Agribusiness vs. Public Health: Disease Control in Resource-Asymmetric Conflict

Rodrick Wallace
Division of Epidemiology
The New York State Psychiatric Institute, USA
Alex Liebman
Department Geography
Rutgers University, USA
Luke Bergmann
Department of Geography
University of British Columbia, Canada
Robert G. Wallace
Institute for Global Studies
University of Minnesota, USA *

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Abstract

In the context of modern civilization, the ecology of infectious disease cannot be described by interacting populations alone, as much of the modeling literature presumes. As a matter of first principle, formalisms and their statistical applications must account for the anthrosphere from which pathogens emerge. With that objective in mind, we first formally examine strategies for controlling outbreaks by way of environmental stochasticities human institutions help set. Using the Data Rate Theorem, we next explore disease control regimens under asymmetric conflicts between agribusiness interests rich in resources and State public health agencies and local communities constrained by those very resources. Military theory describes surprising successes in the face of such an imbalance, a result we apply here. Abduction points to strategies by which public health can defeat agribusinesses in its efforts to control agriculture-led pandemics, the heavy health and fiscal costs of which multinationals routinely pass off to the public.

*Contact: rodrick.wallace@gmail.com, Rodrick.Wallace@nyspi.columbia.edu

1 Introduction

A disease outbreak represents more than a convergence of susceptibles, the infected, and those who have recovered from infection. A recent series of models of Ebola and vector-borne diseases, for instance, focused on the role systemic environmental stochasticity plays in driving outbreaks to extirpation or amplifying their propagation (R.G. Wallace et al. 2016, R. Wallace et al. 2018). The landscapes through which pathogens circulate have definitional impact on the outcomes of outbreaks whatever the evolutionary state of the specific disease agent. Epidemiological causality is found as much in the etiological field as in the object of the pathogen or patient.

A related focus of this line of research applied the Data Rate Theorem linking control and information theories to characterizing how public health efforts control such outbreaks. The warp and woof of pathogen and patient populations are as much weaved together by broader political contestation as by population dynamics. The models hypothesized the effects of anthropogenic clashes on disease outcomes are empirically discoverable. The approach produced a broad class of statistical models that can be fitted to data across pathogen species.

Here we rephrase that work in terms of different 'temperature' measures applicable to disease ecosystems, measures that are, in fact, close analogs to sterilizing immunity in confined systems. We include index measures associated with asymmetric conflict between contending agents with massively different access to conventional material resources. We will outline how a 'weak' institutional cognitive entity – here, governmental and civil society public health interests – can overcome a conventionally stronger opponent – entrenched agribusiness practice – to limit morbidity, mortality, and economic burden.

As has been explored at considerable depth elsewhere, currently practiced intensive agribusiness is unsustainable on even its own terms (e.g., Jones et al. 2013, Leonard 2014, R. Wallace and R.G. Wallace 2015, R.G. Wallace 2016, Cooper 2017). To survive, the agricultural sector must externalize the most damaging consequences of its production model by 'privatizing the profits and socializing the costs'. Consumers, governments, farmers, agricultural labor, livestock, wildlife, and local fields and waterways have long borne the material and fiscal fallout of declines in nutrition, declines in animal and landscape diversities, occupational hazards, pollution, xenospecific pathogens, and restrictions in farmer autonomy. Even the largest food conglomerates would not survive if such costs were returned to company balance sheets.

The imbalance in political power has long imposed definitional impacts upon disease ecologies (Watts 1997, R.G. Wallace 2016). Haalboom (2017) observed across four major Dutch outbreaks over the 20th century, agricultural interests repeatedly dominated the public health sector, a relationship that continued into the present century:

> Historically, agriculture and agriculture-related export have been very important to the Dutch economy. Hence, the specific and material economic interests of the Dutch agricultural sector got priority

over more abstract, general public health interests (along with professional interests to secure tasks in public health protection), which were considered politically relevant only later in time. When these interests asked for more or less the same measures, this resulted in the Dutch 'success stories' of effective control and public health protection, as in the cases of bovine TB in the last phase of its control and BSE. Nevertheless, trade incentives were also of overriding importance in those cases. But when the interests of the agricultural and public health domains clashed, as in the case of salmonellosis, the agricultural power to delay and influence legislation and control was large.

We begin with a conventional model of disease outbreak, exploring the role environmental stochasticity plays on disease dynamics in time, space, and genetic diversity, suggesting that 'economies of scale' characteristic of modern agribusiness practices can produce diseconomies in public health, amplifying disease danger as farms and food production expand in geographic extent and consolidate in number. We next model patterns of 'asymmetric conflict' between agents rich in material resources, as would be applied to agribusiness, and those rich in time and information, a different set of resources historically associated with peasant and smallholder communities. The results suggest potential strategies by which the position of public health as an ecosocial actor can be significantly improved.

2 Stochastic sterilization

Variation in time

We begin by following an established literature in disease modeling summarized by R. Wallace et al. (2018).

Most simply, for the earliest stage of a disease outbreak in a population of susceptible individuals, one can write a deterministic 'exploding' equation as

$$dN/dt = \alpha N(t) \tag{1}$$

at time t where N is the infected population, α is a positive real number representing the rate of growth of the infection, so that, early on, $N(t)$ increases exponentially in time as

$$N(t) = N_0 \exp[\alpha t] \tag{2}$$

The stochastic version of this is an Itô differential equation having the form

$$dN_t = \alpha N_t dt + \sigma N_t dW_t \tag{3}$$

where the second term represents volatility in a Brownian white noise dW_t. This is taken as a random noise signal with equal power within a fixed bandwidth at any center of frequency. The effect of noise is through the parameter σ.

Applying the Itô Chain Rule, a stochastic version of computing the derivative of the composition of two or more functions, to $\log[N]$ produces the relation

$$d\log[N_t] = (\alpha - \frac{\sigma^2}{2})dt + \sigma dW_t \tag{4}$$

where $-\sigma^2/2$ is the 'Itô correction factor'. Heuristically, given enough environmental noise, i.e., $\sigma^2/2 > \alpha$, by Jensen's inequality for a concave function (Cover and Thomas 2006), this gives a lower limit for $\log[E(X_t)] \geq E(\log[X_t]) \to a < 0$. Via stochasticity, that limit will be attained, and any outbreak must eventually be driven to extinction.

For 'colored' noise, with skewed spectral densities of a variety of distributions, if Eq.(3) can be expressed as

$$dN_t = N_t dY_t \tag{5}$$

where Y_t is a stochastic process in some complicated noise process dB_t, then the Doléans-Dade exponential (Protter 1990) can be defined by a semimartingale of bounded variation, and is written as

$$\mathcal{E}(X)_t \propto \exp(Y_t - \frac{1}{2}[Y_t, Y_t]) \tag{6}$$

$[Y_t, Y_t]$ is the quadratic variation of Y_t that, since B_t is not Brownian white noise, need not be simply proportional to time (Protter 1990). Heuristically, by the Mean Value Theorem, if

$$\frac{1}{2}d[Y_t, Y_t]/dt > dY_t/dt \tag{7}$$

$\mathcal{E}$ converges in probability to zero. R. Wallace et al. (2018) expands this analysis to vector-borne diseases, inherently having dimensionality greater than one, i.e., there are both host and vector population dynamics to be addressed. Despite some notable exceptions in the parameter space, sufficient variability still drives the infection to extinction.

A large enough 'stochasticity temperature' $T_t \equiv d[Y_t, Y_t]/dt$ 'sterilizes' disease outbreaks, in this model.

