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General ecological models for human subsistence, health and poverty

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The world's rural poor rely heavily on their immediate natural environment for subsistence and suffer high rates of morbidity and mortality from infectious diseases. We present a general framework for modelling subsistence and health of the rural poor by coupling simple dynamic models of population ecology with those for economic growth. The models show that feedbacks between the biological and economic systems can lead to a state of persistent poverty. Analyses of a wide range of specific systems under alternative assumptions show the existence of three possible regimes corresponding to a globally stable development equilibrium, a globally stable poverty equilibrium and bistability. Bistability consistently emerges as a property of generalized disease–economic systems for about a fifth of the feasible parameter space. The overall proportion of parameters leading to poverty is larger than that resulting in healthy/wealthy development. All the systems are found to be most sensitive to human disease parameters. The framework highlights feedbacks, processes and parameters that are important to measure in studies of rural poverty to identify effective pathways towards sustainable development.

The conclusion of the Millennium Development era in 2015 provided benchmarks for human development, including a target to reduce extreme poverty by 50%[1]. As nearly a billion people in the world still subsist below the international poverty line[2], there remains considerable debate over general causes of persistent extreme poverty. Approximately 70% of the poor in sub-Saharan Africa and Southeast Asia subsist from primary resource extraction: agriculture, timber and fishing[3]. Concomitant to this, around 35% suffer from chronic malnutrition and more than 75% die from infectious diseases[4]. As the global health community has broadened its priorities in light of the sustainable development goals and a movement for planetary health[5], these statistics underline the importance of understanding ecological foundations of economic development based on two core principles: (1) the capital of the poor is often biological in the form of crops, livestock, forests, wildlife, soils and fisheries[3,6–8]; and (2) the dynamics of capital is embedded within systems of ecological interactions or food webs that include pathogens of humans and their biological resources[9–11].

'Poverty traps' commonly refer to the idea that accumulating wealth requires a minimum amount of wealth (for example, beyond subsistence), such that there is enough to be saved and invested for the future. Modern (neoclassical) economic growth theory has shown how poverty traps can arise from nonlinear processes in the growth rate of capital (or wealth). Most models of poverty traps are phenomenological—that is, based on qualitative assumptions about these nonlinear processes—and are rarely derived from explicit understanding of the underlying feedbacks that reinforce poverty, such as disease and resource scarcity[9,12,13]. Owing to effects on child development and labour productivity, the role of health conditions (particularly disease and malnutrition) as a driver of poverty traps has gained increasing attention[9,14,15]. It has also been recognized that poverty is an important risk factor for acquiring and succumbing to disease[16]. The intuitive argument for these coupled disease–poverty systems is that escaping from such traps is difficult for the rural poor, who are highly susceptible to infectious diseases and rely heavily on subsistence agriculture. Notably, human health and resource dynamics are determined by biological processes that are well studied in the scientific literature. Models of the ecological basis of human livelihoods can accordingly be coupled explicitly with economics to identify dynamics that are based on fewer, simpler and more evidence-based assumptions that are rooted in scientific knowledge. Using simple toy models based on such principles, a recent study[11] demonstrated that under specific assumptions, ecologically driven poverty traps can be formed. These models were not analysed broadly to provide a general understanding of how such models behave and did not reveal general conditions that lead to poverty traps.

Here, we present the first general theoretical framework of its kind where ecological, economic and epidemiological factors leading to persistent poverty are generalized and broadly analysed. We show that systems of capital (renewable resources, human capital, physical capital) and natural enemies (for example, infectious diseases and pests) can be described with two fundamental equations, comparable to predator–prey models in population biology. We use this general modelling structure as a blueprint to derive a library of models of increasing complexity to represent specific ecological, economic and epidemiological systems that investigators can apply to systems of interest. These models are then parameterized from country-level data and analysed over the feasible parameter space to explore qualitative and quantitative properties of different regimes of economic development and human health. The analyses show

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that poverty traps, defined as self-reinforcing (that is, stable equilibrium) systems of poverty, are general features of the models.

