

# Chapter 2

## Ebola in the Hog Sector: Modeling Pandemic Emergence in Commodity Livestock

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### 2.1 Introduction

Human impact is increasingly transforming planet Earth into planet Farm. Forty percent of the planet's surface is dedicated to agriculture, with many millions more hectares to be brought into production by 2050 (Foley et al. 2005, Alexandratos and Bruinsma 2012, FAO 2013a). Livestock, representing 72 % of global animal biomass, are simultaneously highly concentrated and widely dispersed across the planet's surface (Smil 2002; Van Boeckel 2013; Robinson et al. 2014) (Fig. 2.1). The livestock sector uses a third of available freshwater and a third of cropland for feed (Steinfeld et al. 2006; Herrero et al. 2013). Feed production, enteric fermentation, manure, animal processing, and transportation in turn produce greenhouse gases at 7.1 gigatonnes CO<sub>2</sub>-eq per year (Gerber et al. 2013).

Agricultural impact extends to emergent disease. If by its global expansion alone, commodity agriculture increasingly acts as a nexus through which pathogens of diverse origins migrate from even the most isolated reservoirs in the wild to the most globalized of population centers (Graham et al. 2008; R.G. Wallace 2009;

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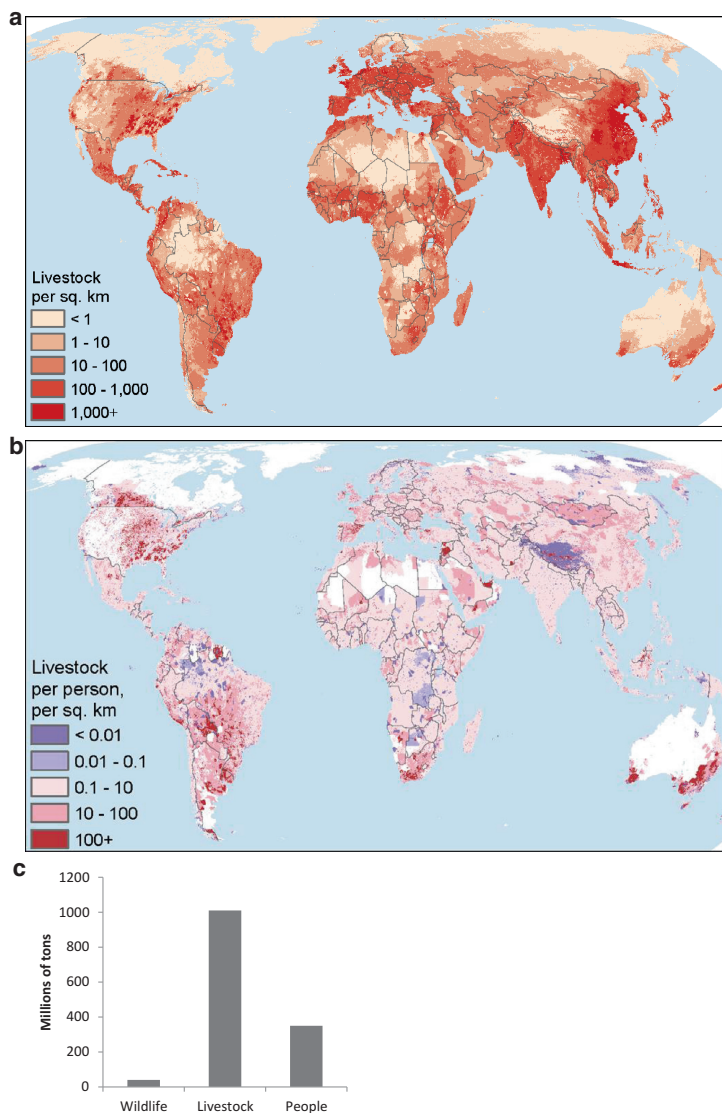
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**Fig. 2.1** Global livestock. **(a)** Total livestock (cattle, chickens, ducks, pigs, sheep, and goats) per  $\text{km}^2$  (2006) (Eckert IV projection). Sixty-four percent of all cattle, chickens, ducks, pigs, sheep, and goats are found on 2 % of Earth's land surface. At the same time, 10 % of these stocks are found across 69 % of land surface. Data from the Gridded Livestock of the World v2.0 (Robinson et al. 2014). **(b)** Livestock per human per  $\text{km}^2$ . Livestock data from Gridded Livestock of the World v2.0 (Robinson et al. 2014). Human data from Global Rural–Urban Mapping Project (GRUMP) v1.0 (2000) (Balk et al. 2006). All areas with less than 0.1 persons/ $\text{km}^2$  masked out. **(c)** Global biomass wildlife, livestock, and people. UNFAO data.

Jones et al. 2013; Liverani et al. 2013; Engering et al. 2013; FAO et al. 2013b). The lengthier the associated supply chains and the greater the extent of adjunct deforestation, the more diverse (and exotic) the zoonotic pathogens that enter the food chain (Wolfe et al. 2005; R.G. Wallace et al. 2010; FAO et al. 2013b). Among such emergent pathogens are industrial *Campylobacter*, Nipah virus, Q fever, hepatitis E, *Salmonella enteritidis*, foot-and-mouth disease, and a variety of novel influenza variants, including H1N1 (2009), H1N2v, H3N2v, H5N1, H5N2, H6N1, H7N1, H7N3, H7N7, H7N9, and H9N2 (Epstein et al. 2006; Myers et al. 2006; Graham et al. 2008; Leibler et al. 2009; Jones et al. 2013; Khan et al. 2013).

Intensive agriculture's diseconomies of scale extend beyond the unintended epidemiological consequences of globalizing transport and distribution. Its production cycles degrade ecosystemic resilience to disease; complicate interventions by treating humans and animals as markets and commodities first; and by its genetic monocultures, high population densities, rapid throughput, and expanding exports accelerate pathogen spread and evolution (R.G. Wallace 2009; Mennerat et al. 2010; Drew 2011; Van Boeckel et al. 2012; R.G. Wallace and Kock 2012; Ercsey-Ravasz et al. 2012; Liverani et al. 2013; Khan et al. 2013; FAO 2013b; R.G. Wallace et al. 2015).

The recent outbreak of *Zaire ebolavirus* in West Africa appears another case of a pathogen's agroecologic transition. ZEBOV, among other Ebola species, had been circulating in the area for at least a decade before emerging as a human-to-human infection (Schoepp et al. 2014; Dudas and Rambaut 2014; Gire et al. 2014). Sudden Ebola spillovers in the region were already associated with socioeconomically driven shifts in land use. Formenty et al. (1999), for instance, described the outbreak of *Tai Forest ebolavirus* in Cote d'Ivoire as arising out of a combination of impacts upon Parc National de Tai:

The habitat in the region has also been modified constantly by human migration from regions north of the forest belt. This process has sharply increased in the last 6 years since the start of the civil war in Liberia; the influx of refugees doubled the local population between early 1992 and early 1993 and again in the summer of 1994. Massive migration of humans with their domestic animals and other commensal organisms could explain environmental perturbation near the forest and consequently in the forest. The increased deforestation pressure resulting from this influx would also result in perturbation of the habitat. Crop activities have developed on the edge of the park and in the park itself. Illegal plantations and poaching into the Tai National Park have increased from 1985 to 1995 and led to the existence of a large area of farmland and broken forest. This area was only 2 km from the home range of the [possibly infected] chimpanzees that were studied.

R.G. Wallace et al. (2014) proposed ZEBOV emerged as a human-to-human infection late 2013 out of another such phase change in agroecology, in this case brought about by regional neoliberal development, juxtaposing multinational mining, logging, intensifying agriculture, and a structurally adjusted medical infrastructure (Bausch and Schwarz 2014; Kentikelenis et al. 2014). Neoliberalism is a program of political economy organized around globalizing laissez-faire economic liberalism, promoting free trade, and reducing state expenditures for the greater population in favor of protecting private property and deregulating economic markets (Harvey 2005; Centeno and Cohen 2012; Ganti 2014).

As a first attempt in framing the mechanism by which ZEBOV emerged in the region, R.G. Wallace et al. (2014) hypothesized the strain arose as oil palm, to which Ebola-bearing fruit bats are attracted, underwent a classic case of creeping consolidation, enclosure, commoditization, and proletarianization in the Guinée forestière. At one and the same time, the transition in agroforestry curtailed artisanal production and may have expanded the human–bat interface over which the virus crosses.

Saéz et al. (2015) have since proposed the initial ZEBOV spillover occurred when children outside Meliandou, Guinea, including the putative index case, caught and played with bats of an insectivore species (*Mops condylurus*) previously documented an EBOV carrier. Whatever the specific source, one of many in the region, shifts in agroeconomic context appear a primary causal factor. Previous studies show *Mops* also attracted to expanding cash crop production in West Africa, including sugar cane, cotton, and macadamia (Noer et al. 2012; Taylor et al. 2013; Stechert et al. 2014).

Indeed, in contrast to bushmeat, burial practices, and specific host reservoirs of arguably undue attention (Jones 2011), from its initial identification in Sudan the Ebola genus appears repeatedly associated with shifts in land use related to nascent capitalization (Groseth et al. 2007). Newly emergent variants appeared connected to area-specific cotton (WHO/International Study Team 1978; Baron et al. 1983), mining (Bertherat et al. 1999), and logging (Morvan et al. 2000), with each outbreak subsequently amplified in local hospitals (Mylne et al. 2014). The WHO/International Study Team (1978) began its description of the very first reported outbreak:

In Sudan, the first cases of hemorrhagic fever are thought to have originated in Nzara township in three employees of a cotton factory situated near the town centre. The factory forms part of a large agricultural cooperative with 2000 employees. A staff of 455 is employed in the factory, which produces cotton cloth from raw cotton grown in small holdings throughout the region.

The financial capital involved seems negligible compared with what globally circulates and more recent influxes into the Sub-Sahara, but novel epizootics may arise from even small shifts in land use (Patz et al. 2004; Murray and Daszak 2013). The Zande Scheme, a colonial developmental strategy begun 1946 around forcing relocated Azande to grow cotton in Nzara's environs and spin and weave it in town, was brought to a halt by civil war in 1965 (Russell and McCall 1973/2013; Onwubuemeli 1974). But upon peace in 1972, the area rapidly repopulated and much of the local rainforest was reclaimed for subsistence farming, with cotton, as of the outbreak, continuing as the area's dominant cash crop (Roden 1974; Smith et al. 1978).