Variation in space

The simplest model of 'spatial stochasticity' follows Murray (1989, Sec. 14.8). In one spatial dimension x, again taking $N(x, t)$ as the number of infected individuals at location x for t, we write a 'diffusion equation' for the initial stage of an epidemic outbreak as

$$\partial N(x, t)/\partial t = \mu \partial^2 N(x, t)/\partial x^2 + \alpha N(x, t) \tag{8}$$

Expansion of the solution to this equation as a spatial Fourier series leads to a time dependence proportional to

$$\exp[(\alpha - C^2 \mu/L^2)t] \tag{9}$$

α is the growth rate, μ the coefficient of spatial diffusion, and C is a constant ≈ 1 depending on the dimension of the diffusion. Infection dies out if the patch size L is less than the critical length $L_c = C\sqrt{\mu/\alpha}$. This leads to a second 'spatial temperature' analog $T_L \equiv 1/L_c = \sqrt{\alpha/\mu}/C$.

For a version of the result that can be applied to diffusion of infection on a 'commuting field' defined by rapid, systematic patterns of travel, see Eqs.(2.11)-(2.14) of R. Wallace et al. (2018).

Variation in genetic structure

The role host genetic structure plays in epidemic propagation has long been explored (e.g. O'Brien and Everman 1988, King and Lively 2010).

Explicitly correcting the deterministic treatment of Anderson and May (1991), Lively (2010) provides a direct but sophisticated stochastic model, assuming a fixed reproductive cycle, as in annual birthing or, in an animal factory farm setting, market-driven turnover.

Lively takes the number of infected hosts having the ith genotype at time $t+1$ as

$$I_{i(t+1)} = g_{i(t+1)}N_{t+1}P_{i(t+1)} \tag{10}$$

$g_{i(t+1)}$ is the frequency of the ith host genotype at time $t+1$. N_{t+1} is the total number of hosts at time $t+1$. $P_{i(t+1)}$ is the probability of infection for the ith host genotype at time $t+1$. Assuming a Poisson distribution, P becomes 1 minus the zero class $\exp[-\lambda]$, where λ is the mean number of matching spores that contact each host. The probability of infection for the ith host genotype at $t+1$ is then

$$P_{i(t+1)} = 1 - \exp[-\lambda] = 1 - \exp[-BI_{i(t)}/N_{t+1}] \tag{11}$$

$I_{i(t)}$ is the number of infected hosts having the ith genotype at time t and B is the number of infectious propagules produced by each infection that make contact with different hosts.

B thus imposes an upper limit on the number of secondary infections.

Lively next assumes that a single infected individual, with genotype i, is introduced into the population of hosts at time t, so that $I_{i(t)} = 1$. The number of secondary infections, i.e., the infamous R_0, is then

$$R_{0i} = g_{i(t+1)}N_{t+1}(1 - \exp[-B/N_{t+1}]) \tag{12}$$

leading to the condition for propagation

$$g_{i(t+1)}N_{t+1} > \frac{1}{(1 - \exp[-B/N_{t+1}])} \tag{13}$$

For large populations, as $N \to \infty$, Eq.(12) gives

$$R_{0i} = g_i B > 1$$
$$B > \frac{1}{g_i} \tag{14}$$

5

For a large population, if the single-strain infection reduces the fitness and the frequency of the susceptible host genotype over time, the infection will die out when g_i becomes less than $1/B$.

Lively (2010) further argues that if the pathogen is introduced by migration at a high rate, there can be multiple coexisting strains of the infective agent, giving a mean value for R_0 as

$$< R_0 >= \frac{\sum R_{0i}}{G} = \frac{N_{t+1}(1 - \exp[-B/N_{t+1}])}{G} \to \frac{B}{G} \qquad (15)$$

as N becomes large, where G is the number of genotypes in the host population.

As Lively puts it,

> Thus, all else being equal, the spread and persistence of infection should more easily occur in genetically homogeneous populations.

In the Mathematical Appendix we elaborate upon the result, further correcting Anderson and May (1991).

The potential applications in time, space, and genetics are foundational. Other 'stochasticity temperatures' more conducive to disease control can be regionally planned. Crop rotation, agrobiodiversity, and ecological pest control can be incentivized by top-down market regulation and trade/tax structures. The city of Belo Horizonte, a city of 2.5 million in Brazil, implemented such a program (Chappell 2018). In conjunction with a state extension service, the city's Municipal Under-Secretariat of Food and Nutritional Security helped establish agroecological practices among outlying rural smallholders, and protect the mega-biodiverse Mata Atlantica-Cerrado, by guaranteeing a market and set prices subsidized for low-income urban consumers.

Mosaic agroecologies at large spatial and population scales, by their rich environmental stochasticities able to preempt most large-scale diseases before they emerge, appear feasible with appropriate government support.

3 Disease control failure

Following closely the arguments of R.G. Wallace et al. (2016), interacting polities, including the modern nation-state, are not merely natural populations or communities. The spread of infectious disease within such a complex organization cannot be treated as simply a problem in population dynamics.

States are cognitive entities. Confronted by any dynamic threat, a state must choose a relatively small set of responses from a much larger domain of possible policies and resources available to it. Choice reduces uncertainty and implies the existence of an information source generating successive messages. The Data Rate Theorem can be deployed to link control and information theories, tracking the effects of control information on control system effectiveness (Nair et al. 2007).

Here, epizootic spillover or some other spreading epidemic is constrained, or, in the case of systemic failure, released, by the adequacy or inadequacy of
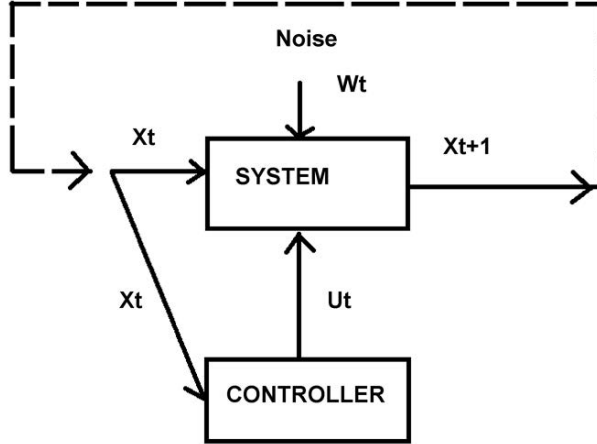
Figure 1: Schematic of a linear plant control system near nonequilibrium steady state. X_{t+1} is the 'plant' response to the control signal U_t and the earlier state X_t. U_t is the output of an information source. W_t is uncontrolled 'noise'.

the control information imposed by the broader social system via public health interventions or, in the longer term, by the quality of broader socioeconomic reform.

Figure 1 shows a schematic of a State's public health system, in the context of a challenge by some growing disease outbreak. We assume that an outbreak has begun to propagate, and that the aim is to contain it.

The system at time t receives a multidimensional state vector X_t and produces a new vector at time $t + 1$, written as X_{t+1}. At time t the system is likewise affected by a 'noise' vector W_t representing uncontrolled inputs, and by a 'control signal' vector U_t from cognitive entities within the state. The basic first-order 'linear plant' dynamics near some nonequilibrium steady state (nss) are then written as

$$X_{t+1} = AX_t + BU_t + W_t \tag{16}$$

with feedbacks indicated by figure 1. A and B are fixed matrices.

In the first stages of an explosive disease outbreak, the system must be seen as inherently unstable, in the sense that the matrix A can be factored by a similarity transformation into one having two diagonal submatrices A^U, A^S and two zero off-diagonal matrices such that A^U has eigenvalues ≥ 1 and A^S has eigenvalues < 1. Thus an epidemic or pandemic contagious disease with 'reproduction rate of infection' > 1 is clearly an inherently unstable system that must be brought under control by a State's public health institutions via control signals U_t.