Model framework

The models are based on classic consumer–resource ecological relationships and are formalized as ordinary differential equations commonly used in both ecology and economics[17,18]. The general model (equations (1) and (2)) describes the rate of change of two broad classes of state variables: capital $\mathbf{x}=(x_1, x_2, x_3,\ldots, x_M)$, including physical, human and biological capital; and 'natural enemies' $\mathbf{z}=(z_1, z_2, z_3,\ldots, z_J)$, that is, parasites, pests, predators and competitors that compete with or consume biological forms of capital:

$$\dot{x}_i=\psi_i(f(\mathbf{x},\mathbf{z}),\mathbf{x},\mathbf{z})-\delta_i(f(\mathbf{x},\mathbf{z}),\mathbf{z})x_i,\ i=1,2,3,\ldots,M \quad (1)$$

$$\dot{z}_i=\beta_j(f(\mathbf{x},\mathbf{z}),\mathbf{x},\mathbf{z})-\gamma_j(f(\mathbf{x},\mathbf{z}),\mathbf{z})z_j,\ j=1,2,3,\ldots,J \quad (2)$$

Figure 1 presents a schematic of a coupled disease–economic model that can be adapted to other forms of capital and natural enemies with slight modifications. A general template for the coupled models is provided in Table 1. In all cases, income (f) is generated from different types of capital (x), which in turn interacts with natural enemies (z) in a variety of system-specific ways. Capital dynamics are described by equations structurally identical to the standard neoclassical economic growth model (Supplementary Information Section 1.1.4)[12,17], where the state variable x corresponds to a measurement of physical capital per person (for example, infrastructure and equipment). The rate of capital growth is determined by the difference between accumulation and loss terms. Capital accumulation (ψ) is assumed to be proportional to a production function (or income) (f) determined by the capital savings rate (r), while capital loss ($\delta_i x_i$) depends on the capital depreciation rate (δ).

For the rural poor, capital takes two primary forms: (1) natural resources, such as plants, livestock, fisheries, soils and wildlife populations[7]; and (2) human capital in the form of health and nutrition[19,20]. To understand the dynamics of such biological capital, we turn to the large body of theory developed in population ecology. Here, the variable x represents a biological population and the growth term (ψ) is determined by rates of biological growth or reproduction. Loss of biological capital represents death or decay (occurring at rate δ), often caused by 'natural enemies' z, intrinsic to all biological systems (equation (2))[21]. Specific natural enemies of the poor include infectious diseases of humans, organisms that parasitize or consume crops, livestock and wildlife, and those that degrade, consume, or damage natural forms of physical capital (such as fungi, termites or rodents). Their dynamics are also described by the difference between their reproductive (β) and mortality (γ) rates.

Natural enemies can cause poverty by inhibiting capital accumulation in a number of ways, namely by reducing labour productivity directly via the production function (f), by reducing rates of human capital acquisition (r), or by increasing rates of capital depreciation (δ), as occurs with pests. In turn, the level of capital accumulation can affect the dynamics of natural enemies by decreasing their reproductive (or transmission) rate, such as through the use of pesticides or better sanitation in the case of human diseases, or by increasing their mortality (clearance, or recovery) rate, such as through better healthcare. A model template for the dynamics of three forms of capital and natural enemies is presented in Table 1, while explicit coupled ecological-economic models based on this template are presented in Table 2.

Table 1 | Model template.

State variable	Growth	Loss	Source
Capital			
Physical capital, x_1	$r_1 f(x,z_1)$	$\delta_1(0,0)x_1$	Refs 12, 17
Human capital, x_2	$r_2(z_1)f(x,z_1)$	$\delta_2(0,z_1)x_2$	Refs 11, 31
Renewable resources, x_3	$r_3(x_3,f(x,z))x_3$	$\delta_3(f(x,z),0)x_3$	Refs 32–35
Natural enemies			
Disease, z_1	$\beta_1(f(x,z),z)(1-z_1)$	$\gamma_1(f(x,z))z_1$	Refs 36–38
Pest, z_2	$\omega b(f(x,z_2))x_3z_2$	$\gamma_2(f(x,z_2))z_2$	Ref. 35

State variables are presented in column 1, growth terms in column 2 and loss terms in column 3. The general structure of the models has the form 'change in state variable = growth − loss', corresponding to equation (1) or (2). The r values are the capital accumulation or growth rates, the δ values are the capital depreciation or decay rates, the γ values are general loss, clearance, or removal rates, β is the human disease transmission rate, b is the consumption rate of renewable resource by pests and ω represents the effective units of plant biomass converted to pest biomass (see Supplementary Information Section 1 for details).