Although Smith et al. (1978) note the roof of Nzara's cotton factory housed large populations of bats, anticipating efforts to identify a source in every Ebola outbreak to follow, the epistemological implications to draw here are broader. The elusive balance between what Gonzalez et al. (2005) identify as Ebola's sudden clinical expression and silent circulation suggests the molecular, clinical,

and epidemiological characteristics of the virus are necessary but insufficient explanations for disease dynamics (Engering et al. 2013; FAO 2013b; R.G. Wallace et al. 2015). The agro-economic context within which wildlife, crops, livestock, and human populations interact appears a foundational cause for characterizing the virus's epizoonosis.

The difficulty in identifying a clear, single reservoir for Ebola in Africa (and now elsewhere) appears to represent more a paradigm's breakdown than a failure in diligence (Olival and Hayman 2014). Ebola's dynamics likely embody contingent interactions across multiple host species, communities, and environments reminiscent of avian influenza (Gilbert and Pfeiffer 2012), with repeated if relatively cryptic transmission across different host guilds in different anthropogenic settings (Plowright et al. 2015). The greater the combinatorial of host species so identified, the less explanatory power traditional modeling apparently offers, including systems of simultaneous equations tracking the transmission mechanics internal to a host population, from susceptibles to the infectious to the removed (e.g., Hayman et al. 2013). Strictly ecological niche models meanwhile omit social sources of external effect (e.g., Pigott et al. 2014). The order of complexity seems to extend beyond the approaches' methodological framework.

To more explicitly articulate the relationships among the inputs underlying pathogen emergence, including Ebola's, we expand here on R.G. Wallace et al. (2014), developing a series of stochastic models integrating epidemiology, spatial dynamics, and economics. The inductive, epiphenomenal analysis introduced here aims to guide field research in a more integrative direction across biocultural domains, even beyond what the One Health approach connecting wildlife, livestock, and human health claims sufficient (Wood et al. 2012; R.G. Wallace et al. 2015).

We begin with a simple analytic model of epidemic population growth under conditions of stochastic uncertainty. We move to modeling a pathogen's spatial dynamics under stochasticity by simple diffusion and by travel flows structured by underlying agroecologies. To introduce an agroecological logic gate for epidemic control, we characterize Ebola's emergence in another part of the global food chain—in commodity hog in the Philippines. Finally, we develop a variation of the Black–Scholes pricing model to sketch the effects of environmental stochasticity on the costs of biocontrol and containment under different agro-economic regimens.

## 2.2 Modeling an Epidemic in a Stochastic Environment

Perhaps the simplest mathematical treatment of the early stages of an epidemic is found in a stochastic differential equation (SDE) (Oksendal 2010; R.G. Wallace et al. 2014).

Taking  $N_t$  as the number of infected individuals at time  $t$ , the outbreak at first will grow according to the SDE

$$dN_t = \alpha N_t + \sigma N_t dW_t \quad (2.1)$$

where  $\alpha > 0$  is the rate of growth,  $\sigma > 0$  the magnitude of system noise under conditions of volatility, and  $dW_t$  represents a simple white noise process. Applying the Ito chain rule to  $\log[N_t]$  to compute the derivative of a stochastic variate, the time dependence of the outbreak is driven by the expression

$$N_0 \exp[(\alpha - \sigma^2/2)t] \quad (2.2)$$

where  $N_0$  is the initial number of infected individuals, and the term  $\sigma^2/2$  represents the classic Ito correction factor.

If noise is sufficiently large, the infection population rapidly declines to zero, in spite of the condition  $\alpha > 0$ .

The constant  $\alpha > 0$  in Eq.(2.1) represents in Kermack and McKendrick's influential deterministic, exactly solvable, and approximate susceptible-infectious-removed (SIR) model (Bailey 1975, Eq.6.1) the basic reproductive number " $R_0$ " factor  $\beta n - \gamma$ , where  $\beta$  is the infection rate,  $\gamma$  the removal rate, and  $n$  the initial uninfected population. If  $\beta n - \gamma > 0$  in that formalism, the epidemic ignites and proceeds up the epicurve.

At this point, reactive control strategies aimed at decreasing  $\beta$  by prophylaxes, hygiene, and/or trade restrictions or at increasing the removal rate  $\gamma$  by vaccination or depopulation can be costly and effort-intensive in human and livestock populations, and difficult in and even detrimental to wildlife, especially for pathogens of high mutation rates and high dispersal (Capua and Marangon 2007; Kleczkowski et al. 2012; Humphries-Waa et al. 2013; Knight-Jones and Rushton 2013; Longworth et al. 2014; Zhang et al. 2014).

By contrast, the second term of Eq.(2.1), in which the noise  $dW_t$  is amplified by the factor  $\sigma N_t$ , introduces a volatility that differentiates our approach from Kermack–McKendrick. In this treatment, environmental stochasticity is no mere fuzzy error, rather representing a keystone impact upon epidemiological systems. Sufficient noise snuffs the epidemic candle, as it were. Tornatore et al. (2005) use a similar but more complicated stochastic model, finding a similar condition for global asymptotic stability.

Indeed, such preventative control may be intentionally "farmed." Following Khasminskii (1966/2006; 2012, Theorem 4.1), the formalism in Appendix extends the model to multidimensional systems subject to nonlinear cross-influences in which relatively stable endemic modes—equilibrium states of dynamic Markov processes—can be suddenly triggered into epidemic explosions. In the other direction, we hypothesize careful internal structuring—what may be called "epidemic prevention farming"—can produce the noise sufficient enough to control outbreaks over a wide swath of epidemiological parameters.

While Kermack–McKendrick SIR models are often represented as having complex probability structure (e.g., Bailey 1975), recasting epizootics within an SDE can open up additional insight, allowing the well-understood methods of differential equations to be extended in a relatively simple manner with the Ito chain rule and related tools. The approach can often cut through considerable mathematical underbrush.

A simple spatial version of our initial model follows Okubo (1980), introducing diffusion of the form,

$$\partial N(x, t)/\partial t = \mu \partial^2 N(x, t)/\partial x^2 + \alpha N(x, t) \quad (2.3)$$

We assume “cultivation,” in a large sense, takes place on spatial patches of dimension  $L$ , separated by a fixed distance across which infection must undergo spatial diffusion.

A solution using spatial Fourier series then leads to a time dependence proportional to

$$\exp[(\alpha - C^2 \mu/L^2)t] \quad (2.4)$$

where  $\alpha$  is again growth rate,  $\mu$  is the spatial diffusion coefficient, and  $C$  is a constant of order 1 that depends on the dimensionality of the diffusion.

Thus the infection dies out if  $L$  is less than the critical patch size  $L_c = C\sqrt{\mu/\alpha}$ . The result can be obtained directly from dimensional analysis. Indeed, such a calculation has served as the basis for the design of pest control strategies. See the classic text by Murray (1989, Sect. 14.8) for details.

How do the initial SDE and spatial diffusion model relate? Under both treatments, if ecological noise is sufficiently large, the infection population will collapse whatever its coefficient of growth. We can track the relationship deeper into the models’ mechanics, with both conceptual and practical implications.

Following Tornatore et al. (2005), in Eq. (2.1) we are assuming a stronger role for volatility—the  $\sigma N_t$  term—than what follows from a simple diffusion approximation to the deterministic Kermack–McKendrick approach (e.g., Tuckwell and Williams 2007). The latter generates a volatility term  $\propto \sqrt{\sigma N_t}$ . Indeed, such correspondences are often bounded in very particular ways. Beddington and May’s (1977) map  $dN_t = \alpha N_t \rightarrow \alpha(t)N_t = (\alpha_0 + \sigma dW_t)N_t$  is needed to make Eq. (2.2) consistent with the spatial analysis of Eq. (2.4), a correspondence reduction suggesting that stochastic generalizations of deterministic models are not the easy routine often assumed.

The implications extend out to how we think about the phenomena modeled. The influence of stochasticity appears to be of its own domain, distinct from factors driving deterministic population growth and explicitly dependent on underlying agroecological, epidemiological, and socioeconomic contexts, constructs, and policies, a conclusion Liu (2013) and Cai et al. (2013), modeling SIR epidemics, arrived at themselves.

R.G. Wallace et al. (2014) use the model elaborated upon here to frame the recent Ebola outbreak in West Africa, hypothesizing a key ecological role for the expansion of palm oil agroforestry from artisanal, episodic, and dispersed to year-round hybridized monocrop. Expanding patches of such commodity plantations, in conjunction with increasing deforestation, provide an attractive alternate habitat for behaviorally plastic species of fruit bats and, following Saéz et al. (2015), insectivore species thought to act as Ebola reservoirs.



The method serves as the foundation for an SDE model of noise-driven phase transitions in a physical system (Horsthemke and Lefever 2006), suggesting a more general analysis of disease dynamics could use the noise strength  $\sigma$  as a “temperature” analog in modeling punctuated “socioviral” evolutionary transitions (e.g., R. Wallace and R.G. Wallace 2015).

## 2.3 A More Realistic Spatial Model

The spatial mechanisms used in epidemic theory, as in Eq. (2.3), are usually variants on simple diffusion. More realistic treatments examine space that is strongly structured by travel flows, which focus on biological and social processes that embody the detailed mechanisms for spatially contagious diffusion at different scales.

Gould and R. Wallace (1994) explore the spread of another infectious disease of African origins—HIV/AIDS in the USA—using empirical measures of travel intensity derived from a Markov analysis. The model is based on the area density of the equilibrium distribution of a stochastic matrix  $\mathbf{P}$  indexed by exchange measures between spatial subunits that is normalized to unit row sums (Kemeny and Snell 1976). The approach leads to a canonical form related to, but significantly different from, classical diffusion theory, in that spatial homogeneity is now replaced by the complex structures of actual travel in the affected region. An important feature is the identification, by dimensional analysis, of a “characteristic area” associated with diffusion in a travel field that expands as the infection propagates.