The Data Rate Theorem (Nair et al. 2007) states that the rate of control information provided by the public health system, $\mathcal{H}$, must be greater than the

rate at which the unstable system generates 'topological information', a relation written as

$$\mathcal{H} > \log[|\det(A^U)|] \equiv H_0 \tag{17}$$

where det is the determinant of a matrix.

The rate at which a disease outbreak generates 'topological information' depends on the flow of infection along paths of contact between central cities, cities and their suburbs via the daily commute, and between individuals along social contact nets. We envision this as determined by some function of a composite scalar ρ akin to a density in a traffic model (e.g., R. Wallace 2018a).

For a pathogen such as influenza, Ebola, or one more intimately connected to social structure in its transmission such as HIV, ρ would be calculated as the length of a principal component from some multivariate analysis of data involving rates of deforestation, plantation or factory farming, confiscation of artisanal farmlands, housing overcrowding, percent of population in poverty, rates of deindustrialization, deurbanization, and violent crime, and so on (e.g., Wallace et al. 1999). Equation (17) then becomes

$$\mathcal{H}(\rho) > f(\rho)H_0 \tag{18}$$

where α_0 now represents the inherent topology of the underlying transportation and contact networks at the scale of interest. How might we characterize the functions $\mathcal{H}(\rho)$ and $f(\rho)$? Can we explicitly connect disease to the underlying socioeconomics? A complicated calculation, using an exactly-solvable Black-Scholes approximation (R.G. Wallace et al. 2016, Section 4.3) finds in first order

$$\mathcal{H} = \kappa_1\rho + \kappa_2 \tag{19}$$

where the κ_i are nonnegative constants.

Taking the same level of approximation, we assume $f(\rho)$ in Eq.(18) can be similarly expressed as $\kappa_3\rho + \kappa_4$ so that the stability relation is

$$T_C \equiv \frac{\kappa_1\rho + \kappa_2}{\kappa_3\rho + \kappa_4} > H_0 \tag{20}$$

where we now define T_C as the control temperature of the system.

At low ρ the stability condition is $\kappa_2/\kappa_4 > \alpha_0$, and at high ρ it becomes $\kappa_1/\kappa_3 > \alpha_0$. If $\kappa_2/\kappa_4 \gg \kappa_1/\kappa_3$, then at some intermediate value of ρ the essential inequality may be violated, leading to uncontrolled growth of infection (Figure 2).

This formalism describes a general phenomenon, imposed on all inherently unstable control systems by a theorem as powerful as the Central Limit Theorem that determines the Normal distribution. The ability of a political entity – for instance, an entrenched agribusiness system – to control its own resources and contain an opposition can be similarly compromised. As we will discuss below, under the challenge of a public health opponent, ρ is then replaced by the 'shadow price' that an opposition imposes on the conduct of agribusiness-as-usual.
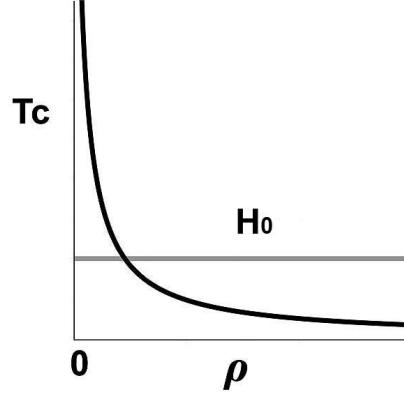
8

Figure 2: The horizontal line represents the critical limit H_0. If $\kappa_2/\kappa_4 \gg \kappa_1/\kappa_3$, at some intermediate value of accumulated environmental insult ρ, resulting in a low value of the 'control temperature' T_C, the relation $(\kappa_1\rho + \kappa_2)/(\kappa_3\rho + \kappa_4)$ falls below that limit, and the growth of infection becomes uncontrollable. We will argue, however, that similar dynamics may affect a powerful agribusiness entity when ρ is replaced by 'shadow price' and/or other burdens imposed by a coalition of highly adaptive public health opponents.

4   Asymmetric conflict

In addition to producing large-scale environmental and health damage, multinational agribusiness embodies large-scale political, economic, and social power that extends beyond material impact. An economic sector of such political power imposes an institutionalized sociopathy that is normalized in governance and research as the guiding spirit of an age, in this case organized around a metabolic rift that divides the bounded regenerative ecologies upon which all life depends from the present expectations of infinite economic growth (Lefebvre 1974/1992, Foster et al. 2010, R.G. Wallace and Kock 2012, Malm 2016, R.G. Wallace et al. 2018).

History shows that escape from the consequences of such a trap demands its severe price. Courtesy of (mostly) WWII's Eastern Front – typically grouped as the battles of Moscow, Stalingrad, Kursk, and Operation Bagration – the world was, at great cost, liberated from Nazi ideology and state. There are other modes of liberation. The outgunned Vietnamese expelled both French and American colonial armies, as did the Algerians the French. The Afghans expelled the British and Russians, and seem intent on the same with the Americans. Western colonial systems after WWII suffered expulsions across Africa, India, and Indonesia. In most cases, the means to liberation – or, depending on one's perspective, of subversion – involved conflicts between entities of vastly different material resources. These contests are described by military strategists

(e.g., Farrell et al. 2013) as 'asymmetric conflict'.

War is only one of many forms of such conflict. There are structures underlying successful asymmetric conflict that may be harnessed by public health, environmental groups, and farming communities, within both the State and civil society, against what is now a multiscale and spatially discontinuous 'colonial' system multinational agribusiness has constructed across national borders as a series of proverbial 'Soybean Republics' operating outside or in parallel to nation-states (Turzi 2011, Haesbaert 2011, Meyforidt et al. 2014, Craviotti 2016, Oliveira 2016).

Examples of asymmetric advantage abound. During the American war in Vietnam, approximately 85% of combat incidents were initiated by local Viet Cong and North Vietnamese forces. Such initiative eventually decided the war. The current Afghanistan stalemate reflects a roughly similar ratio for ground combat, in spite of the U.S.'s drone war dominance. Under other circumstances, such an imbalance in information and initiative would be treated as unfair and grounds for punishment. In stock market trading, grossly different information rates are called 'insider trading' and subject to prosecution.

We can model the conditions under which an asymmetry of field intelligence can overcome asymmetry of power, in the context of constraints imposed by Clausewitzian fog, friction, and 'shadow price' (Wallace 2020).

We begin by considering a target institution made up of a number cooperating and interacting cognitive entities. This composite entity operates on a landscape of conflict in which there are, for it, three essential resources. The model can be extended to greater numbers of resources, at the expense of more algebra. We take these resources as information passing between subsystems of the cognitive entity, provided at an overall internal channel capacity $\mathcal{C}$, 'sensory' information about the embedding environment, provided from the 'outside' at a channel capacity $\mathcal{H}$, and material, energy, and/or finances provided at an overall rate $\mathcal{M}$. We envision a 3×3 matrix $\mathbf{Z}$ with the diagonal $\{\mathcal{C}\,\mathcal{H}\,\mathcal{M}\}$, but having off-diagonal elements representing interaction between the three basic rates, analogous to, but different from, a correlation matrix. An $n \times n$ matrix has n invariants that remain fixed when the matrix undergoes certain symmetry transformations, and these can be used to construct a scalar measure much like a principal component analysis, via the standard polynomial relation

$$p(\gamma) = \det(\mathbf{Z} = \gamma \mathbf{I}) = \gamma^n + r_1 \gamma^{n-1} + ... + r_{n-1}\gamma + r_n \qquad (21)$$

det is the determinant, γ a parameter, and $\mathbf{I}$ the $n \times n$ identity matrix. The invariants of $\mathbf{Z}$ are the coefficients of γ in $p(\gamma)$, normalized so that the coefficient of γ^n is 1. Typically, the first invariant is the trace of $\mathbf{Z}$, and the last $\pm$ the determinant.