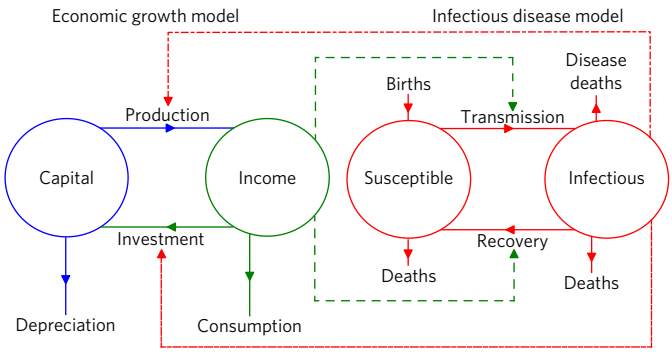

Figure 1 | Schematics of the coupled disease–economic model. For the economic growth sub-system to the left, income is generated from capital through production. Part of the income is consumed, while the other part is reinvested into capital. Capital in this sub-system can be regarded as economic (for example, human or physical capital) or biological (for example, renewable resources). For the infectious disease sub-system to the right, disease transmission occurs between susceptible and infectious individuals. All individuals are born susceptible; they die naturally, while infectious individuals can also be killed by the disease or recover from infection. Dashed green lines indicate that disease transmission and recovery from disease are functions of income. Dash-dotted red lines indicate that economic productivity and investment in capital are functions of infectious disease. Disease affects income through labour productivity. Note that income is not a state variable.

Results

The resulting coupled model (equations (1) and (2)) broadly resembles a wide range of classic consumer–resource ecological systems[22], such as predator–prey, crop–pest, or infectious disease systems[18,23], with some distinct characteristics. A common feature of classic ecological models is negative feedback, where increases in one state variable (such as the number of prey or susceptible individuals) drive up the other state variable (such as the number of predators or infected individuals), whereas increases in the latter drive down the former. The resulting feedbacks prevent the variable or population from growing unbounded. The long-term dynamics are characterized by a globally stable attractor in the form of sustained oscillations (stable limit cycles) or a stable equilibrium that may be approached through damped oscillations. By contrast, the dynamics of our core human disease models ((i)–(ii) in Table 2) differ because each of the state variables negatively influences the other: capital reduces disease and disease reduces capital. The dynamics are thus characterized by

Table 2 | Coupled systems.

Model	*h*	*h*(*I*)	*k*	*y*(*I*)	*c*	*p*	*n*	Income function and model equations
								$y_1 = \phi f(0, h, 0),$
(i)	✓	✓						$\dot{h} = r_h \Phi(I) y_1 - \delta_h h$ $\dot{I} = \beta(y_1)(1-I)I - \gamma(y_1)I$ $y_2 = \phi f(0, h, I)$
(ii)	✓	✓		✓				$\dot{h} = r_h \Phi(I) y_2 - \delta_h h$ $\dot{I} = \beta(y_2)(1-I)I - \gamma(y_2)I$ $y_3 = \phi f(k, h, I)$
(iii)	✓	✓	✓	✓				$\dot{k} = r_k y_3 - \delta_k k$ $\dot{h} = r_h \Phi(I) y_3 - \delta_h h$ $\dot{I} = \beta(y_3)(1-I)I - \gamma(y_3)I$ $y_4 = (\phi + \pi_c \delta_c c) f(k, h, 0), \quad E = k^{\alpha_k} h^{\alpha_h}$
(iv)	✓		✓		✓			$\dot{k} = r_k y_4 - \delta_k k$ $\dot{h} = r_h \Phi(0) y_4 - \delta_h h$ $\dot{c} = r_c c\left(1 - \frac{c}{c_0 E}\right) - (\delta_c E + \mu_c)c$ $y_5 = (\phi + \pi_c \delta_c c) f(k, h, I), \quad E = k^{\alpha_k} h^{\alpha_h}$ $\dot{k} = r_k y_5 - \delta_k k$
(v)	✓	✓	✓	✓	✓			$\dot{h} = r_h \Phi(I) y_5 - \delta_h h$ $\dot{c} = r_c c\left(1 - \frac{c}{c_0 E}\right) - (\delta_c E + \mu_c)c$ $\dot{I} = \beta(y_5)(1-I)I - \gamma(y_5)I$ $y_5 = (\phi + \pi_c \delta_c c) f(k, h, I), \quad E = k^{\alpha_k} h^{\alpha_h}$ $\dot{k} = r_k y_5 - \delta_k k$
(vi)	✓	✓	✓	✓	✓	✓		$\dot{h} = r_h \Phi(I) y_5 - \delta_h h$ $\dot{c} = r_c c\left(1 - \frac{c}{c_0 E}\right) - (\delta_c E + b(y_5)p + \mu_c)c$ $\dot{I} = \beta(y_5)(1-I)I - \gamma(y_5)I$ $\dot{p} = \omega b(y_5) pc - d(y_5)p$ $y_3 = \phi f(k, h, I)$
(vii)	✓	✓	✓	✓			✓	$\dot{k} = r_k y_3 - (\delta_k + n(I))k$ $\dot{h} = r_h \Phi(I) y_3 - (\delta_h + n(I))h$ $\dot{I} = \beta(y_3)(1-I)I - (\gamma(y_3) + \nu + \lambda)I + \nu I^2$ $y_5 = (\phi + \pi_c \delta_c c) f(k, h, I), \quad E = k^{\alpha_k} h^{\alpha_h}$ $\dot{k} = r_k y_5 - (\delta_k + n(I))k$
(viii)	✓	✓	✓	✓	✓		✓	$\dot{h} = r_h \Phi(I) y_5 - (\delta_h + n(I))h$ $\dot{c} = r_c c\left(1 - \frac{c}{E}\right) - (\delta_c E + \mu_c)c$ $\dot{I} = \beta(y_5)(1-I)I - (\gamma(y_5) + \nu + \lambda)I + \nu I^2$