The argument is direct. Following Gould and R. Wallace (1994), we assume a basic model

$$n_i(t) = f(t, \rho_i/A_i) \quad (2.5)$$

where  $i$  is the index of the spatial area of interest,  $n_i$  the case rate per unit population,  $t$  is the time,  $A_i$  the area of subunit  $i$ ,  $\rho_i$  is the Markov equilibrium distribution associated with unit  $i$ , and  $f$  is a monotonic increasing function. The vector  $\rho$  is defined by the relation  $\rho = \rho\mathbf{P}$ . Since  $n_i$  is a dimensionless number, we must have an appropriate dimensionless monotonic increasing relation

$$n_i(\tau) = F\left[\frac{\rho_i}{A_i}\mathcal{A}(\tau)\right] \quad (2.6)$$

where  $\tau \equiv t/T_0$ ,  $T_0$  is a characteristic system time, and  $\mathcal{A}(\tau)$  is a characteristic area that grows as the disease spreads. The generalization to a continuous system would be direct, involving introduction of spatial variates  $\mathcal{X}$ , and functions

$$n(\tau, \mathcal{X}), d\rho/d\mathcal{A}|_{\mathcal{X}}$$



$F$  may actually be fairly complex, incorporating dimensionless “structural” variates for each subdivision  $i$ , for example, local percentage indices of income stratification or occupational status, crime rates, etc., in the case of R. Wallace et al. (1997).

We conjecture, however, that  $\mathcal{A}(\tau)$  itself expands according to a stochastic diffusion process, even though the process defined by  $F$  is a kind of deterministic mixmaster driven by systematic local travel patterns. From the perspective of the polio examples in R. Wallace and R.G. Wallace (2015), it may be necessary to treat the spread of the characteristic area as a carrying capacity problem, since, once the infection has become endemic, it is “everywhere.” The appropriate SDE follows as

$$d\mathcal{A}_\tau = [\alpha \mathcal{A}_\tau (1 - \mathcal{A}_\tau / K)] d\tau + \sigma \mathcal{A}_\tau dW_\tau \quad (2.7)$$

which generalizes Eq. (2.1) to a carrying capacity  $K$ , in the presence of white noise.

Using the Ito chain rule on  $\log(\mathcal{A})$ , one obtains—as a consequence of the added Ito correction factor—the long-time endemic limits

$$\begin{aligned} \mathcal{A} &\rightarrow 0, & \alpha &< \frac{\sigma^2}{2} \\ \mathcal{A} &\rightarrow K \left(1 - \frac{\sigma^2}{2\alpha}\right), & \alpha &\geq \frac{\sigma^2}{2} \end{aligned} \quad (2.8)$$

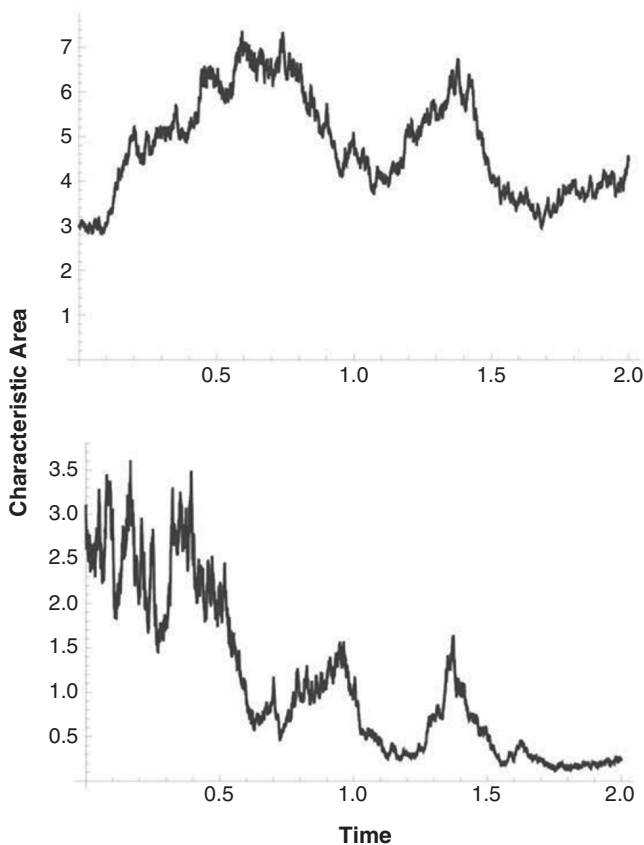
Thus, in this model, the “noise”  $\sigma$  can depress the final endemic level of infection even for a propagating epidemic.

Figure 2.2 shows two simulation examples, with  $\sigma$  below and above criticality. For the first, the characteristic area fluctuates about a lowered endemic level. For the second, the area compresses toward extirpation.

R. Wallace et al. (1999) analyze data for very large-scale AIDS spread in the USA, and other contagious processes, using related Markov methods. The team found HIV diffused down the hierarchy of US cities—from large economic centers to second cities—by way of the structure of national travel as well as the policies of deindustrialization and inner city discrimination driving unsafe behaviors spreading HIV. The approach may be adaptable to slower systems that do not equilibrate.

Transitions in Ebola’s characteristic area may be defined by a similarly broad combination of factors, in this case including migration patterns and agroecomic shifts in land use across wildlife and human populations. Developing industrial agriculture influences not only the epizootic interface across species, but also road networks, cheap transport, and the ecoimmunology of human populations subjected to shifts in labor requirements, demographics, diets, and associated habits of behavior.

Should ZEBOV be characterized in this way by noise  $\sigma$  below criticality, the recent contraction in the outbreak in West Africa may be short-lived, perhaps even in the face of welcome changes in public health intervention (Washington and Meltzer



**Fig. 2.2** Simulating  $\mathcal{A}(\tau)$  based on the Ito chain rule expansion of  $\log(\mathcal{A}_\tau)$  using Eq. (2.7). The simulations apply the ItoProcess function in Mathematica 10 for white noise.  $\mathcal{A}(0) = 3$ ,  $K = 10$ ,  $\alpha = 1$ ,  $\sigma = 0.5, 1.5$ . The critical value for  $\sigma$  is  $\sqrt{2}$ . 2000 time steps. While the *upper trace* fluctuates at values less than  $K$ , the *lower* collapses toward zero.

2015; Merler et al. 2015; WHO 2015). Under such circumstances, ZEBOV may now be endemic in human and reservoir populations alike, prone to repeated spillover and rebound.

There appears something fundamental still missing in the catalog of mechanisms by which a marginal epizootic embedded in deep agroforestry turns into a regionalized urban epidemic. To start the effort at more explicitly characterizing the interactions influencing the virus's shift across biocultural domains, we next review another Ebola species' emergence. Anthropogenic impacts on its "wild" reservoir's habitat appear to have brought the comparatively fast-evolving *Reston ebolavirus* into its own human-proximate host, ecology, and molecular phenotype (Carroll et al. 2013).

## 2.4 REBOV in Commodity Hog

Ebola has entered the global food chain by means other than neoliberalizing horticulture in Sub-Saharan Africa.

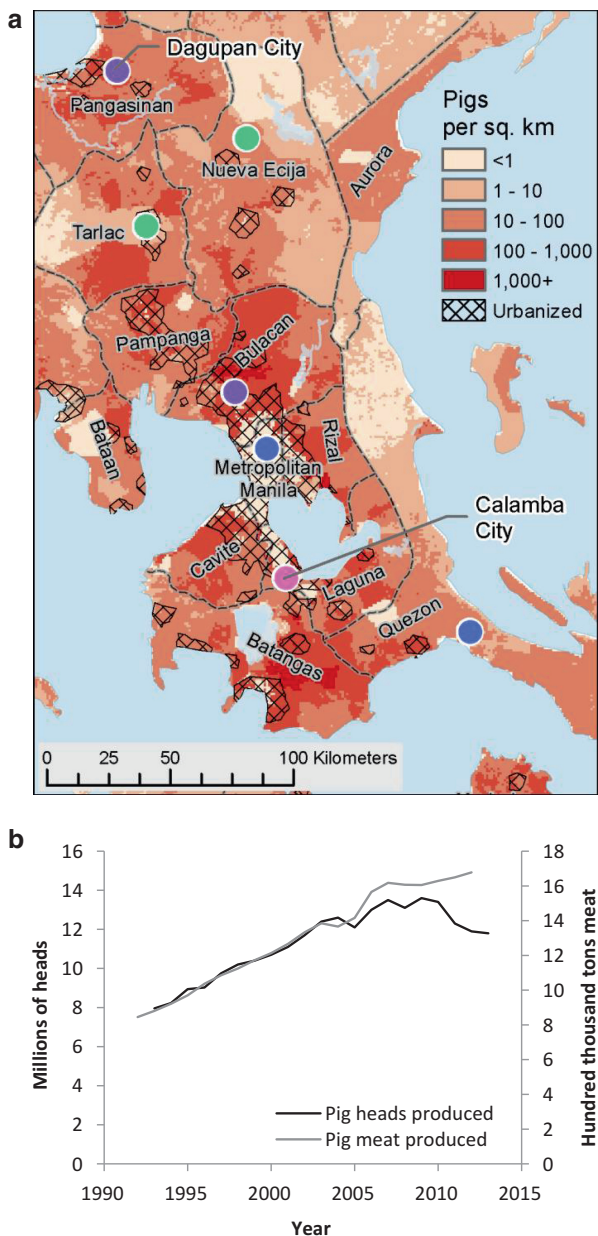
In 2008, half-a-world away in the Philippines, in an area that Peterson et al. (2004) projected as a suitable Ebola niche, *Reston ebolavirus* emerged twice in hog raised in industrial filieres, outside Metro Manila (1.7 million people) and Dagupan City (150,000) (Barrette et al. 2009; Miranda and Miranda 2011; Sayama et al. 2012) (Fig. 2.3a). The REBOV outbreaks followed several in cynomolgus macaques in the Philippines between 1989 and 1996, although the virus's regional origins remain obscured as purifying selection may mask the phylogenetic age of ebolavirus lineages and filovirus-like elements have been detected in bat and other small mammal genomes dating back tens of millions of years (Taylor et al. 2010; Wertheim and Pond 2011).

The Win Farm in Sto. Nino, Pandi, Bulacan, one of the piggeries on which REBOV emerged in 2008, is 30 km from the Diliman Arboretum Forest and 125 km from the Quezon National Forest Park (Fig. 2.3a). One apparent natural reservoir, the wide-ranging karst fruit bat *Rousettus amplexicaudatus*, was found IgG ELISA-positive for REBOV NP in the two parks (Heaney et al. 2005; Cardinosa and Reyes 2009; Taniguchi et al. 2011). The highly vagile *Rousettus* forages widely across orchards, other agricultural areas, and disturbed secondary forest.

