Preface

Disease outbreaks are as much markers of as they are threats to modern civilization. What successfully evolves and spreads depends on the matrix of barriers and opportunities a given society presents its circulating pathogens (FAO 2013; Engering et al. 2013; R.G. Wallace et al. 2015).

For most of its history, *Vibrio cholerae* predated upon plankton in the Ganges delta (Johnson 2006). Only once humanity switched to urban sedentism and later by nineteenth century trade and transport became increasingly integrated in geography and economy did the cholera bacterium evolve an explosive human-specific ecotype.

Simian immunodeficiency viruses, spilling over for centuries, emerged out of their nonhuman *Catarrhini* reservoirs as HIV only when colonial expropriation turned subsistence bushmeat and the urban sex trade into commodities of industrial scale (Wallace 2010; Pepin 2011; Timberg and Halpern 2013).

Domesticated stock served as sources for human diphtheria, influenza, measles, mumps, plague, pertussis, rotavirus A, tuberculosis, sleeping sickness, and visceral leishmaniasis (McNeill 1977/2010; Wolfe et al. 2007). Ecological changes brought upon landscapes by human intervention selected for spillovers of malaria from birds and dengue fever, malaria, and yellow fever from wild primates.

The new pathogens selected for improvements in medical technologies and public health (Watts 1997, Colgrove 2002). In turn, a daisy chain of innovation in agricultural and industrial methods, accelerating demographic shifts and new settlement, rejuxtaposed potential host populations, promoting new rounds of spillover (Kock et al. 2012; R.G. Wallace et al. 2015).

Presently, humanity's organizing ethos orbits neoliberal capitalism, even in opposition (Plehwe et al. 2006). Neoliberalism is a program of political economy aimed at globalizing laissez-faire economics for multinationals, promoting free trade, and shifting state expenditures in favor of protecting private property and deregulating economic markets (Harvey 2005, Centeno and Cohen 2012, Ganti 2014). Once applied at a particular locale, the doctrine has considerable impact on local landscapes and functional ecosystems alike, with decisive effect upon the

fortunes of infectious disease (Maye et al. 2012; R.G. Wallace and Kock 2012; Jones et al. 2013; Maye et al. 2014; Degeling et al. 2015; Mann et al. 2015; Wiethoelter et al. 2015; R. Wallace et al. 2016).

In this volume, we present the emergence of an urbanized Ebola in West Africa in late 2013, spreading from human to human and infecting 28,000, as a quintessential example of such a neoliberal transition. Societal shifts extending from local environmental and social spaces out to global relational geographies turned what until this point had been a backwater virus into a sudden protopandemic threat.

The first paper collected here, published in fall of 2014 as the outbreak in West Africa finally captured the globe's broader attention as possibly more than a regional threat, positions the outbreak as global in origins. R.G. Wallace et al. (2014) describe the likely cascade by which multilateral structural adjustment and a multinational land rush encroached upon regional forests and truncated medical infrastructure. The resulting increases in Ebola spillover likely accelerated the emergence of a human-to-human infection. The authors hang the specific mechanism on the spread of monoculture oil palm to which Ebola-bearing species of bats are attracted and along which the latter's interface with humans likely expanded (Shafie et al. 2011).

The team pegs such epidemiological shifts, of which Ebola's urbanization appears representative, to a model of changes in environmental stochasticity (R. Wallace and R.G. Wallace 2015). The ecosystemic fluctuations across populations of a "noisy" forest typically truncate the chains of transmission on which such a virulent pathogen depends. Such noise asymptotically drives the pathogen population to local extirpation. When monoculture production is suddenly imposed upon such an agroforestry, the inherent disruption to pathogen transmission is stripped out, accelerating spillover and explosive growth across the host populations that remain.

The second paper, published here for the first time, revisits an outbreak of another Ebola, the Reston species, among industrial hog in the Philippines in 2009 and more recently in China (Barrette et al. 2009; Pan et al. 2014). The shifts in stochasticity at the heart of the first paper are extended into a more realistic travel-based formalism of spatial spread, an agroecological logic gate for epidemic control, and a more explicit connection to economic models of agricultural production by way of a variation of the Black-Scholes approach to option pricing.

The latter model shows that pathogen emergence in intensive production may outpace the margins agrifood extends to biocontrol. The financial holes that result appear largely filled only after the outbreak begins and by other stakeholders entirely. Livestock, contract farmers, public health, smallholder production, wildlife, the environment, and governments across administrative units are asked to bear the costs in health and/or finances. Should such substantial costs be returned to company ledgers, agribusiness as we know it would cease to exist. R. Wallace et al. (2016) hypothesize that as industrial livestock expands, in contrast, including across areas of Africa in which Ebola has already emerged as a human infection, multiple novel phenotypes are likely to emerge out of such an agroeconomic frame.

The third paper expands the epistemological implications of the West African outbreak, particularly within the context of what appears to be the development of an efficacious vaccine (Henao-Restrepo et al. 2015). Blips in new cases con-

tinue to intermittently reappear across the three countries most affected—Guinea, Liberia, and Sierra Leone—even as the outbreak is repeatedly declared ended (e.g., Barbarossa et al. 2015). The paper hypothesizes that the social environment driving the ecotypic shift in Ebola in the region may be strong enough to offset the epidemiological advances offered by the new vaccine and other proximate interventions (R.G. Wallace et al. 2016). Emergency and palliative responses cannot be separated from structural context. The virus, even when maneuvered back upon its proverbial heels, threatens to reemerge by virtue of causes beyond the biomedical, however important the latter.

The fourth paper, published here for the first time, applies a control theory model that prioritizes social policy and economic structure. The ecology of infectious disease emerges from more than aggregate measures of population dynamics, depending decisively upon such context.

The fifth paper, again published here first, examines mechanisms by which a deadly pandemic may become the trigger for an avalanche of socioeconomic disintegration that can carry far heavier burdens of morbidity and mortality than the disease outbreak itself.

Indeed, big picture, these papers together explore the relationships among the ostensible objects driving or dragging upon the disease—the pathogen and vaccines against, for instance—and the fields within which such objects relate. As we review here, growing banks of data at the molecular and clinical levels indicate that the Ebola strain in West Africa is little different from its forest predecessors. As causality emerges from the interaction between objects and field, a shift in regional agroeconomics can engender a parallax effect in which an unchanged object expresses new properties by virtue of a shift in context alone (Zizek 2006).

In short, as humanity's epidemiological history attests, context is more than merely a veritable stage on which pathogens and immunity clash. The regional agroeconomic impacts of global neoliberalism are foundational, felt across biocultural organization, down so far as virion and molecule. Exploring such connections frames what will likely be a cutting-edge science of the twenty first century (R.G. Wallace et al. 2015).

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