequency or duration of exposure to pathogens, or by affecting their susceptibility to infectious.¹ Social stress theory has emerged as a key mechanism through which a person's susceptibility to infectious disease might be shaped by their social environment.^{2,3} The work by Marko Elovainio and colleagues⁴ published in this issue of *The Lancet Public Health* provides an important addition to this line of inquiry—making a strong case that the experience of loneliness is a robust predictor of susceptibility to hospital-treated infectious diseases.

There are multiple strengths of the Elovainio and colleagues' study, including the utilisation of prospective cohort data which allows for the clear establishment of the temporal relationship between the exposure and outcome, a long follow-up period with a large study population, and the replication of analyses in a second cohort. Although the authors investigated both loneliness and social isolation, the main headline of their work was regarding loneliness. They reported a hazard ratio for hospital-treated infections of 1.41 (95% CI 1.36–1.45) in an age-adjusted and sex-adjusted model. This association was attenuated but robust in a model controlling for demographic factors, lifestyle factors, and other health conditions, and was replicated in a second large cohort study.

In an era when infectious disease has again become a prominent feature in our collective consciousness, we must continue to push the boundaries of the factors we consider in both the study and control of infectious diseases. In response to Elovainio and colleagues, and to further extend their work, we highlight two key areas for which additional development is needed to facilitate a continued expansion of our understanding in this growing area of research.

First, a robust data infrastructure is needed to collect comprehensive data on diverse aspects of the social environment as well as infectious diseases. A key strength of the current

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investigation was that they were able to leverage two existing, large-scale cohort studies that had linked medical records with clinical diagnostic data. However, for many infectious diseases, including COVID-19 and influenza, we do not have access to such robust data on the social environments of individuals nor the clinical diagnoses of these illnesses. This could be improved by encouraging routine population-level surveillance of infectious diseases to also collect rich demographic and social data. Additionally, existing large-scale cohort studies such as the English Longitudinal Study of Ageing or the National Study of Adolescent to Adult Health that already collect robust social environment data could include infectious diseases as part of the regular health outcome ascertainment for their sample.

Second, standards for routine data collection, and data collected during a pandemic or other emergency, should be developed and their use incentivised. The ongoing COVID-19 pandemic has laid bare the importance of the social environment in shaping disease burden and severity and has spurred a large amount of research in this area. Data collected throughout the pandemic have provided crucial insight into the role of the social and built environments in shaping infectious disease burden, but the generalisability of these data is limited by a lack of coordination and standardisation. Some of these standards and practices are already underway (eg, Common Data Elements,⁵ ATSDR Rapid Response Registry,⁶ and the Social, Behavioral, and Economic COVID Coordinating Center⁷), but they need to be both broadened and incentivised to be meaningfully incorporated into practice. Without such incentives—be they from funders, statistical agencies, or other parties—these data will continue to be collected in an ad-hoc manner, limiting their value for broader research.

In their Article, Elovainio and colleagues provide an important contribution to our understanding of the complex interplay between the social environment and infectious diseases by identifying an association between experiences of loneliness and risk of hospitaltreated infections. Continued research into the relationship between the social and built environment and infectious disease risk is crucial, but will require thoughtful changes to the public health data infrastructure.

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