The outbreaks among lab *Macaca* in Reston, Texas, Sienna, and the Philippines were traced to a single monkey farm and exporter in Calamba, Laguna (Miranda and Miranda 2011) (Fig. 2.3a). The 2008 outbreak showed REBOV more widely, if heterogeneously, distributed (Sayama et al. 2012). At the Sto. Nino farm, 79 % and 81 % of the swine tested positive for REBOV-NP and -GP specific antibodies by IgG-ELISA. At the second piggery hit, the Lambino farm in periurbanized Barangay Parian, Manaoag, Pangasinan, 90 % and 67 % tested positive. None tested positive on a Tarlac farm sampled 2 years later, following negative tests at two inspection check points in St. Nino, San Jose City, Nueva Ecija (Barrette et al. 2009; Sayama et al. 2012).

The 2008 swine strains shared 95 % similarity and the same clade with those of the macaque outbreaks, but significantly greater genetic divergence from each other than from the 1989 reference isolate, suggesting both continual circulation and polyphyletic origins (Barrette et al. 2009). Multiple substrains appear endemic and the 80 % prevalence suggests either broad environmental exposure or hog-to-hog transmission. Barrette et al. (2009) also identified six clinically unaffected farm workers (4 %) who, denying all contact with bats and monkeys, expressed IgG antibodies to REBOV, confirming human infection (Bausch 2011).

Hog comprises 60 % of total Filipino meat output, at 24.5 million head and 956,250 tons annual live weight (Costales et al. 2007; Lapus 2014). Forty-seven percent of production takes place on the island of Luzon where the 2008 outbreaks emerged in Bulacan and Pangasinan, the provinces hosting the country's two greatest concentrations of commercial hog (Costales et al. 2003; Alawneh



**Fig. 2.3** REBOV in the Philippines. **(a)** Hog farm outbreaks, Pandi, Bulacan and Manaog, Pangasinan, 2008 (purple); negative hog tests, Tarlac City and St. Nino, San Jose City, Nueva Ecija, 2008–2010 (green); positive IgG ELISA in Geoffroy's rousette bat *Rousettus aegyptiacus*, 2008–2009 (blue); positive cynomolgus macaques *Macaca fascicularis*, 1989–1996 (pink); on Livestock Geo-Wiki distribution of pigs in Central Luzon, Philippines, 2006 (pigs/km<sup>2</sup>) (Barrette et al. 2009; Taniguchi et al. 2011; Miranda and Miranda 2011; Sayama et al. 2012; Robinson et al. 2014). *Projection*: Cylindrical equal area with a standard parallel of 10 degrees. **(b)** Hog production in the Philippines. Pig heads and pig meat produced, 1992–2013. UNFAO data.

et al. 2014). By the Import Liberalization Program, Ginintuang Masaganang Ani Livestock Program, the Agriculture and Fisheries Modernization Act, and World Trade Organization obligations for minimum access volume for multinational hog producers, a rural, smallholder-based sector on the island has been transformed into conurbanized chains of advanced commercial operations, doubling production (David 1997; Kelly 2000; Delgado et al. 2003; Costales et al. 2003; Bello 2003; Verburg and Veldkamp 2004; Costales et al. 2007; Stanton et al. 2010) (Fig. 2.3b). Hog inventories are growing at 4.5 % a year on Luzon, and, as in the USA and other industrial countries, farm numbers there are declining and heads-per-farm increasing (Catelo et al. 2008). Production continues to consolidate and capitalize with an eye toward exporting output across the ASEAN Free Trade Area.

Kelly (1998) describes the resulting shifts in landscape,

Large swathes of irrigated agricultural land in the 'rice bowl' provinces of the Central Luzon and Southern Tagalog regions have been converted to a variety of urban and industrial uses: export processing zones and industrial estates; institutions such as hospitals and universities; leisure landscapes such as golf courses, resorts and theme parks; and, most significantly in terms of the area involved, residential sub-divisions. The result is a reworking of the social and economic, as well as the physical, landscape of formerly 'rural' areas, such that even within the same household the urban-industrial economy might co-exist with agricultural production.

Land conversion, commoditized food chains, and deforestation have broadened the interfaces wild REBOV reservoirs, livestock, and human populations apparently share.

Shively and collaborators (Coxhead et al. 1999; Shively 2001; Coxhead and Jayasuriya 2002; Shively and Pagiola 2001, 2004) have argued policies of import substitution industrialization, aimed at replacing foreign imports with domestic production in capital-intensive sectors, have by their weak growth and underemployment prompted increasing smallholder land colonization, agricultural intensification, and deforestation. But expanding imports, land leasing, and foreign direct investment; lowering duties on corn for internationally connected hog producers; and contract husbandry have together proven no panacea either. Farm consolidation, differential farmgate pricing structured by the WTO Agreement on Agriculture at smallholder expense, and 'flexible' zoning for politically connected land expropriation have had detrimental impact on the environment, especially in Central Luzon (Kelly 1998, 2000; Pagiola and Holden 2001; Costales et al. 2003; Bello 2003; Habito 2011; Kelly 2011; Borrás and Franco 2011). The new economic paradigm has spurred speculation on both marginal arable land and primary forest; expanded agriculture into new zones of production; and driven struggling smallholders into cities, pluriactive cycle migration, or further into what forest remains.

Behaviorally plastic frugivore bats are documented to roost in plantation farms that have deforested or disturbed traditional habitats, a transition linked to pathogen spillover into human and hog alike (Chua 2003; Halpin and Mungall 2007; Luby et al. 2009, Leroy et al. 2009, Shafie et al. 2011). Deforestation is particularly severe in the Philippines, producing limited vegetative cover and patchy food distributions for fruit bats, mitigated in part by among other crops oil palm, ethanol cassava, taro,

coconut, and orchards grown directly on livestock operations for fodder and shade (Moog 1991; Shively 2001; Verburg and Veldkamp 2004; Heaney et al. 2005, Jones et al. 2009, Borras and Franco 2011; Sedlock et al. 2008).

By this point REBOV may also be endemic to the regional hog food chain, alongside a growing list of pathogens, including *Actinobacillus pleuropneumoniae*, atrophic rhinitis, classical swine fever, *Haemophilus parasuis*, *Mycoplasma hyopneumoniae*, porcine circovirus type 2, type 2 porcine reproductive and respiratory syndrome virus, pseudorabies virus, swine influenza virus, hepatitis E, and a variety of septicemia, hemophillosis, coccidiosis, nematodiasis, gastroenteritis, and parvoviral infections (Tateyama et al. 2000; Drew 2011; Khan et al. 2013; Alawneh et al. 2014; Ng and Rivera 2014).

REBOV was recently reported in commercial hog in China, where half of the world's domesticated pigs live. Pan et al. (2014) report REBOV detected on three hog farms 35 km from each other in Shanghai, sharing 96–99 % sequence similarity with two Philippine variants and the Manaoag strain the Shanghai isolates' immediate basal ancestor. The recycled inputs, overlapping cohorts, and extensive value-added networks of typical regional hog production may promote infection across farms, even within the all-in/all-out model of production (Graham et al. 2008; Atkins et al. 2010; Otte and Grace 2013). Bausch (2011) additionally hypothesized refrigeration associated with commercial hog processing could preserve the fragile virus. On the other hand, REBOV has also been reported in China in Leschenault's rousette fruit bats (*Rousettus leschenaulti*) (Yuan et al. 2012).

The two epizootic transitions—repeated spillover across a wider agroecological interface and endemicity in the hog chain—need not be mutually exclusive. Carroll et al. (2013) hypothesize REBOV underwent a genetic bottleneck in the Philippines consistent with both a decline in a host reservoir driven by deforestation and a founder event in newly commercialized hogs.

## 2.5 Pandemic Ebola by Way of Commodity Livestock?

What of any direct epidemiological fallout, other than the potential economic impacts of such a new agroepizootology? REBOV has infected humans handling hogs along the value-added chain, but to date to no clinical danger or measurable human-to-human transmission.

On the other hand, the ZEBOV outbreak in West Africa signaled virulent variants can shift from limited spillovers in deep forest to regional, and potentially pandemic, H2H spread. Indeed, human history is marked by such epidemiological transitions. Diphtheria, influenza, measles, plague, pertussis, tuberculosis, cholera, HIV/AIDS, dengue, malaria, and yellow fever, among other examples, are all human diseases that originated in shifts in host reservoir, characteristic area, functional ecology, and/or modes of transmission (Pearce-Duvet 2006; Wolfe et al. 2007; Kock et al. 2012; R.G. Wallace et al. 2015).

In that context, a growing body of experimental research is closely exploring the extent to which Ebola can transmit from hogs to humans. Marsh et al. (2011) challenged 5-week-old pigs with  $10^6$  TCID<sub>50</sub> of REBOV-08 by the oronasal route and, in a separate experiment, subcutaneously. The team detected viral shedding from the nasopharynx 2–8 days post-exposure in pigs with subclinical infection and 6–8 days from the rectum, with infiltration across most other organs and tissues, producing gross abnormalities in lymphoid and respiratory systems.

Kobinger and Weingartl's group (Kobinger et al. 2011; Weingartl et al. 2012; Nfon et al. 2013; Weingartl et al. 2013) showed ZEBOV can infect and sicken pigs, which can transmit the virus to cynomolgus macaques—standing in for humans—without direct contact, by airborne or sapronotic transmission. The pigs suffered a respiratory syndrome that can be mistaken for other porcine diseases, characterized by the dysregulated recruitment of non-infected lymphocytes, upregulated cytokines, proapoptotic induction, pulmonary consolidation, and hemorrhagic alveolitis. In contrast, the macaques presented no respiratory symptoms but suffered systemic collapse associated with hemorrhagic fever and a dysregulated cytokine response.

Even as ZEBOV is clearly the more virulent of the two Ebola types, REBOV infections are nearly as deadly when hogs are coinfectd with other pathogens associated with the commodity chain, including off the long list in the previous section, PRRSV and porcine circovirus type 2 (Barrette et al. 2009; Carroll et al. 2013). Other combinations of porcine respiratory co-infection have been shown to induce synergistic morbidity (Stark 2000; Neumann et al. 2005). The molecular mechanisms remain to be worked out, but rich networks of molecular reciprocal activation have been identified in other pathogen guilds, including between HIV and many of its opportunistic infections (Lehrnbecher et al. 2000; Lawn 2004; Guo et al. 2004; Sun et al. 2005).