We define a scalar index Z as an appropriate monotonic increasing scalar function of the n matrix invariants $r_1, r_2, ..., r_n$. This is not a trivial matter, and will hold much of the art of the science. The simplest such index would be $Z = \mathcal{C} \times \mathcal{H} \times \mathcal{M}$, but interaction crossterms in $\mathbf{Z}$ are likely to be important in the real world.

The Rate Distortion Theorem provides a different, and deep, perspective on cognition and its failure in the particular context of the Data Rate Theorem of the previous section. Central focus is on the difference between what is initially ordered by a powerful institution and what is actually observed, taking cognitive control to involve a 'channel' along which a 'message' is transmitted. The difference between what the institution wants and what happens is defined in terms of a scalar distortion measure D. For any such channel, there is a Rate Distortion Function $R(D)$, always convex in D (Cover and Thomas 2006), that determines the minimum channel capacity – a free energy measure from Feynman's (2000) perspective, representing an information transmission rate – needed to ensure that the average distortion between intent and execution is less than or equal to D.

Taking $R(D)$ as a free energy measure, and following the standard view from chemical reaction theory (Laidler 1987), the rate of cognition control above the punctuated threshold arising from the Data Rate Theorem, which we called H_0 above, can be characterized as a Boltzmann pseudoprobability:

$$Pr[R > H_0] = \frac{\int_{H_0}^{\infty} \exp[-R/g(Z)]dR}{\int_0^{\infty} \exp[-R/g(Z)]dR} = \exp[-H_0/g(Z)] \qquad (22)$$

where, again, H_0 is the limit from Eq.(17), and $g(Z)$ is a scalar temperature analog depending on the scalar resource rate index Z that must be calculated from first principles. This is not a physical system in which temperature is an independent parameter.

Treating the denominator of Eq.(22) as a statistical mechanical partition function produces an expression for a free energy Morse Function (Pettini 2007) F as

$$\exp[-F/g(Z)] = \int_0^{\infty} \exp[-R/g(Z)]dR = g(Z)$$
$$F = -\log[g(Z)]g(Z) + H_0 \qquad (23)$$

This permits the definition of an associated entropy-analog as the Legendre transform

$$S = -F + ZdF/dZ \qquad (24)$$

The next step is to define system dynamics in terms of the Onsager approximation from nonequilibrium thermodynamics (de Groot and Mazur 1984), so that, in first order,

$$dZ/dt \approx \mu dS/dZ = f(Z) \qquad (25)$$

Taking $F(g(Z))$ as a general expression, and expanding in terms of Eqs.(24) and (25), leads to a second order ordinary differential equation having the implicit solution

$$F(g(Z)) = X(Z) = -C_1 Z - Z \int \frac{f(Z)}{Z} dZ + C_2 + \int f(Z) dZ$$

$$g(Z) = F^{-1}[X(Z)] \qquad (26)$$

Here, $F = -\log[g(Z)]g(Z) + H_0$, and an explicit solution can be found in terms of a special function, the Lambert W-function, as

$$g(Z) = \frac{X(Z)}{W(n, X(Z))}$$

$$X(Z) = -C_1 Z - Z \int \frac{f(Z)}{Z} dZ + C_2 + \int f(Z) dZ$$

$$f(Z) =$$

$$-\frac{\left(\ln(g(Z)) \left(\frac{d^2}{dZ^2} g(Z) \right) g(Z) + \left(\frac{d^2}{dZ^2} g(Z) \right) g(Z) + \left(\frac{d}{dZ} g(Z) \right)^2 \right) Z}{g(Z)}$$

$$\frac{X(Z)(1 + W(n, X(Z)))^2 \frac{d^2}{dZ^2} X(Z) - \left(\frac{d}{dZ} X(Z) \right)^2 W(n, X(Z))}{(1 + W(n, X(Z)))^3 X(Z)} < 0 \qquad (27)$$

W is the Lambert W-function of order n that solves $W(n, X) \exp[W(n, X)] = X$, and the last relation is the necessary and sufficient condition that $g(Z)$ is concave – i.e., $d^2 g(Z)/dZ^2 < 0$ – over the region of interest, since it is the 'temperature' $g(Z)$ that will ultimately drive the cognition rate from Eq.(22). Concavity in $g(Z)$ will prove to be central to our overall argument, as it determines when 'overload' begins to cripple an institution's rate of cognitive function.

On Clausewitz landscapes (Wallace 2020a), as opposed to 'more normal' cognitive systems, friction – here characterized by the relation $dZ/dt = f(Z)$ – is one of the most central driving forces, and will set the stage for $g(Z)$, subject to the separate boundary conditions C_1 and C_2. The fourth form of Eq.(27) essentially precludes assigning a simple S-shaped relation to $g(Z)$, since the frictional relation $dZ/dt = f(Z)$ is imposed by embedding realities mostly beyond the explicit control of a particular cognitive entity. This is an important point.

Note that the zero-order branch of the Lambert W-function is real only for $-\exp[-1] < X < \infty$, and the $n = -1$ branch is real only for $-\exp[-1] < X < 0$, leading, in figure 3, after some thought on the boundary values C_1 and C_2, to inverted-U signal transduction forms having punctuated dynamics. Here, the Clausewitzian friction is taken as $f(Z) = \beta - \alpha Z$, with $\alpha = 1$, so that $Z \to \beta$, having boundary conditions $C_1 = 1, -1$ $C_2 = -1, -3$. If $X(Z) < -\exp[-1]$, a challenged system's rate of cognition collapses.

With regard to optimization, a multicomponent cognitive system is confronted with the necessity of high rates of cognition at all scales and levels of organization, but under overall resource rate constraint. The dynamics of such
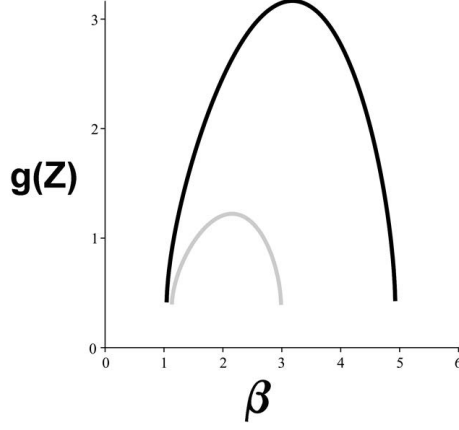
12

Figure 3: Signal transduction form for $g(Z)$ from Eq.(27), taking $f(Z) = \beta - \alpha Z$, $\alpha = 1$. $Z \to \beta/\alpha$, and β varies. The condition for a real-valued 'temperature' is $X(Z) > -\exp[-1]$. $C_1 = 1, -1$ and $C_2 = -1, -3$. Only for Z in a limited range is $g(Z)$ real-valued, as represented by the inverted-U, in a manner highly dependent on system parameters.

a system can again be crudely addressed using a standard Lagrangian optimization across cognition rate.

While 'real world' optimization is likely to be more in the realm of 'the hand reaches, the eye measures the gap', any optimization procedure will be confronted by problems similar to those limiting Lagrangian optimization.