Explicit formulations of coupled ecological–economic models based on the template presented in Table 1. Model (i) is a basic 2D system with one form of capital (human capital, *h*) coupled to one form of natural enemy (infectious disease, *I*). Model (ii) incorporates the disease explicitly in the income equation (denoted by *y*(*I*) in model (i)), representing lost labour productivity. Model (iii) includes physical capital *k*, in the neoclassical growth tradition in model (ii). System (iv) couples the economic model with a basic renewable resource, *c*, such as crops or livestock, without a natural enemy. System (v) adds human disease to system (iv). System (vi) is a combination of systems (i)–(v) and includes diseases both of humans (*I*) and the renewable resource (*p*), as with crop pests or livestock disease. Systems (vii) and (viii) are respective variants of systems (iii) and (v) with human population growth denoted by *n* (additional variants of these models and their analysis are presented in the Supplementary Information). Corresponding to Table 1, $x_1 = k$, $x_2 = h$, $x_3 = c$, $z_1 = I$ and $z_2 = p$. The y_i values, $i \in \{1, 2, 3, 4, 5\}$ are per capita incomes, $\beta(y_i) = \beta_{max}\beta_y/(y_i + \beta_y)$ is the human disease transmission rate, $\gamma(y_i) = \gamma_{min}\gamma_y(y_i + 1)/(y_i + \gamma_y)$ is the human recovery rate, *m* is the number of pathogens, *b* is the renewable resource consumption rate by pest, *d* is the pest death rate, *r* is the capital accumulation rate, δ is the capital depreciation rate, c_0 is a scaling constant of the carrying capacity for the renewable resource, $\Phi(I) = (1 - \xi I)m$ and $f(k, h, I) = \Phi(I)^{1-\alpha_k-\alpha_h} k^{\alpha_k} h^{\alpha_h}$ (see Supplementary Information Section 1 for further details on the models).

positive reinforcing feedbacks that are known to generate bistability (see Supplementary Information Section 3.1 for details), with the actual outcome—that is, steady development or persistent poverty—potentially contingent on history. When such relationships exist throughout the state space, the system converges monotonically, with no transient oscillations, to one of the stable equilibria, the only kind of attractor[24]. Phase and bifurcation diagrams for basic two-dimensional (2D) systems ((i) and (iv) with human capital (*h*) equals zero), and time evolution bifurcation diagrams for models (iii), (iv) and (vi) depict a poverty trap emerging from interactions between economic growth, infectious diseases and renewable resources (Fig. 2; Supplementary Figs 4–9 for additional examples and analyses of other models). In more complex systems that incorporate additional interactions, positive