The steps in molecular evolution on one possible path to REBOV pathogenicity were recently mapped. Pappalardo et al. (2016) computationally analyzed 196 Ebolavirus genomes for Specificity Determining Positions across the virus's nine proteins, including protein–protein interface sites and enzyme active sites, that differentiate the Ebola species. REBOV, the only species non-pathogenic to humans to date, differed from its pathogenic counterparts in a GP SDP and multiple SDP across structural proteins VP24, VP30, VP35, and VP40.

Pappalardo et al. noted eight structurally identifiable SDP differed across Ebolavirus in membrane-associated protein VP24, which is involved in the formation of the viral nucleocapsid and regulating viral replication. The protein is an antagonist to host interferon, binding to karyopherins  $\alpha 1$ ,  $\alpha 5$ , and  $\alpha 6$ , and critical in Ebola adaptation to novel hosts. The team found seven SDPs on the same VP24 face, implying an interface with other viral proteins or host cell. Mutations to two SDPs have been shown to block KPNA5 ( $\alpha 5$ ) interactions, critical to host adaptation, and are adjacent to a mutation that reduces interferon antagonism (Xu et al. 2014; Ilinykh et al. 2015). A third replacement a hundred amino acids down breaks a hydrogen bond, further destabilizing the protein. REBOV pathogenicity appeared impaired on all accounts here.



Pappalardo et al. account for other species-defining differences. In transcriptional co-factor VP30 the removal of two hydrogen bonds in C-terminal dimer formation and the burial of two functional residues may change the balance of transcription/replication in REBOV. Among its multiple functions, VP35 antagonizes interferon signaling by binding to host dsRNA. REBOV VP35 is overall more stable, with a reduced affinity for dsRNA. Pappalardo et al. show two SDP here: one near the linker, the other at the dimer interface, the latter shortening VP35's aspartate side chain, in all likelihood increasing the distances among nearby residues sharing hydrogen bonds, destabilizing the dimer complex. One SDP introduces backbone flexibility and destabilization to the octameric form of VP40 involved in viral transcription. Another SDP likely breaks and shortens one of VP40's helices and changes its hydrophobic core.

The authors conclude REBOV is only a few SDP shifts from evolving human pathogenicity. Although virulence may arise by any number of means, the comparison maps one short 'path to victory' for a dangerous REBOV.

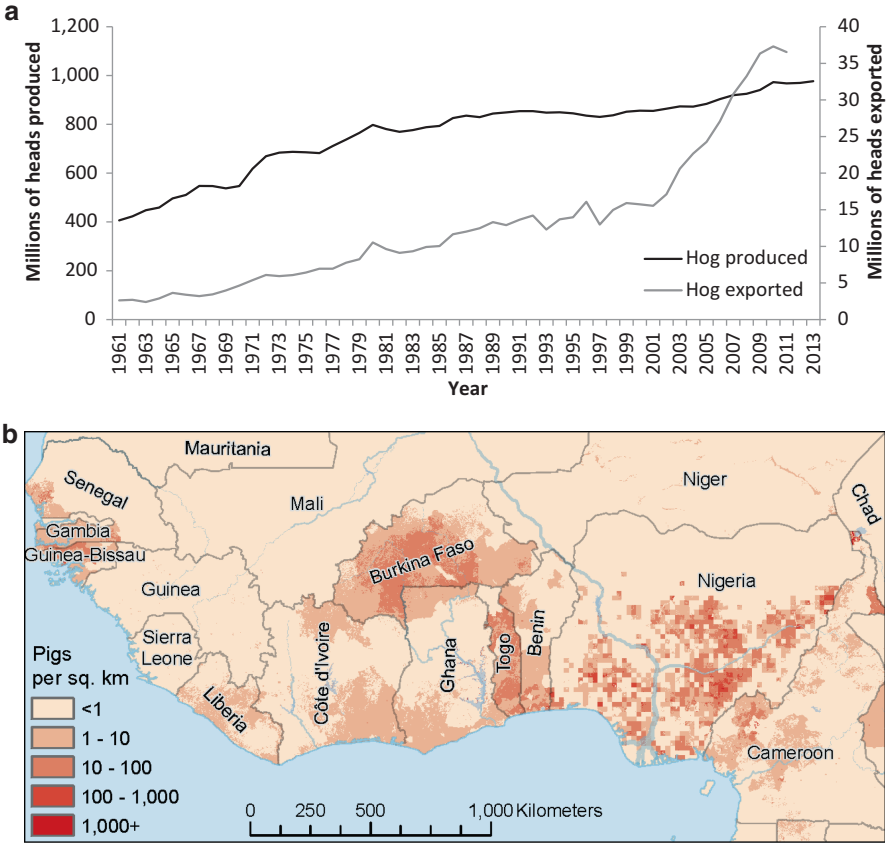
The results so far together suggest virulent Ebola and human spillover across a global hog value chain expanding in size and extent a distinct possibility (Drew 2011) (Fig. 2.4a). As Marsh et al. (2011) conclude of REBOV:

The evidence of virus shedding and replication of virus in internal organs [of swine] in the absence of clinical disease represents a potential source of infection to farm, veterinary, and abattoir workers. This appears to be an unprecedented emergence of filovirus infection in a new host that may have important biosecurity implications for both livestock health and emergence in the human food chain. Although REBOV has not been seen to result in any human disease, the basis for this observed attenuation remains unknown. The consequence of REBOV becoming pathogenic in humans is serious, and ongoing undetected infections and replication in pigs and other animals with REBOV may result in the emergence of viruses that are more pathogenic in humans and/or livestock.

Bausch (2011) adds:

...EBOV pathogenicity can be enhanced by serial passage in animals and cell culture... A similar result through unintentional serial passage in pigs is not out of the question... [T]he laboratory findings of Kobinger et al., combined with the previous results of field investigations in the Philippines...highlight the possibility of EBOV as a foodborne pathogen. This is cause for consideration, further scientific study, and prudent surveillance and prevention measures in the livestock industry in implicated areas of the world.

These preliminary results suggest the epidemiological stakes are high indeed. Multiple modes by which animal and public health might successfully intervene should be explored. Further study, then, especially in the light of environmental stochasticity's intrinsic impact on epidemic growth, need be extended to the broader agro-economic context out of which new Ebola variants emerge and, as we explore in the next section, may perhaps be controlled.



**Fig. 2.4** Expanding hog sector. (a) Global hog production and international exports, 1961–2013. UNFAO data. (b) Distribution of hog across West Africa, 2006 (pigs/km-sq) (Robinson et al. 2014). *Projection:* Cylindrical equal area with a standard parallel of 10°. East Africa—across Uganda, Rwanda, and parts of Kenya—is characterized by comparable intensification.

## 2.6 An Agroecological Logic Gate for Epidemic Control

In an effort to reimagine the agroeconomic geographies that propagate pathogens, as Ebola apparently instantiates in West Africa and the Philippines, we need aim to empirically define a critical ecosystemic “temperature” at which an outbreak is instead “sterilized”. Specifically we need to incorporate measures of the emergent “friction” that agroeconomic and policy barriers impose upon propagating pathogens (Levin et al. 1998; R. Wallace 2002; Meentemeyer et al. 2012; Boyd et al. 2013).

There are complications, however. As discussed earlier, the noise terms in Eqs. (2.1) and (2.7), the  $dW_t$ , are not necessarily simple white noise characterized by a single intensity parameter. Indeed, the relatively straightforward treatment

of fractional Brownian motion in R.G. Wallace et al. (2014) implies that such a “temperature” is likely to be a composite function of the intensity and color of both temporal and spatial noise. Some conceptual simplification, however, is possible via an extension of the embedding mathematics to include Levy-type jumps—random walks with step-lengths drawn from a heavy-tailed probability distribution.

Equations (2.1) and (2.7) are expressed in terms of classic white noise having the simple quadratic variation  $[W_t, W_t]_t = \sigma^2 t$ , where  $\sigma$  is the single available parameter, the noise magnitude (Oksendal 2010; Protter 1990). The arguments of Eqs. (2.1) and (2.7) can be extended to other kinds of noise, having arbitrary quadratic variation and discontinuous stochastic jumps, since they have the characteristic form

$$dZ_t = Z_{t-} dY_t \quad (2.9)$$

where  $Y_t$  is a stochastic process, and  $t-$  indicates left-continuous. Letting  $\Delta Y_t = Y_t - Y_{t-}$ , representing the jump process, the generalization is via the Doleans–Dade exponential, the solution of a class of stochastic differential equations defined by a semimartingale of bounded variation (Protter 1990). Then

$$Z_t = \exp(Y_t - 1/2[Y_t, Y_t]_t^C) \prod_{s \leq t} (1 + \Delta Y_s) \exp(-\Delta Y_s) \quad (2.10)$$

where  $[Y_t, Y_t]_t^C$  is the path-by-path continuous part of the quadratic variation of  $Y_t$ . This is written as

$$[Y_t, Y_t]_t^C = [Y_t, Y_t] - \sum_{0 \leq s \leq t} (\Delta Y_s)^2 \quad (2.11)$$

The product term in Eq. (2.10), with (Levy-like) jump processes having nonzero  $\Delta$ , converges.

The essential point emerging from the formalism is that, if the structurally imposed environmental stochasticity  $[Y_t, Y_t]_t^C$  is monotonically increasing in time at a greater rate than  $Y_t$ , the exponential factor  $\exp(Y_t - 1/2[Y_t, Y_t]_t^C)$  in Eq. (2.10) drives the system toward extinction. The Appendix examines more complicated multidimensional processes in which (relatively) low level endemic infection can be either driven to epidemic explosion or, the better outcome, stabilized.

Setting Levy-like jumps aside for the moment, it is possible to extend Eq. (2.9) as

$$dZ_t = dL_t + Z_t dY_t \quad (2.12)$$

where  $L_t$  is a generic stochastic “seeding” process, here representing a pathogen’s incursion from outside the system of interest. Following Protter (1990), if the stochastic “frictional” variability represented by the quadratic variation  $[Y_t, Y_t]$  is sufficiently large, there will be no explosion of infection, which will, at worst, asymptotically converge on the imported cases, i.e.,  $Z_t \rightarrow L_t$ .

This is a striking outcome in a broadly comprehensive model.

