

Epidemiologists have used maps to understand disease transmission risk for at least 160 years, including John Snow’s fabled, fine-scale resolution maps of London that spatially linked human cholera cases to water infrastructure. However, for much of this history, these spatial tools have remained rudimentary and singularly focused on understanding the spatial distribution of human cases for public health. Welcome to the 21st century, where science and data unavailability.

In this spirit, A. Townsend Peterson’s Mapping Disease Transmission Risk is a welcome addition. His main thesis is repeated like a mantra throughout the text, and was well received by me—disease mapping must move beyond geography and better incorporate ecology and biogeography. Peterson defends this thesis over 20 (mostly very short) chapters primarily via a broad, conceptual overview focused on methodological considerations and caveats to ecological niche modeling (ENM), and through brief descriptions of numerous relevant case studies. To his own admission, “This volume is neither a manual nor a how-to book; furthermore, it is not a comprehensive review of spatial epidemiology or a full presentation of ecological niche modeling” (p. xi).

Although the volume’s focus is on ENMs (a correlative approach), I agree strongly with Peterson that better understanding of disease biology and the mechanisms of transmission are key to developing useful risk maps and moving toward a predictive framework. However, I disagree that the transmission systems of avian influenza and other diseases are too complex and “necessitate analyses at the level of human cases” (p. 63). Similarly, there is scant mention of how to integrate dynamic models (e.g., SIR models) with spatial approaches to better inform these mechanisms of transmission. In general, I found the volume repetitive in parts and the author quick to highlight caveats but slow to offer tangible solutions (Chapter 14 is a notable exception). For example, a table compiling available data resources or a section on optimal study design, including what field data to collect when not available online, would have been useful in Chapter 3 (particularly for graduate students), rather than focusing on poor data quality and data unavailability.

Lastly, Peterson’s very brief and collective treatment of ecological communities, land-use change, control programs, socioeconomic factors, and human population, behavior, and demographics as “risk modifiers” (Chapter 15) was deficient. These factors are central to understanding and modeling disease risk, not just “modifiers,” and any framework to map risk must go beyond plotting the probable ecological niche of a pathogen to a more complete integration of the entire suite of anthropogenic and ecological factors that contribute to disease emergence, amplification, and spread.

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What constitutes a population? This question is particularly timely in light of increased human mobility and exogamy (outbreeding). Furthermore, novel statistical approaches and affordable genetic