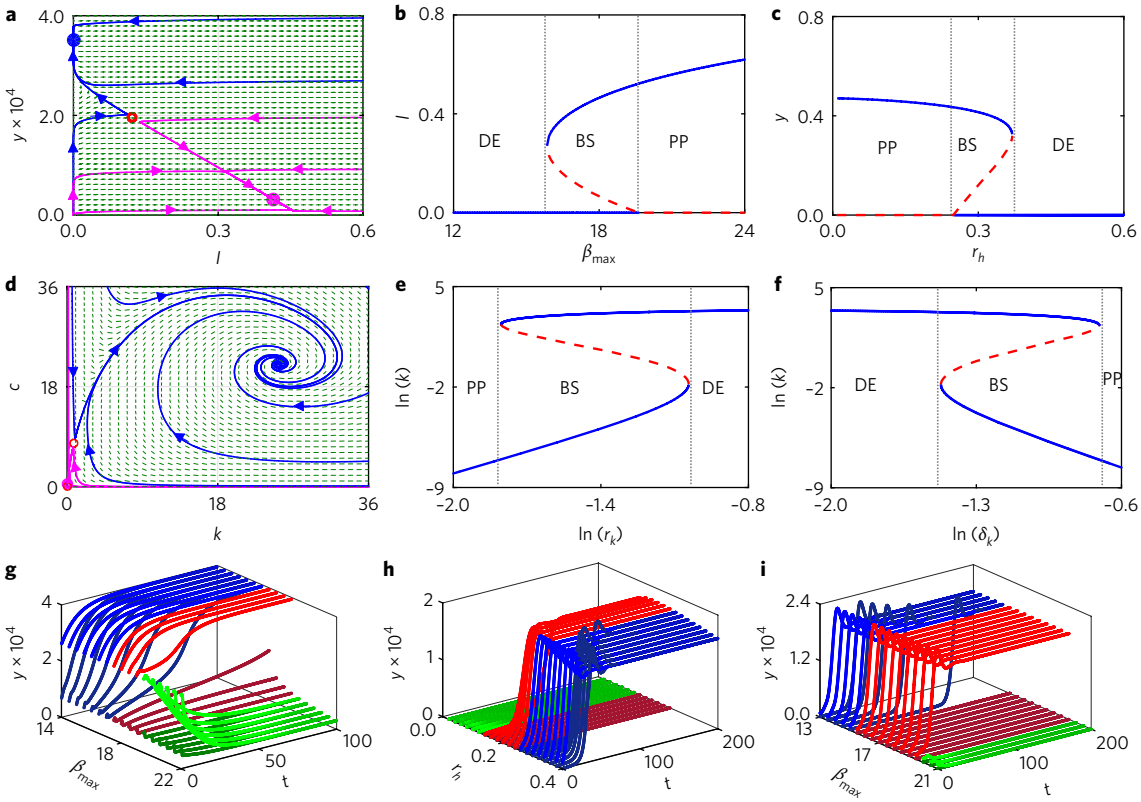

Figure 2 | Phase and bifurcation diagrams for the coupled systems in Table 2. a–c, These diagrams correspond to the 2D disease–economic model ((i) in Table 2). **d–f**, These diagrams correspond to a 2D version of the renewable resource–economic model ((iv) in Table 2). **g–i**, These diagrams represent the temporal trajectories for different regimes in systems (iii) (**g**), (iv) (**h**) and (vi) (**i**) of Table 2. Poverty traps are represented for income, y, versus disease I (**a**) and renewable resources, c, versus capital, k (**d**), where the trajectory for each variable is determined by the initial conditions. Trajectories converging to the development equilibrium (blue dot) are depicted by blue lines, while those converging to the poverty trap (magenta dot) are depicted by magenta lines. The red circle represents the unstable equilibrium or 2D threshold. Bifurcation diagrams for disease prevalence (**b** and **c**) and capital (**e** and **f**) are presented. The figures show how the system can shift regimes from globally stable poverty (PP), to bistability (BS), to globally stable development (DE) as parameters (transmission, investment and depreciation) change. Panels **g–i** illustrate these bifurcations now from the perspective of temporal trajectories that start from two different initial income values, as a function of the maximum transmission rate, β_{max}, and capital investment rate, r_h. These bifurcations correspond to regime shifts of globally stable development (blue lines), bistability (red lines) and globally stable poverty (green lines). Details are provided in Supplementary Information Section 3 and the parameters used to generate the figure are presented in Supplementary Table 2 (for results on the dynamics of other systems, see Supplementary Fig. 6).

and negative feedbacks coexist and the resulting dynamics cannot be easily deduced from simple analytical considerations.