Remarkably, these results can be interpreted in terms of the Data Rate Theorem, which imposes necessary conditions on information transmission and links control theory with information theory:

An outbreak can be conceptualized as a signal sent through a channel, whose topology is embodied by the local ecology. Environmental stochasticity—noise against the signal—can disrupt the broadcast. That is,  $d[Y_t, Y_t]^C/dt$  (or, for Eq. (2.12),  $d[Y_t, Y_t]/dt$ ) can be viewed as an imposed control signal on a highly nonlinear “pathogen factory” analog logic gate.

A logical operation, controlled here explicitly by agroeconomic policy, is performed on multiple inputs, in this case a network of agroecological relationships, to produce a single logical output, here the state of an epizootic population.

The Data Rate Theorem, a generalization of the classic Bode Integral Theorem for linear control systems, describes the stability of feedback control under data rate constraints (Nair et al. 2007). What is the smallest feedback data rate above which such an unstable dynamical system can be stabilized? In our context, what is the smallest amount of policy-determined environmental stochasticity we need to stabilize or prevent an expanding outbreak?

Given a noise-free data link between a discrete linear “factory” and its controller, unstable modes can be stabilized only if the feedback data rate  $\mathcal{H}$  is greater than the rate of “topological information” generated by the unstable system. For the simplest incarnation, if the linear matrix equation of the “factory” is of the form  $x_{t+1} = \mathbf{A}x_t + \dots$ , where  $x_t$  is the  $n$ -dimensional state vector at time  $t$ , then the necessary condition for stabilizability is

$$\mathcal{H} > \log[|\det \mathbf{A}^u|] \quad (2.13)$$

where  $\det$  is the determinant and  $\mathbf{A}^u$  is the decoupled unstable component of  $\mathbf{A}$ ; that is, the part with eigenvalues  $\geq 1$ . Thus there is a critical positive data rate below which there does not exist *any* quantization and control scheme able to stabilize an unstable system. Here,  $\mathcal{H} \equiv 1/2d[Y_t, Y_t]^C/dt$  (or  $1/2d[Y_t, Y_t]/dt$ ), and a sufficient “data rate” condition can be expressed, applying the mean value theorem for monotonicity, as

$$\mathcal{H} = \frac{1}{2}d[Y_t, Y_t]^C/dt > dY_t/dt \quad (2.14)$$

A somewhat different approach, deriving a policy driven, fully information-theoretic  $\mathcal{H}$  via large deviations theory, is outlined in Appendix.

As REBOV’s origins in the Philippines clearly demonstrate, the dynamics of infection initiation and spread—including structured spatial diffusion and stochastic jumps—may be quite complicated. But a sufficient agroecological control signal, produced by adequate variation in space, time, and mode of production—high enough “noise”—will almost always stabilize and control pathogen outbreaks, largely limiting them to either imported cases or intermittent spillovers that burn out in the face of the area’s ecological resilience and/or public health capacity

(Hogerwerf et al. 2010; Lewnard et al. 2014). However frequent or deadly such outbreaks—think up to this point Ebola in Central Africa—they are largely constrained to local domains.

## 2.7 The Costs of Biocontrol and Containment

As our description of REBOV in the Philippines intimated, the crux of epidemic control clearly extends out beyond susceptible-infectious-removed dynamics, the effects of the microeconomics of livestock production on disease (e.g., Boni et al. 2013; Allen and Lavau 2014), or even the agroeconomically informed spatial modeling with which we began, however, illuminating all such modeling may be. Causality extends out into the way the fabric of the global economy interpenetrates regional agroepizootology (Wilson et al. 1994; R. Wallace and R.G. Wallace 2015; R.G. Wallace et al. 2015).

We take a step in that direction, relating epizootology and economic policy in a more explicit fashion. We apply the Black–Scholes approach to option pricing in finance toward modeling the cost in resources needed to control the agroecological logic gate introduced in the previous section (by which pathogen populations explode or stabilize) (Black and Scholes 1973).

Let  $\mathcal{H}_t$  be the value of  $\frac{1}{2}d[Y_t, Y_t]^C/dt$ , our environmental stochasticity, or the value of the rate function in a large deviations analysis of the Mathematical Appendix at time  $t$ . Under conditions of both white noise and macroscopic volatility, a general relation can be written as

$$d\mathcal{H}_t = f(t, \mathcal{H}_t)dt + b\mathcal{H}_t dW_t \quad (2.15)$$

where the magnitude of the noise *in this iterated system* is now expressed as  $b$  to avoid confusion with the earlier development.

Let  $M(\mathcal{H}_t, t)$  represent the rate of available resources—direct short- and long-term financial investment plus “opportunity costs” from those expenditures—needed to achieve  $\mathcal{H}_t$  at time  $t$ , and expand using the Ito chain rule,

$$\begin{aligned} dM_t = & \left[ \partial M / \partial t + f(\mathcal{H}_t, t) \partial M / \partial \mathcal{H} + \frac{1}{2} b^2 \mathcal{H}_t^2 \partial^2 M / \partial \mathcal{H}^2 \right] dt \\ & + [b \mathcal{H}_t \partial M / \partial \mathcal{H}] dW_t \end{aligned} \quad (2.16)$$

As in the original Black–Scholes model—which uses different terminology—we define  $\mathcal{L}$  as the Legendre transform (Pettini 2007) of  $M$ , taking the involutive transformation of function  $M$  to produce

$$\mathcal{L} = -M + \mathcal{H} \partial M / \partial \mathcal{H} \quad (2.17)$$

Using the heuristic of replacing  $dX$  with  $\Delta X$  in these expressions, and applying the results of Eq. (2.16), gives the relation

$$\Delta \mathcal{L} = \left( -\partial M / \partial t - \frac{1}{2} b^2 \mathcal{H}^2 \partial^2 M / \partial \mathcal{H}^2 \right) \Delta t \quad (2.18)$$

Analogous to the classic Black–Scholes calculation, the terms in  $f$  and  $dW_t$  cancel out, so that the effects of noise are subsumed in the Ito correction involving  $b$ . This invokes powerful assumptions of regularity that may be violated. Matters then revolve about model robustness in the face of such violation.

As the Legendre transform of  $M$ ,  $\mathcal{L}$  is a kind of entropy that can be expected to reach a constant rate of production at nonequilibrium steady state (nss). Then  $\Delta \mathcal{L} / \Delta t = C \geq 0$ ,  $\partial M / \partial t = 0$ , so that

$$-\frac{1}{2} b^2 \mathcal{H}^2 \partial^2 M / \partial \mathcal{H}^2 = C \quad (2.19)$$

The solution is

$$M_{\text{nss}} = \frac{2C}{b^2} \log[\mathcal{H}_{\text{nss}}] + \kappa_1 \mathcal{H}_{\text{nss}} + \kappa_2 \quad (2.20)$$

at nonequilibrium steady state.

In this form the treatment suggests two basic policy options. If  $\kappa_1 = 0$ , then *the cost of epidemic control grows only as the log of the policy-driven variate  $\mathcal{H}$* . If  $\kappa_1 > 0$ , then the cost will be dominated by linear growth in  $\mathcal{H}$ .

The inference is that the overall financial cost of epidemic prevention and control depends on agro-economic policy’s impact on environmental stochasticity. In the context of this analysis, regional planning that introduces “frictional” variation in space, time, and mode on agroecology, raising  $b$  in Eq. (2.20), controls costs specific to an outbreak at a rate much lower than linear. In contrast, the biocontainment option, represented by the linear-dominated form of Eq. (2.20), produces a very large constant of proportionality,  $\kappa_1$ .

The inherently explosive epizootologies of commodity agricultures—however, frequently biocontained—appear exorbitantly expensive as a matter of first principle.

## 2.8 Discussion and Conclusions

The ZEBOV virus behind West Africa’s Ebola outbreak appears a commonplace phenotype, with a typical case fatality rate, incubation period, and serial interval (WHO Ebola Response Team 2014). How, then, to explain the unprecedented outbreak? While the virus did not fundamentally change, Africa did (Gatherer 2015). The region’s economically driven transformations in land use appears to have changed the agro-economic matrices through which environmental stochasticity acts

as an inherent brake upon pathogen momentum at the population level (Bausch and Schwarz 2014; R.G. Wallace et al. 2014). Commodity agriculture's spatial and functional expansions may by virtue of deforestation and monocultivation destroy many a pathogen. But in stripping out the agroecological "friction" diverse functional geographies impose on systems of potential susceptibles, such production may liberate many another pathogen, especially those circulating among reservoir hosts that adapt to the new agriculture (e.g., monkeys, birds, and bats).

The outbreak of REBOV in hog in 2008, marking another such agroecological transition, was initially greeted with as vociferous if less widespread public and scientific alarm (WHO 2009; Cyranoski 2009). While public interest subsequently receded, researchers across disciplines began a number of research lines carefully investigating REBOV's molecular, clinical, and epidemiological courses (Barrette et al. 2009; Bausch 2011; Kobinger et al. 2011; Marsh et al. 2011; Miranda and Miranda 2011; Sayama et al. 2012; Weingartl et al. 2012; Nfon et al. 2013; Pappalardo et al. 2016). The reports are punctuated with urgent if also characteristically conditional warnings as to both the virus's endemicity in the food chain and its potential for evolving virulent human-specific phenotypes.

By a series of stochastic models we aimed here at integrating the problematics REBOV and other Ebola species present across biocultural domains, including their molecular, geographic, and economic contexts. The punctuated emergence of a spreading pathogen via the operation of the kind of policy-driven biological logic gate we inferred here may affect not only the timing and extent of individual outbreaks, as in R.G. Wallace et al. (2014), but may embody a generalized pathogen response to agroecological pressures that, in addition to shifts in spatial patterns, select for new ecotypes and modes of transmission.

The impacts appear to extend to the financial costs of intervention. Systems that minimize environmental stochasticity's impact upon pathogen population growth incur explosive costs when new variants successfully emerge. Whether such a framework can explain any single outbreak remains to be tested. For instance, Bartsch et al. (2015) estimate that the direct societal costs of all Ebola cases in Guinea, Liberia, and Sierra Leone through mid-December 2014 range from USD \$82 million to \$356 million. That is a large sum, considering West Africa's exchange rates. In effect, at the risk of reductionist utilitarianism, area-specific environmental stochasticity may represent another valuable ecosystem service humanity is presently scuttling in favor of short-term earnings, one forest plot at a time (Farber et al. 2002; Zhang et al. 2007).