The Lagrangian optimization problem for the overall probability/reaction rate relation of Eq.(22):

$$L = \sum_i \exp[-H_0^i/g(Z_i)] + \lambda \left(Z - \sum_i Z_i \right)$$
$$\partial L/\partial Z_i = 0$$
$$\frac{H_0^i \left(W\left(X\left(Z_i \right) \right) \right)^2 \frac{d}{dZ_i} X\left(Z_i \right)}{\left(X\left(Z_i \right) \right)^2 \left(1 + W\left(X\left(Z_i \right) \right) \right)} e^{-\frac{H_0^i W(X(Z_i))}{X(Z_i)}} = \lambda$$
$$\partial L/\partial Z = \lambda \qquad (28)$$

Here, L is the Lagrangian, W is the Lambert W-function of order zero, $X(Z)$ is as in Eq.(9), and λ is the economic shadow price (Jin et al. 2008) imposed by the resource constraints.

We can then solve the third expression of Eq.(28) for H_0^i, deriving a synergistic relation between λ and the $X(Z_i)$:
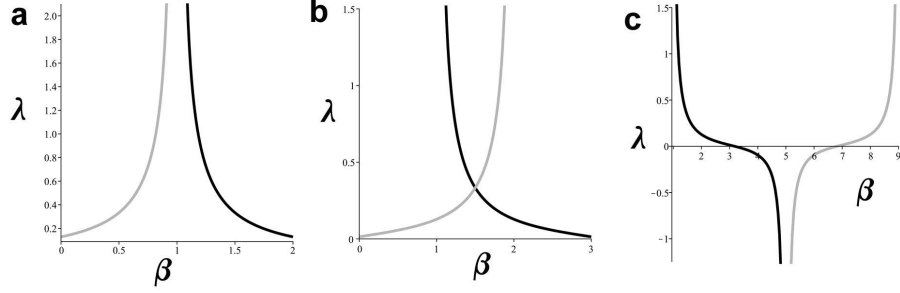
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Figure 4: We examine a two-component equally weighted 'exponential delay' system for which the optimum resource division is equal sharing. For the third expression of Eq.(28), $f(Z) = \beta - \alpha Z$. The Lambert W-function is of order zero. Here, $\alpha = H_0 = 1$, $C_1 = -1$, $C_2 = -3$, $Z = \beta/\alpha$, and β varies. (a) $\beta = 2$ (b) $\beta = 3$. (c) $\beta = 10$. Here, even where optimization is possible, it may be precluded by shadow price λ.

$$H_0^i = -\frac{X(Z_i)}{W(X(Z_i))} W\left(-\frac{\lambda X(Z_i)(1 + W(X(Z_i)))}{\left(\frac{d}{dZ}X(Z_i)\right)W(X(Z_i))}\right) \qquad (29)$$

Necessary conditions for the solutions to be real numbers are

$$-\exp[-1] \le X(Z_i) < \infty$$

$$-\exp[-1] \le -\frac{\lambda X(Z_i)(1 + W(X(Z_i)))}{\left(\frac{d}{dZ}X(Z_i)\right)W(X(Z_i))} < \infty \qquad (30)$$

These relations link λ, the environmentally-imposed shadow price – another stress measure, in a large sense – to the Z_i, the scalarized rates at which essential resources are delivered to each subcomponent under an overall limit.

Figure 4 shows an example of the third part of Eq.(10) for a two-component, equally-weighted system. The expected division of Z is, of course, $Z_{max}/2$. The delay function $f(Z)$ in $X(Z)$ is assumed to be the 'exponential' model, so that $f(Z(t)) = \beta - \alpha Z(t)$. For figure 4, $H_0 = 1, \alpha = 1, C_1 = -1, C_2 = -3, Z = \beta/\alpha$, and β varies, taking the values 2, 3, and 10. For the smallest and largest values, optimization is not possible, a consequence of the inverted-U form. For $\beta = 3$, optimization is possible *only if the shadow price λ is in the proper range.*

That is, if the environmentally-imposed shadow price λ is beyond the range of possible system response, optimization is not possible.

The essential inference is that shadow price burdens will interact with resource rate to impose punctuated cognitive failure across a hierarchically organized system.

Again, more sophisticated optimization methods – 'the hand reaches, the eye measures the gap' – can be expected to display recognizably similar patterns of punctuated failure.

14

Van den Broeck et al. (1994, 1997), and Horsthemeke and Lefever (2006), among others, have explored the role of 'noise' in driving phase transitions in physical systems. We adapt their methods, based on the stability criteria of stochastic differential equations, to examine 'noise'-driven phase change associated with contention by cognitive agents on a Clausewitz landscape.

Eq.(27) allows calculation of the nonequilibrium steady state average of $g(Z)$ under stochastic variation in terms of Z, via the Ito Chain Rule (Protter 2005).

The relation $dZ/dt = f(Z) = \beta - \alpha Z$ leads to the stochastic differential equation

$$dZ_t = f(Z_t)dt + \sigma Z_t dW_t$$
$$dZ_t = (\beta - \alpha Z_t)dt + \sigma Z_t dW_t \qquad (31)$$

where the second term imposes stochastic volatility proportional to Z, driven at the rate σ. dW_t represents Brownian white noise, the standard starting point for such analyses, which can be extended to 'colored' noise (Protter 2005).

Applying the Ito Chain Rule to Z^2, the variance of Z can be calculated from the second form of Eq.(31) as

$$< Z^2 > - < Z >^2 = \left(\frac{\beta}{\alpha - \sigma^2/2} \right)^2 - \left(\frac{\beta}{\alpha} \right)^2 \qquad (32)$$

Variance explodes as $\sigma^2/2 \to \alpha$, independent of β and $Z_\infty = \beta/\alpha$. The rate constant α counts on its own.

Using Eq.(31) as the base SDE it is possible to use the Ito Chain Rule on $g(Z)$ itself. The result for the mean value of g at nonequilibrium steady state, i.e., when $< dg_t > = 0$. This convolutes the Lambert W-function with σ, α, and β, and the boundary conditions C_1 and C_2, representing a synergism between the effects of fog, delay, and bifurcation phase transitions. Unfortunately, the computer algebra result is too long for the LaTex compiler, and is given in the Mathematical Appendix as an image from the computer algebra program Maple 2019. The Ito Chain Rule calculation for $g(Z)$ is quite routine, if tedious.

Setting $\alpha = 1$, $C_1 = -1$, $C_2 = -3$, figure 5 applies the *implicitplot3d* numerical procedure of Maple 2019 to the pattern in $< dg_t > = 0$ for the variates β, Z and σ. The Lambert W-function in the expression for $< dg_t > = 0$ imposes necessary conditions driving bifurcation phase transitions on the dynamics – the behavior – of cognitive process under fog and frictional stochasticity.

This procedure generates distinct equivalence classes in $\{Z, \beta, \sigma\}$. The lowest plane in figure 5 appears to be a kind of grinding down under the burden of noise, with a nonequilibrium steady state possible until σ becomes sufficiently large, here, displaying a raised probability of an inflammatory explosion well before the ultimate limit of $\sigma = \sqrt{2}$. Transition between equivalence classes representing groupoid symmetry-breaking phase transitions, as discussed in more detail below, remains to be more fully explored, but appears to involve yet another 'temperature' parameter, as studied in Wallace (2020b) for biological systems.
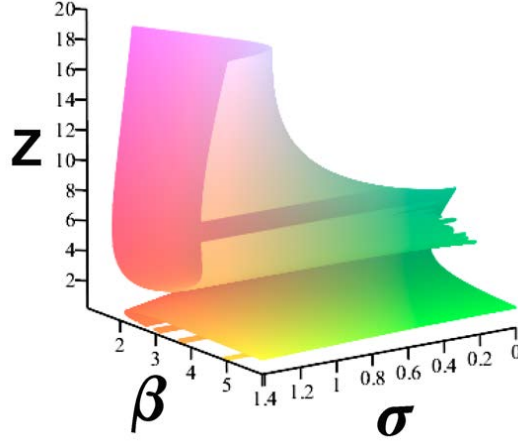
Figure 5: Equivalence classes in β, Z, and σ defined by the relation $< dg_t >= 0$. $\alpha = 1 \, C_1 = -1$, $C_2 = -3$. Recall that the condition for variance stability in Z is $\sigma^2/2 < \alpha$, in this case, $\sigma < \sqrt{2}$. Here, however, the probability of an inflammatory explosion rises significantly well before that ultimate limit. The bottom plane represents a grinding down of a nss under noise.