For a deeper understanding of the dynamical properties of these systems, we numerically integrate them and conduct bifurcation and sensitivity analyses. We do this for 11 variants of the general system. The first eight variants (Table 2) represent two forms of consumer–resource relationship (disease and renewable resources) with different levels of complexity; specifically, different kinds of capital (physical (k), human (h), renewable resource (c)), natural enemies (human pathogens (I), agricultural pests (p)), human population growth, and different types of feedback between the economic and biological systems (see the Supplementary Information for details). First, we parameterize the basic model with data on income and disease burdens for countries around the world (Supplementary Information Section 2.1). For each model, we then numerically explore the full range of feasible parameter space to determine the regions of parameter space that generate the three different regimes of dynamic behaviour: globally stable development, globally stable poverty and bistability. Globally stable poverty and bistability both represent ways that systems can fail to achieve stable development. If the parameters are uniformly distributed, one can interpret the size of the space in the pie charts (Fig. 3b–i) as broadly reflecting how likely a given behaviour is to occur in a particular system. Owing to a lack of data on the real distribution of parameters, we conduct sensitivity analyses on the parameter space to determine which parameters are most likely to influence each outcome[25] (see Supplementary Information Section 4 for details on the methods).

The results indicate that bistability is a nontrivial (18–20%) part of the parameter space in the basic economic–disease model (Fig. 3b,c). Moreover, globally stable poverty represents the largest portion of the parameter space for all the models explored, especially for the more complex models that account for the role of resources and their biological interactions (Fig. 3b–i). Taken together, these two outcomes correspond to about 55% or more of parameter space.

The results can be explained by relative effects of positive versus negative feedbacks in the systems. The first three models (Fig. 3b–d) consist of disease, human capital and physical capital (model (iii)) for which the dynamics are dominated by mechanisms of positive feedback. They produce bistability in substantial portions of the parameter space. The last three models (Fig. 3e–g) consist of capital and natural resources, where density-dependent growth of the natural resources has intrinsic mechanisms of negative feedback. These resource limitations dominate the mechanisms