The model series we present here is inductive in nature—an important caveat. In the spirit of mathematical ecologist Evelyn Pielou (1977), the models aim at helping raise questions that appear largely absent from the disease literature. Specifically we aim at identifying the necessary conditions under which broad classes of zoonotic pathogens respond to anthropogenic shifts. The simplicity of the models presented here speaks to general conditions across systems, regardless of their biologies. Namely, pathogen fitness is *fundamentally* integrated with its population biological (and sociological) context. Agroecology is an epidemiological cause of foundational impact and no mere second-order complication (Hinchliffe 2015).



Ostensibly adjunct models could be conditionalized in various ways for testing specific systems, including, in this case, REBOV's statistical economic geographies and social phylogeographies, analyses of the value chains the virus passes through, dynamic modeling of its socio-ecological niches, an economically informed life history model of the evolution of its virulence, cost differentials across interventions, and the Markov travel analysis that we sketched around Eq. (2.5) (e.g., R. Wallace et al. 1999; Mayer 2000; R.G. Wallace 2004; Walsh et al. 2005; Atherstone et al. 2014; Lewnard et al. 2014; Pigott et al. 2014).

Following R.G. Wallace et al. (2014) and R. Wallace and R.G. Wallace (2015), one might use a number of measures of  $\sigma$  in Eqs. (2.2) and (2.7), of the structure of environmental stochasticity  $[Y_t, Y_t]_t^C$  in Eq. (2.10), or  $[Y_t, Y_t]$  associated with Eq. (2.12). These could include neoliberal or neocolonial expropriation, or the elimination of traditional farming strategies that previously isolated pathogens from livestock, wildlife, and humans at regional spatial scales. See R. Wallace (2014) for a more general exploration of canonical failure modes afflicting what are essentially cognitively "farmed" systems, here with a "crop" represented by an epidemic outbreak that one wishes to minimize. R. Wallace (2015) reexpresses these considerations explicitly in terms of an analog biological logic gate or Boolean-like circuit.

One might operationalize such broadly painted political economies in absolute geographies such as hectares leased to multinationals per total agricultural area, the number of farms consolidated in the past 10 years, and agricultural exports. On the other hand, agroeconomies are marked by relational geographies across industrial sectors (Bergmann 2013a,b; R.G. Wallace et al. 2015). Landscapes are entrained by transnational commodity chains and circuits of capital, including financial and productive circuits, with critical local effects. Products from globalized croplands, forests, or pastures eventually contribute to consumption or capital accumulation in other countries. Other landscapes are enmeshed primarily within local circuits of production and exchange.

Bergmann (2013b) extends analysis beyond characterizing landscapes that directly produce traditional agricultural exports to identifying the forests and fields that are part of commodity webs supporting export-oriented development, producing goods or services for international markets. He further differentiates foreign consumption/accumulation of "direct" agricultural goods; processed agricultural goods; manufactured goods as far afield as electronics and vehicles; and services, including air transport, insurance, and education. How such circuits of capital structure disease ecologies is a matter of ongoing research.

Such broad impact might produce large-scale ecosystemic shifts that set off spikes in pathogen population or in their cladogenesis, inducing new patterns of transmission, virulence, and endemicity (e.g., Carroll et al. 2013). That is, changes in policy or socioeconomic structure can trigger a large-scale biological logic gate and "desterilize" a natural or human ecosystem in which a pathogen has been traditionally held at a low endemic level, or simply had not previously evolved.

REBOV's emergence may represent such a canonical example. Luzon's agroeconomic geography appears to have undertaken a phase change from

rural, smallholder-based production into a conurbanized daisy chain of advanced commercial operations that doubled island hog (Kelly 2000; Delgado et al. 2003; Costales et al. 2003, 2007; Catelo et al. 2008). In conjunction with deforestation, such a vertically integrated expansion appears to have broadened the interface livestock and REBOV-bearing bats share (Shively 2001; Sedlock et al. 2008; Verburg and Veldkamp 2004; Heaney et al. 2005; Borrás and Franco 2011; Sedlock et al. 2011; Carroll et al. 2013). As per Bergmann et al.'s program, such a modern-day disease ecology is likely neither unconnected from the rest of the world nor erased by globalization (R.G. Wallace et al. 2010). The socioecological environments out of which REBOV and other Ebola species are evolving into food commodity pathogens are often the complex and layered products of past and present and of global and local impacts.

Indeed, a growing array of pathogens appear characterized by such multi-order anthropogenic origins, often deeply embedded within neoliberal capitalism (R.G. Wallace et al. 2015). Chua (2003) describes a similar if locale-specific transition for Nipah virus in Malaysia:

... [A]vailable data... suggest that a complex interplay of multiple factors led to the spillage of the virus from its natural reservoir host into the domestic pig population with subsequent spread to humans... [O]ver the last two decades, the forest habitat of... fruitbats... in Southeast Asia has been substantially reduced by deforestation for pulpwood and industrial plantation... [A series of weather events then] led to acute reduction in the availability of flowering and fruiting forest trees for foraging by flying-foxes in their already shrinking habitat... This culminated in an unprecedented encroachment of flying-foxes into cultivated fruit orchards in the initial outbreak area... These anthropogenic events, coupled with the location of piggeries in orchards and the design of pigsties in the index farms allowed transmission of a novel paramyxovirus from its reservoir host to the domestic pig and ultimately to the human population and other domestic animals.

We argue in contrast, by their diversity in time, space and mode, traditional and conservation agricultures can create numerous functional barriers—a kind of sterilizing temperature—limiting pathogen evolution and spread (Diaz et al. 2006; Burdon and Thrall 2008; Perfecto and Vandermeer 2010; R. Wallace and R.G. Wallace 2015).

For instance, pastoralist livestock production, until recently supplying 67 % of Kenya's available meat, promotes wildlife conservation in the semi-arid Sub-Saharan (Kock 2005; Kock et al. 2010). The last of the unrestricted ungulate populations are associated with such systems. Disease plays a critical role for both semi-arid wildlife and livestock, including indirectly multiplying the impact of other pressures such as predation. Foot-and-mouth disease and malignant catarrhal fever, among other diseases, selected for strict limits on land use and animal movement.

But growing awareness of the complexities of the wildlife-livestock interface in association with a management philosophy erring on the side of integration have revised thinking around disease control in rangelands. First, there appears little actual physical contact between livestock and wildlife, even in integrated semi-arid systems. The separation is driven by the animals' own niche behavior,

leaving disease spillover between wild and domesticated animals largely sapronotic and vector-borne (e.g., anthrax, Rift Valley fever, and theileriosis). Accumulating molecular evidence indicates that many of the diseases wildlife and livestock putatively transmit to each other are in fact host-specific products of vicariant cospeciation, although host switching does intermittently occur (Huyse et al. 2005; Foster et al. 2009; Widmer and Akiyoshi 2010; Ruecker et al. 2012; Hoberg and Brooks 2015).

Diseases that have had transcontinental impact (e.g., rinderpest, bovine tuberculosis) have been imported from abroad and are density-dependent, part and parcel of millenia of anthropogenic disruption (Kock et al. 2010). In these cases, vaccination and fencing have been deployed. Control has also now turned to separating livestock into small “export zones” in which livestock are fattened and from which wildlife are excluded, synergistically supporting pastoralist pluriactivity. Behavioral adaptations are also introduced by the pastoralist themselves. Kock (2005) describes one such adaptation controlling malignant catarrhal fever:

Generally, the livestock keepers will avoid calving grounds, but when they have no choice the grazing strategy of the pastoralists shows considerable understanding of the epidemiology of the disease. The virus is highly sensitive to drying, heat, and ultraviolet light, so under natural conditions the pastoralists have learned that since wildebeest calve at night, by 10:00 a.m. the pasture is sterile (in terms of MCF) and infection can be avoided.

The economies of scale of capitalized production remove such functional diversity and are paradoxically extended to those pathogens its agrosystems select, even in systems exercising model biocontrol (Brown 2004; R.G. Wallace 2009; Hogerwerf et al. 2010).

For Ebola, the 2008 REBOV outbreak and the transmission studies of deadly ZEBOV to and between pigs together imply commoditized husbandry is open to devolving into a “plague plantations” across a large part of the world, particularly in the USA, Europe, and China, but increasingly other states, as the Philippines demonstrates, where intensive confined feedlots have come to dominate animal protein production. Commodity hog is producing industrial densities in growing areas of West and East Africa alike, including in Guinea-Bissau, Burkina Faso, Togo, Nigeria, Rwanda, and Uganda, several of which have hosted Ebola outbreaks (Atherstone et al. 2014) (Fig. 2.4b). Ebola outbreaks in Uganda have taken place in areas with high pig density (Weingartl et al. 2013). We hypothesize REBOV’s transition in the Philippines foreshadows the kind of agroeconomic domain shift Ebola may already be undertaking in Africa.

One need be concerned about whatever hog mortality may result, of course, with clear implications for human health (Bausch 2011). But subclinical infections are themselves emblematic of a growing prevalence, especially with REBOV now in China, where half the world’s hog population is raised. The greater the population infected, the larger the “laboratory” in which Ebola can recombine, coinfect with other prevalent industrial pathogens, and experiment with human-specific phenotypes (Frank 1994; R.G. Wallace 2009; Mennerat et al. 2010; Drew 2011; FAO 2013b; Fuller et al. 2013).

Contrary to the sector's operational premises, pathogens evolve, or, by shifting host population states alone, are maneuvered into new evolutionary stable strategies (Levin 1968; Lipsitch and Nowak 1995; R.G. Wallace et al. 2014). Hog's expanding economic geographies may produce a "critical connectivity" across farms at multiple spatial scales that changes the rules under which the evolution of virulence takes place, permitting virulent strains previously selected against by a cap on available susceptibles to invade local variants (Boots et al. 2004; Messenger and Ostling 2009; Atkins et al. 2010).

The damage imposed by such economies of scale is routinely described in the industrial literature, with considerable attention given to the means by which to offload associated costs (e.g., Arthur and Albers 2003; Fulton 2006). By a moral hazard of existential significance, the overhead is externalized to the occupational hazards of production; livestock health and welfare; wildlife by way of deforestation, pollution and disease spillover; local environs polluted by manure runoff; consumer health via food-borne pathogens and metabolic disorders; and damage to transportation and health infrastructure (Singer 2005; Catelo et al. 2008; R.G. Wallace and Kock 2012; Leonard 2014; Genoways 2014). Such costs are routinely floated by taxpayers and governance across administrative units. Subsidies and bailouts benefit individual companies exercising political influence but are also deployed as a matter of national economic policy (R.G. Wallace 2009, RG Wallace 2012).