Some insight emerges from the real-value requirement of the Lambert W-function in the $< dg_t >= 0$ equation:

$$-C_1\,Z - Z\left(-\alpha\,Z + \beta\,\ln(Z)\right) + C_2 - 1/2\,\alpha\,Z^2 + \beta\,Z > -\exp(-1) \qquad (33)$$

The dynamics of bifurcation phase transitions depend heavily on the nature of noise influence. 'Noise' and time delay 'friction' become synergistic with cognitive system phase transitions, generating tortuously complex and critically unstable patterns of function and failure. These instabilities can be exploited by a sufficiently astute adversary of the primary institution under siege.

The U.S. command's focus on Khe Sanh on the eve of the Tet offensive, in spite of considerable intelligence suggesting the offensive (e.g., Bowden 2017), offers one example. A similar cognitive failure can be found in Douglas MacArthur's blind advance into North Korea against growing evidence of Chinese military buildup in Manchuria. The U.S. occupation of Iraq in 2003, and subsequent sequelae, also come to mind, as does the long-term impact of the current drone war in Afghanistan and across other Middle East and African states.

Indeed, unrestrained conflict often becomes a tit-for-tat 'language that speaks itself' (e.g., R. Wallace 2018b, Ch.6).

What happens if the embedding conflict ecosystem shadow price defined by λ is outside the range for which some value of $Z_i \leq Z$ can be found? Again, as Jin et al. (2008) explain, under pathological circumstances, no optimization can

exist, and the system-of-interest fails, possibly triggering a spreading collapse as connected subcomponents are also overloaded and fail.

These insights have been explicitly discussed in the military science literature.

5 John Boyd's OODA Loop

Military theorist John Boyd was the principal architect of the famous 'left hook' in the 1993 Gulf War that so devastated the conventional Iraqi army. Generalizing from his experiences of the 'thin, warm' fog-of-war characterizing mid-20th century air combat, Boyd explored the intimate relation between cognition and control that is inherent to virtually all contention, including arguably the conflict between agribusiness and public health we address here. His work developed from a simple sequential dynamic model of Observation, Orientation, Decision, and Action, to the elaborate feedback system of his later presentations. Commodore Prof. Frans Osinga (2007) of the Netherlands Defense Academy describes Boyd's approach as follows:

> The OODA loop model as [later] presented by Boyd...represents his view on the process of individual and organizational adaptation in general, rather than only the military-specific command and control decision-making process that it is generally understood to depict. It refers to [a] conceptual spiral...to the process of learning, to doctrine development, to command and control processes and to the Popperian/Kuhnian ideas of scientific advance. The (neo-)Darwinists have their place, as do Piaget, Conant, Monod, Polanyi and Hall, while Prigogine and Goodwin are incorporated through Boyd's concluding statement in the final slide that follows [his presentation of] the OODA loop picture:
>
> "The key statements of this presentation, the OODA Loop Sketch and related insights represent an evolving, open-ended, far from equilibrium process of self-organization, emergence and natural selection."
>
> This relates the OODA loop clearly to Complex Adaptive Systems, the role of schemata and to the process of evolution and adaptation. Once again it shows that where the aim is 'to survive and prosper' in a non-linear world dominated by change, novelty, and uncertainty, adaptation is the important overarching theme in Boyd's strategic theory.

Figure 6, from Osinga (2007), is adapted from Boyd's presentation and shows his fully-developed adaptive cognitive/control process model, which should be compared to the inherently unstable control system model of figure 1.

We have, in the previous sections, provided something of a roadmap for formal exploration of Boyd's approach to conflict. On the other hand, Boyd's analysis suggests that in the conflict between agribusiness and public health,
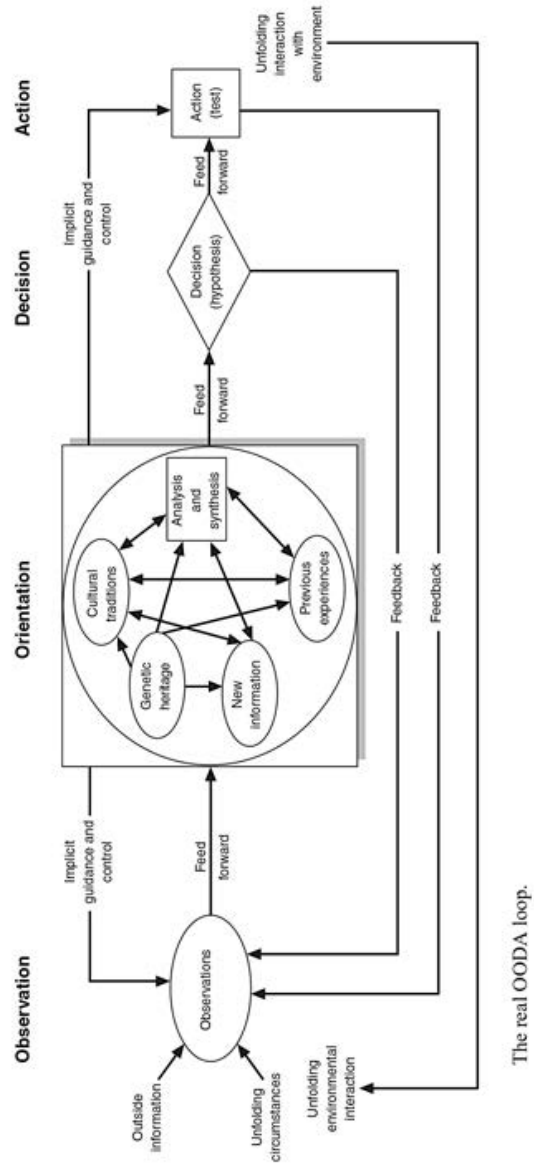
17

Figure 6: Adapted from Osinga (2007). John Boyd's fully-developed OODA loop convolutes cognition with control, under the conditions of uncertainty inherent to conflict on a Clausewitz landscape strongly dominated by friction and the fog-of-war. The key to survival for a 'weak' agent is greater adaptability than its 'stronger' opponent.

in which agribusiness holds considerable material advantage, it will be necessary for public health advocates to focus on adaptation, changing the 'rules of the game', and associated strategy and tactics, faster than the opposition can respond. One objective should be to force 'shadow prices' on conventional agribusiness practices that can be met only by changing those practices in a manner that protects, rather than threatens, the health and safety of large human populations.

That is, asymmetry in material resources can be met by a synergism of 'richness' in intelligence and of flexibility. Conversely, a materially weak opponent can successfully challenge the materially superior agency by placing unbearable shadow prices on that agency's own material, communication, intelligence, and flexibility resources.

One military example stands out, if only because it illuminates the conditional complexity of such an 'adaptation' surface. As described above, German Bewegungskrieg tactics in WWII were eventually countered on the Eastern Front. Nonetheless, in spite of having overwhelming material superiority before and after Kursk, Allied forces were unable to break the German army until January 1945. The Germans retained superior – or at least adequate – internal communication, intelligence, and tactical flexibility until that late date.