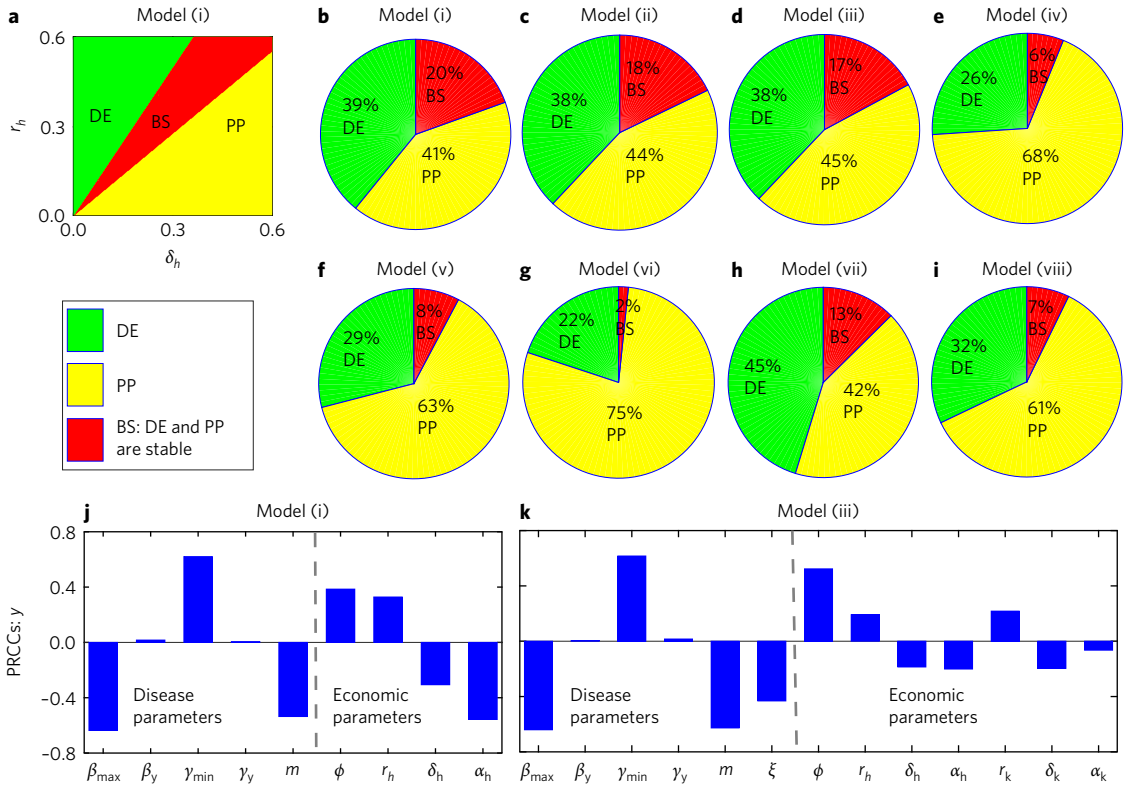

Figure 3 | Development regimes and important parameters. a, Combinations of the capital investment rate r_h, and the capital depreciation rate δ_h, for model (i) in Table 2 that result in a given dynamical regime: DE (green region), globally stable development equilibrium (all initial conditions lead to the same high level of wealth and health); BS (red region), bistability (both the poor and wealthy equilibria coexist, are stable, and the outcome depends on initial conditions); and PP (yellow region), globally stable poverty equilibrium (all initial conditions lead to the same low level of wealth and health). **b**–**i**, Percentage of parameters that result in each dynamical regime: DE, BS and PP. The different pie charts correspond to different models in Table 2, namely: the basic coupled infectious disease–economic models (i) (**b**) and (ii) (**c**); the coupled infectious disease–economic model with physical capital and direct effect of disease in income (iii) (**d**); the coupled renewable resource-economic model (iv) (**e**); the coupled infectious disease–economic–renewable resource model (v) (**f**); the coupled infectious disease–economic–renewable resource–pest model (vi) (**g**); and variants of models (iii) (**h**) and (v) (**i**) with human population growth (described by models (vii) and (viii)). **j**,**k**, Partial rank correlation coefficients (PRCCs) from global uncertainty and sensitivity analyses (details in Supplementary Information Section 4) depicting the contributions or significance of parameters (see Supplementary Table 2 for descriptions of the parameters) to per capita income for the coupled infectious disease–economic models (i) (**j**) and (iii) (**k**) described in Table 2. The larger the magnitude of the PRCC, the more significant the effect of the parameter is in the uncertainty or variability of the output. The sign of the PRCC indicates whether an increase in a parameter leads to an increase (when it is positive) or decrease (when it is negative) in the output. Uncertainty or variability in the maximum disease transmission rate, β_{max}, and the minimum recovery rate, γ_{min}, introduces the greatest uncertainty or variability in equilibrium disease prevalence, per capita income or the percentage of trajectories that converge to the poverty trap (see Supplementary Figs 7 and 9 for details and for analyses of additional systems).

of positive feedback, resulting in a greater portion of parameter space leading to globally stable poverty.

The sensitivity analysis further identifies the most important parameters that drive each of the systems in Table 2. Maximum disease transmission and minimum recovery rates are consistently found to be the most important drivers of the economic and health outcomes in all the systems, even as their complexity increases with a higher number of parameters (Fig. 3j,k; Supplementary Figs 7 and 9). Other parameters, such as the diversity of pathogens in the system, technological progress and the economic elasticity coefficients, are also important. For the coupled economic–renewable resource model ((iv) in Table 2), the intrinsic growth rate, the carrying capacity coefficient and the harvest constant are important determinants of income. However, these parameters are not consistently influential across the range of models (Table 2; see Supplementary Information Section 4 for details).

Discussion

Recognizing that the capital of the poor is rooted in biological consumer–resource relationships[7,9,26] allows us to develop a set of general models that couple population ecology and economic growth theory. These models reveal inherent nonlinearities in the accumulation of biologically generated capital. Our findings indicate that stable poverty and bistability (that is, poverty traps) are features of coupled ecological–economic systems. As the ecological complexity of the systems grows—particularly in the form of renewable resources and their natural enemies—the parameter space that generates globally stable poverty seems to expand. Thus, in our model, natural resources and infectious diseases provide ultimate 'limits' to economic growth.