Bello (2003), for instance, describes a gambit by which the very efforts to reverse Filipino protectionism were parlayed into a means by which to block outbound domestic exports on epizootic grounds:

Even Australia, an ally of the Philippines in the so-called Cairns Group, a grouping of developed and developing agro-exporting countries, beat up on the Philippines by invoking sanitary and phytosanitary standards, a standard Washington tactic. In mid-2002, after years of being petitioned to admit Philippine cavendish bananas, the Australian government decided against the import. The ostensible reason was the risk of the Philippine banana carrying pests and diseases that could ruin the Australian banana industry. Yet the Philippine bananas had been shipped since the sixties to countries with high quarantine standards, including Japan and New Zealand.

Much of the academic and nongovernmental response to the disease challenges such production raises appears two-pronged in nature (R.G. Wallace et al. 2015). The first approach is a multilevel analysis described as broad in scope that also omits the ideological commitments behind the shifts in land use driving emergent disease. Plowright et al. (2015), for instance, layer the causes underlying the emergence of bat zoonotics from virion and immune response up through community ecology and anthropogenic context. Yet such a comprehensive analysis includes a telling omission:

Coincident with [expanding urban and periurban land use and increasing presence of horses] is another [factor]: the range of black flying-fox is expanding rapidly southward at rates faster than projected on the basis of climate change scenarios... Black flying-fox have a stronger association with Hendra virus spillover events than other flying-fox species..., and may be more likely to feed on the marginal foods that support resident populations in

anthropogenic landscapes. . . Thus, the range shift of black flying-fox may contribute to the increasing incidence and recent southern extension of Hendra virus spillover events in the subtropics.

The underlying neoliberal shifts in land markets, agricultural consolidation, and biosecurity that are driving bat dynamics in the Hendra zone are left unremarked upon (McKenzie 2011; Beer 2012; Higgins et al. 2012; Maye et al. 2012, and Lawrence et al. 2013). Plowright et al. avoid even “deforestation” in spite of the evidence, including accumulated elsewhere by several co-authors, linking forest loss to bat dynamics and Hendra (Field et al. 2001; Daszak et al. 2006; McFarlane et al. 2011).

The second archetypal response gives the impression of convergent accommodation (e.g., Khan et al. 2013). One Health proponents Morse et al. (2013), advocating wildlife, livestock, and human health be simultaneously addressed, appear to frame intervention around *assisting* such development:

The challenge to true pandemic prevention (and pre-emption) is how to address the underlying drivers that are essentially ecological (e.g., juxtaposition of livestock production and wildlife populations) or occur on large spatial scales because of economic activity (e.g., change in land use related to development of tropical forests). . . Incentives for industries with roles in activities that propagate pandemics could be linked to development initiatives. For example, concessions in development of logging or mining could include better food supply chains as an alternative to bushmeat hunting, better clinics for migrant workers than are available, and more intensive surveillance of livestock at these crucial interfaces. . . [D]evelopment agencies [are now focusing] on improving individual countries’ abilities to identify new zoonoses early and mitigate quickly any new health threats arising within their borders.

There is a grave irony in replacing bushmeat with the better food supply chains Morse et al. recommend. Clearly de-commoditizing bushmeat associated with logging and mining camps is a step forward in disease control (Bowen-Jones et al. 2003), but should intensive agriculture take its stead, forest pathogens may now regularly access human populations, accelerating the very processes that appear to have led to the ZEBOV outbreak in West Africa.

No one need oppose better clinics for migrant workers to conclude Morse et al. lay individualistic responsibility for cleaning up after such “drivers” on the smallholders and indigenous populations neoliberal agriculture expropriates:

The importance of human exposure throughout the [pandemic] emergence process also suggests that simple behavioural precautions could greatly reduce risk. Risks to hunters, food handlers, and livestock workers from occupational exposure could be reduced in hotspots of emerging infectious diseases through routine sanitation and biosafety precautions. . .

The “hotspots of emerging infectious diseases” now span a vast and synergistic amalgam of expanding commodity plantation and confined feedlot livestock and poultry. Such a system, reducing the ecosystemic “friction” against pathogen emergence below many a region’s controlling threshold, requires a level of biosecure containment beyond the economic margins and practical limits of its own model of production. In this context, advocating washing hands plays as disingenuous political theater, particularly in countries where millions live without access to clean water and much of what is available is pledged to commodity agriculture.

In contrast, a Structural One Health offers an approach that, among other possibilities, explicitly addresses the relationships among transnational circuits of capital, shifts in agroecological landscapes, and the emergence of new diseases (R.G. Wallace et al. 2015). Such foundational context, extending well beyond an epicenter's borders, offers a critical entry into devising successful interventions into human pathogens originating out of agriculture.

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## Mathematical Appendix

### *Epidemic Prevention Farming*

The models above focus primarily on explosive epidemic outbreaks and their containment costs, incorporating as well the influence of sudden Levy jumps. In general, however, endemic levels of infection, fluctuating about some mean, would be expected, and the central question then surrounds the transition between endemic and epidemic modes.

Khasminskii's (1966/2006, 2012, Theorem 4.1) version of Eq. (2.1) provides insight, using a linear first approximation to some complicated, multidimensional, cross-influence function expanded about a quasi-stable equilibrium point. This gives the system of stochastic equations

$$dx_t^i = \sum_{j=1}^l b_i^j x_t^j dt + \sum_{r=1}^n \sum_{j=1}^l \sigma_{i,r}^j x_t^j dW_t^r \quad i = 1, \dots, l \quad (2.21)$$

where  $dW_t^r$  is white noise and the  $b$  and  $\sigma$  terms are constants.

Khasminskii defines two associated matrices,

$$a_{i,j}(x) = \sum_{k,s=1}^l \sum_{r=1}^n \sigma_{i,r}^k \sigma_{j,r}^s x^k x^s, \quad \mathbf{B} = ||b_i^j||$$

under the condition that, for  $\mathbf{A}$ , the inner product condition

$$(\mathbf{A}(x)\alpha, \alpha) \geq m|x|^2|\alpha|^2 \quad (2.22)$$

always holds.

Khasminskii invokes two new variates,  $\lambda = x/|x|$  on the unit sphere, and  $\rho = \log[|x|]$ , expanding  $d\rho_t$  using the Ito chain rule to obtain

$$d\rho_t = \left[ (\mathbf{B}\lambda_t, \lambda_t) + \frac{1}{2} \sum_{i=1}^l a_{i,i}(\lambda_t) - \sum_{i,j=1}^l a_{i,j}(\lambda_t) \lambda_t^i \lambda_t^j \right] dt + \sum_r (\sigma(r) \lambda_t, \lambda_t) dW_t^r \quad (2.23)$$

where  $\sigma(r) = \|\sigma_{i,r}^j\|, i, j = 1, \dots, l$ .

Define

$$Q(\lambda) = (\mathbf{B}\lambda, \lambda) + \frac{1}{2} \sum_{i=1}^l a_{i,i}(\lambda) - \sum_{i,j=1}^l a_{i,j}(\lambda) \lambda^i \lambda^j$$

$$J = \int Q(\lambda) d\lambda$$

where the integral is taken over the unit sphere. (Khasminskii 1966/2006, 2012, Theorem 4.1) shows that, if  $J < 0$ , the complex stochastic process converges to an endemic equilibrium distribution. If  $J > 0$ , then the probability that  $|x_t| \rightarrow \infty$  as  $t \rightarrow \infty$  is 1.

Thus, for any given cross-influence matrix  $\mathbf{B}$ , there is a set of structures defined by the matrix  $\mathbf{A}$ —under the condition of Eq. (2.22)—that will contain a pathogen outbreak to endemic levels. Conversely, given an endemic distribution, sufficient alteration of either the structural matrix  $\mathbf{B}$  or of the “noise” matrix  $\mathbf{A}$  would trigger an epidemic outbreak  $|x_t| \rightarrow \infty$ .

Extension of this result involving jump processes can be found in Khasminskii et al. (2007).

## Large Deviations

Something similar to Eq. (2.14) can be simply derived via a standard large deviations argument.

Following Dembo and Zeitouni (1998), let  $X_1, X_2, \dots, X_n$  be a sequence of independent, standard Normal, real-valued random variables and let

$$S_n = \frac{1}{n} \sum_{j=1}^n X_j \quad (2.24)$$



Since  $S_n$  is again a Normal random variable with zero mean and variance  $1/n$ , for all  $\delta > 0$

$$\lim_{n \rightarrow \infty} P(|S_n| \geq \delta) = 0 \quad (2.25)$$

where  $P$  is the probability that the absolute value of  $S_n$  is greater or equal to  $\delta$ . Some manipulation, however, gives

$$P(|S_n| \geq \delta) = 1 - \frac{1}{\sqrt{2\pi}} \int_{-\delta\sqrt{n}}^{\delta\sqrt{n}} \exp(-x^2/2) dx \quad (2.26)$$

so that

$$\lim_{n \rightarrow \infty} \frac{\log P(|S_n| \geq \delta)}{n} = -\delta^2/2 \quad (2.27)$$

This can be rewritten for large  $n$  as

$$P(|S_n| \geq \delta) \approx \exp(-n\delta^2/2) \quad (2.28)$$

That is, for large  $n$ , the probability of a large deviation in  $S_n$  follows something much like the asymptotic equipartition relation of the Shannon–McMillan Theorem.

This result can be generalized to more complicated probability spaces using Sanov's Theorem, the Gartner–Ellis Theorem, and related developments (Dembo and Zeitouni 1998) to show that large deviations paths of length  $n$  all have approximately the probability

$$P(n) \propto \exp(-n\mathcal{H}[\mathbf{X}]) \quad (2.29)$$

where  $\mathcal{H}$  is of the form  $-\sum_i P_i \log(P_i)$  for some probability distribution. Under the conditions of our analysis,  $P(n)$  is the probability of an excursion from the absorbing state of  $n = \text{zero infections}$ .  $\mathcal{H}$  thus quantifies an information source representing the active imposition of control strategies to prevent a large-scale outbreak of infection, and the Black–Scholes cost analysis carries through.

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