The problem of the 'weak', then, is to develop an adaptable strategy that places a carefully crafted synergism of unbearable shadow prices on the various essential gearings of current agribusiness practice and its relations to the machineries of governance, a strategy that adapts more rapidly than can or will be met by entrenched power. Such campaigns are by definition difficult and may demand more than merely more of the same: among possibilities, careful but flexible strategic planning, greater intelligence in agribusiness strategy and operations, decentralized execution, and the element of repeated but unpredictable surprise.

6 Discussion

Placing public health first

In spite of their ill repute among scientists, normative dichotomies can serve as first-order criteria. In an age of climate debates scientists have effectively lost depending on data alone (Goeminne 2010), it appears Hume's guillotine, distinguishing what is from what we researchers wish, is circumstantially false. For the case presented here, it is useful, and arguably necessary as a matter of scientific practice, to distinguish the language of business – the language of capital's imaginarium (Lacan 1972, Böhm and Batta 2010, Carley and Molina 2011, Holmes 2013, Tomšič 2015, Vanheule 2016, Fracchia 2017) – from the language of public health.

Failures in public health's conflict with agribusiness have produced collateral damage in the millions of people and animals hit by deadly pathogens and other environmental spillovers propagating far beyond the farm gate (Tscharntke et

al. 2005, Gilchrist et al. 2007, Leibler et al. 2010, Atkins et al. 2010, Ercsey-Ravasz et al. 2012, Smit et al. 2012, Allen and Lavai 2014, Rotz and Frase 2015, Martin-Prével et al. 2016, Allen et al. 2017, Jones et al. 2017, R.G. Wallace 2018a). The clash can be resolved in the broader population's favor, permitting social reproduction generation to generation in the face of what presently are by all accounts a series of protopandemics.

To that aim, in the series of models presented here we examined strategies for the 'sterilization' of infectious disease at the population level via stochasticities that conflict with current agribusiness 'economies of scale' privatizing profits and socializing costs. We have used, in addition, the perspectives of control theory and of the asymmetric conflict between entrenched agribusiness interests rich in resources and public health entities constrained by the many pressures exerted by those very resources on the structures of governance. Asymmetric conflict has been surprisingly successful in military realms. Abduction of appropriate strategies from those examples may also prove successful for the prevention and control of mass-fatal, agribusiness-driven pandemics. Indeed, we argue that the ability to rapidly and systematically adapt strategies and tactics is as central to success in public health as it is in armed conflict.

We are not alone in thinking so. In the light of Q fever's devastating effects to health and economy alike 2007-2010, the Dutch public health establishment made a conscious effort, albeit tightly circumscribed in the context of the goat industry from which the pathogen sprung, to impose new regulation (Smit et al. 2012, Van der Hoek et al. 2012, Quammen 2012, Hogerwerf et al. 2013, Hogerwerf 2014). An evaluation commission explicitly recounted the means by which during the outbreak the Ministry of Agriculture prevailed over the Ministry of Health, with adequate responses to the outbreak delayed for ulterior reasons, specifically undue appeals to farmer privacy and withholding locale data of infected farms (Van Dijk et al. 2010). The commission recommended the Ministry of Health take the lead in outbreaks that might follow and that incidence data be shared immediately with public health stakeholders – e.g., the National Institute for Public Health and the Environment and municipal health services – to permit the promptest response. Such a 'zoonosen structuur' has since been devised and implemented.

Devastating public health outcomes, however, are no guarantee changes in the public's favor will be pursued. A pandemic strain that emerges out of agriculture, an increasingly predominant mode, can always be blamed on a virus, as proved the case for swine flu H1N1 and Ebola Makona (R.G. Wallace 2016, R.G. Wallace et al. 2016). Indeed, to that aim, agribusiness has expanded lobbying efforts and political contributions aimed at governance across jurisdictions, from local governments to UN agencies (Davis 2003, McCue 2012, Clapp 2014). Changing the 'rules of the game,' as the modeling here implies, is a necessary but often insufficient step for any path to public health victory. In many fora, power isn't just handed over. Conflicts can play out in more than bureaucratic tussles between ministries.

Agribusiness and counterinsurgency

Agricultural policy regularly bleeds into paramilitary and military action, the latter illustrated – in examples more recent than the touchstones of Chiquita in Guatemala or Operation Produce in Indonesia – by the U.S. Military Agribusiness Development Teams in Afghanistan and Orders 24, 27, and 81 under Coalition Provisional Authority rule opening Iraq to foreign seed breeders and prohibiting farmers "from re-using seeds of protected varieties." (Grajales 2011, Johnson et al. 2011, Johnson et al. 2012, Ballvé 2013, Baker 2014, Woods 2018). For agribusiness in contested territory, the comparison here between military science and the struggle over land use and its public health impacts is no mere abduction.

Ag-related counterinsurgency appears to be evolving inside agribusiness's new multiterritoriality and along its spatially discontinuous networks of fluctuating territorial embeddedness across borders and funding sources (Patel 2013, R. Wallace et al. 2018).

A team of international legal experts implicated the Netherlands Development Finance Institution (FMO), among other financial institutions, in the 2016 murder of internationally known Honduran environmental activist Berta Cáceres. Without the capital needed to build the Agua Zarca Hydroelectric Project proposed for Río Blanco between the departments of Intibucá and Santa Bárbara in western Honduras, Desarrollos Energéticos Sociedad Anónima, founded just for the project, secured financing from international sources. Altholz et al. (2017) charged FMO, the Central American Bank for Economic Integration (CABEI), and the Finnfund, among other groups that supplied project capital, with "willful negligence" in Cáceres's murder.

The funds pursued a strategy with shareholders, executives, managers, and employees of DESA, private security companies working for DESA, public officials, and state security agencies "to control, neutralize and eliminate any opposition." Among the tactics DESA and its security contractors pursed were setting community groups against each other, smear campaigns, infiltration, surveillance, threats, contract killing, sabotaging communication equipment, and co-optation of justice officials and security forces. Altholz et al. report:

> These [international foundations] through repeated complaints and reports by international consultants, had prior knowledge of the strategies undertaken by DESA. Nevertheless, they failed to implement appropriate, effective and timely measures to guarantee respect for the human rights of indigenous communities affected by the Agua Zarca dam, much less to protect the life and integrity of Berta Isabel Cáceres Flores. Nor did they make sufficient efforts to ensure the appropriate criminal investigations.

In July 2017, FMO and Finnfund confirmed exiting the Agua Zarca project, but Berta Cáceres's murder represents only one of what appears a surge in targeted assassinations around the world aimed at countermanding community efforts at blocking corporate land grabbing and environmental destruction.

Global Witness (2014) reported 1000 environmental activists have been killed since 2002:

> On average two people are killed every week defending their land, forests, and waterways against the expansion of large-scale agriculture, dams, mining, logging, and other threats. Often they have been forced from their homes or seen their livelihoods harmed by environmental devastation. Some victims were environmental protesters killed in crackdowns, others murdered by hired assassins because they lived on a desirable plot of land.

These murders, many associated loosely enough to offer international private-public development partnerships plausible deniability, increased to four a week by 2017 (Global Witness 2018). For the first time since these deaths began to be tabulated, agribusiness moved past mining as the most dangerous sector, with 46 activists killed and a spike in massacres. The Business Human Rights Resource Centre also documented 388 attacks upon campaigners in 2017, more than a quarter associated with conflicts over agribusiness projects (Bachi 2018, Kelly 2018, Global Witness 2018).