Poverty traps may be most broadly defined as any self-reinforcing system that allows poverty to persist. The economics literature has mostly focused on bistability, where populations can settle into either a wealthy state or a poor state, depending on the initial levels of wealth[13]. Technically, bistable conditions imply that a sufficiently large change in initial conditions can shift the system into the basin of attraction of the alternative stable state. It would follow that substantial one-time changes (or sufficiently large 'exogenous shocks') in income or population health can lead to permanent changes in the long-term outcomes of the system. Such notions have been central to arguments for 'big push' policies characteristic of the Millennium Development era[9,27].

In contrast, if the persistence of poverty involves a globally stable equilibrium, then breaking such a poverty trap necessitates permanent and sustained changes in structural properties of the system (that is, the parameters). These changes can technically operate in two ways, by either driving a single poor equilibrium towards higher values, or generating a different, globally stable development equilibrium via a bifurcation. Thus, in cases of both globally stable poverty and bistability, fundamental and (swift) changes in the development trajectories of poor populations can be induced by changes to parameters of the system. Although temporary shocks can have long-term benefits in the case of bistability, structural (parametric) improvements confer additional advantages to economic development that temporary changes cannot achieve. Specifically, reinforcing globally stable development equilibria create conditions for resilience (that is, returning to equilibrium after shocks), while bistability implies that downward shocks—such as financial collapse, conflict, natural disasters, or disease outbreaks—can be catastrophic for countries with fragile economic development trajectories (for example, the Ebola outbreak in Liberia and Sierra Leone).

In summary, policies that influence parameters have inherent advantages over policies that induce positive 'shocks', even in cases of bistability.

Regardless of the development regime, the sensitivity analyses of our models showed that epidemiological parameters in the form of the disease transmission and recovery rates are the most consistently important determinants of long-term health and wealth dynamics. This is presumably because of the substantial population-level feedbacks that characterize infectious diseases. Their parameters have relatively straightforward policy interpretations: policies that result in greater access to healthcare independent of income correspond to lower rates of transmission and faster rates of recovery. Thus, assuming the costs are sufficiently low, our models are consistent with other findings that improving health systems can promote economic growth and help to lift populations out of poverty[28]. Economic parameters such as the technology coefficient and capital investment rates are also influential, albeit to a lesser extent. There are examples of countries that have made structural changes to health systems of the kind described above. Rwanda, for example, achieved all its health-related Millennium Development Goals through universal health coverage with social insurance systems, providing broad and robust access to healthcare for the poor, while economic growth has been among the highest in Africa[29,30].

Our library of models presents knowledge gaps that need to be addressed in future research. The general idea that poverty can increase the chance of acquiring and succumbing to disease, and that chronic diseases trap humans in poverty, are well established in the literature[16]. Yet, we still lack robust metrics to calibrate the models adequately. Important extensions thus include collecting empirical data to characterize these systems more deeply, and exploring not only qualitative equilibrium outcomes, but system dynamics. To integrate this work more deeply with economic theory, extensions could include explicit consideration of human behaviour, where system parameters such as the rates of saving, disease transmission and recovery are dynamic variables determined by human decisions. The existence of 'externalities' in the system, where the benefits and costs of individual decisions have impacts on third parties (such as transmitting disease or consuming a common pool resource), imply that the outcome of individual optimization processes would not necessarily result in optimal outcomes for society. These additional complexities can readily be incorporated into the framework presented here and can provide further insights into sustainability science. Understanding feedbacks between poverty, biological resources and disease is thus of primary importance for deriving integrated intervention strategies that can simultaneously lead to improved health, sustainable growth and healthy ecosystems.

Code availability. All the models in the main text and the Supplementary Information were analysed using MATLAB version R2015a. All the codes can be made available upon request.

Data availability. No datasets were generated or analysed during the current study.

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Author contributions

C.N.N. and M.H.B. conceived the paper, C.N.N. conducted analysis. All authors performed research and wrote the paper.

Competing interests

The authors declare no competing financial interests.

Additional information

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