Peasant innovations in asymmetric conflict

Peasants and smallholders resist such campaigns, militarized or not. The recent literature on 'repeasantization' can be recast in terms of peasants, agricultural laborers, and less-capitalized farms regaining power and autonomy against agribusiness hegemony (e.g., Schneider and Niederle 2010, Viá 2012, Rosset and Martinez-Torres 2014, Pahnke 2015, McMichael 2016). van der Ploeg (2009) theorizes the peasant condition as a suite of interrelating conditions that permit persistence and resilience in the face of class hostility. Among these are "a 'self-controlled resource base', 'co-production' or interaction between humans and nature, cooperative relations that allow peasants to distance themselves from monetary relations and market exchange, and an ongoing 'struggle for autonomy' or 'room for maneuver' that reduces dependence and aligns farming with the interests and prospects of the producers...''' (Edelman 2011).

Some of these advantages arise from exactly the adaptations on which the modeling here converges. Peasants lose economies of scale but during downturns can survive as subsistence operations or, conversely, outcompete suffering industrial wage-labor farms by selling their products at lower prices, a history of market adaptation that predated industrial production (Banaji 1990, R.G. Wallace 2018b).

In contradiction to their repute, the successes of economies of scale are context-dependent, allowing for epistemic alternatives (Reinhardt and Barlett 1989). The efficiencies of monoculture consolidation must race against diseconomies of production, from bad produce to pollution and disease outbreak. The natural economy of agriculture imposes multiple limits, including upon the speed and simultaneity of production. Agribusiness must rely on its political power to survive itself, importing near-slave labor, globalizing production

and distribution, cornering subsidies and regulation regimens in their favor, all products of massive State intervention (R.G. Wallace 2018b). Agribusiness finds its advantages not in the microeconomics of production, as often presented in moralist terms, but in commanding market access.

Peasants across the globe have innovated asymmetrical strategies opposed to such fait accompli, from the very first enclosures of the common field (Brenner 1976, Huizer 1976, Scott 2008, La Vía Campesina 2010, R.G. Wallace 2018b,c). In some contexts, these strategies transitioned into military action or were undertaken simultaneously (Taber 1965/2002, Hawes 1990). During the Guinean war on Portuguese colonialism, the PAIGC under Amílcar Cabral, trained as an agronomist, supported the return to autonomous resource management severed from the colonial export economy (McCulloch 1981, Chabal 1983, Neves 2017).

Models that explicitly connect disease dynamics to these political economies, as we have presented here, can contribute toward strategizing advances in public health prevention and farmer autonomy a growing literature indicates are necessary in scaling down infectious disease outbreaks of agricultural origins. Against the sovereign epistemic individualism at the core of a dated notion of natural science (Code 2013) and a related response skepticism opposed to collective action (Doan 2016), researchers need openly articulate and act on the accumulating evidence that successful public health and disease control broadly require the commons in land and community. The specifics in form and content represent a cutting-edge science of the 21st century.

Mathematical Appendix

Correcting Anderson and May

The basic deterministic statement of the Anderson and May (1991) treatment of genetic diversity and epidemic spread is that the rate of change of infected individuals of genetic strain i is

$$dI_i/dt = \beta I_i S_i - v I_i \tag{34}$$

where, letting $S_i = g_i N$ and taking v as the 'removal rate', we get

$$dI_i/dt = (\beta g_i N - v) I_i \tag{35}$$

which, *for fixed β*, explodes as $N \to \infty$.

The essential point, however, is to recognize that the argument leading to Eq.(12) *applies to the infectivity parameter β*. That is, the corrected deterministic equation is

$$dI_i/dt = I_i(\beta_0(1 - \exp[-B/N])g_i N - v) \to I_i(\beta_0 B g_i - v) \tag{36}$$

for which '$R_0' \equiv \beta_0 B g_i / v$ declines below 1 with increase in genetic diversity, i.e., other things being equal, with sufficient decline in g_i.

We can, however, do even better by introducing more stochasticity via a volatility term in I. We then obtain the SDE

$$dI_i \approx (\beta_0 B g_i I_i - v I_i)dt + \sigma I_i dW_t \tag{37}$$

Carrying out an Itô chain rule expansion on $\log(I)$ gives a Doléans-Dade exponential

$$\mathcal{E}(I) \to \propto \exp\left[[\beta_0 Bg - (\sigma^2/2 + v)]t\right] \tag{38}$$

which, because of the added $\sigma^2/2$ term – further stochasticity – collapses even more rapidly than the solution to the corrected deterministic model.

Ito Chain Rule calculation

The nonequilibrium steady state relation $< dg_t > = 0$ from Eq.(27), using the base SDE in Z of Eq.(31).

$$
\begin{aligned}
&\left(\left[-2\left(-\beta \ln(Z) + \left(-\frac{\sigma^2}{2} + \alpha\right)Z - CI\right)\right.\right. \\
&\quad \left(-2\ln(Z)Z\beta + \alpha Z^2 + (-2CI + 2\beta)Z\right. \\
&\quad \left. + 2C2\right)(\alpha Z - \beta)\,\mathrm{LambertW}\left(\right. \\
&\quad -\ln(Z)Z\beta + \frac{\alpha Z^2}{2} + \frac{(-2CI+2\beta)Z}{2} \\
&\quad \left. + C2\right)^2 + \left(-8\left(\left(\frac{\sigma^2}{4} + \alpha\right)Z\right.\right. \\
&\quad \left. - \beta\right)Z\beta^2 \ln(Z)^2 + 12\left(Z^3\alpha^2 + \left(\left(\frac{\sigma^2}{3}\right.\right.\right. \\
&\quad \left. - \frac{\alpha}{3}\right)\beta - \frac{4CI\left(\frac{\sigma^2}{4} + \alpha\right)}{3}\right)Z^2 \\
&\quad + \left(\frac{4}{3}CI\beta + \frac{2}{3}C2\alpha - \frac{2}{3}\beta^2\right)Z \\
&\quad \left. - \frac{2C2\beta}{3}\right)\beta \ln(Z) - 4Z^4\alpha^3 + ((2\alpha\sigma^2 \\
&\quad - 4\alpha^2)\beta + 12CI\alpha^2)Z^3 + ((-4\sigma^2 \\
&\quad + 8\alpha)\beta^2 - 4CI(-\sigma^2 + \alpha)\beta - 8C2\alpha^2
\end{aligned}
$$

$$
\begin{aligned}
&+ (-8CI\beta^2 + (-4C2\sigma^2 + 8CI^2 \\
&+ 8C2\alpha)\beta + 8CIC2\alpha)Z - 8CIC2\beta) \\
&\mathrm{LambertW}\left(-\ln(Z)Z\beta + \frac{\alpha Z^2}{2}\right. \\
&+ \frac{(-2CI+2\beta)Z}{2} + C2\right) - 2\left(\right. \\
&-\beta\ln(Z) + \left(-\frac{\sigma^2}{2} + \alpha\right)Z - CI\right)(\\
&-2\ln(Z)Z\beta + \alpha Z^2 + (-2CI+2\beta)Z \\
&\left. + 2C2\right)(\alpha Z - \beta)\Bigg/\Bigg(2(\\
&-2\ln(Z)Z\beta + \alpha Z^2 + (-2CI+2\beta)Z \\
&+ 2C2)\left(1 + \mathrm{LambertW}\left(-\ln(Z)Z\beta\right.\right. \\
&\left.\left.\left.\left. + \frac{\alpha Z^2}{2} + \frac{(-2CI+2\beta)Z}{2} + C2\right)\right)^3\right)\right) \\
&= 0
\end{aligned}
$$

Acknowledgments

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