International Economic Review

ORIGINAL ARTICLE

Growth with Deadly Spillovers

Pietro F. Peretto[1] | Simone Valente[2]

[1]Department of Economics, Duke University, Durham, North Carolina, Durham, North Carolina, USA | [2]Department of Economics, Norwegian University of Science and Technology (NTNU), Trondheim, Norway

Correspondence: Pietro F. Peretto (pp7@duke.edu)

Received: 26 April 2022 | **Revised:** 2 December 2024 | **Accepted:** 20 February 2025

Keywords: endogenous growth | environmental externalities | mortality

ABSTRACT

Pollution causes premature deaths but plays almost no role in macroeconomic analysis. To fill this gap, we build a tractable model of innovation-led economic growth and endogenous fertility in which production generates deadly spillovers, that is, emissions that increase mortality. Such spillovers affect income growth, population size, and welfare. They also create steady states that would not exist otherwise. Emission taxes increase population size and may even raise long-run growth if they reduce mortality. Subsidies to primary production have opposite effects and may push resource-rich economies toward economic and demographic implosion. Cross-country regressions support hypotheses and predictions of the model.

JEL Classification: O12, O44, Q56

1 | Introduction

Pollution is one of the world's most significant causes of premature death (WHO 2016) and this phenomenon is by no means limited to underdeveloped countries. The Lancet Commission estimates that pollution is responsible for 9 million deaths every year, an increasing share of which is not connected to conditions of extreme poverty but rather to the unintended consequences of industrialization and urbanization.[1] The economics literature on the subject is mostly empirical and confirms the scale and pervasiveness of the problem (e.g., Ebenstein et al. 2015; Arceo et al. 2016). Despite this evidence, however, macroeconomic analysis neglects the role of *deadly spillovers*: there are no models that account for the simultaneous endogeneity of economic growth, environmental degradation, mortality, and fertility. This type of models are however necessary to address fundamental questions, first and foremost: how does pollution affect macroeconomic performance through excess deaths?

Unlike the conventional pollution externalities studied in environmental economics, that is, emissions that reduce the utility of individuals and/or the efficiency of firms, deadly spillovers work through excess deaths that reduce labor supply and household expenditure, activate reallocation of resources across sectors—including the polluting primary sector and R&D activities that drive productivity growth—and prompt households to revise saving and fertility decisions. Understanding how these propagation channels determine macroeconomic performance is a necessary first step to study a number of questions of direct interest to empirical research and policymaking. What are the effects of deadly spillovers on income dynamics when we account for demographic change, in particular when both fertility and mortality are endogenous? What are the short- and long-run effects of pollution taxes and/or subsidies to polluting sectors? What are the consequence of pollution-caused mortality in less populated resource-rich countries that typically display high emissions per capita? Is population implosion a possible outcome in the long run?

The answers to such questions hinge on how the mortality rate responds to changes in population size. We study this mechanism in a model where a polluting primary sector exploits

a natural resource, horizontal and vertical innovations raise the productivity of intermediate producers, emissions increase mortality, and household choices determine fertility. A distinctive property of our framework is that it produces equilibrium paths where population converges to a finite size while income per capita grows via endogenous innovation. This property extends the results derived in Peretto and Valente (2015), which builds a theory of finite population on a finite planet. In contrast to many balanced-growth models that predict exponential population growth, the framework replicates the fertility decline experienced in most industrialized countries while being consistent with the view in demography that population will eventually stop growing due to the feedback effects operating in a closed system, that is, a finite planet. [2] In Peretto and Valente (2015), there is no pollution, mortality is exogenous, and population growth eventually stops because of the fertility response to income per capita. We introduce pollution externalities and endogenous mortality, obtaining a model where the *mortality response to pollution* affects economic growth and welfare, and becomes an independent force stabilizing the population level in the long run. Our main results are as follows.

First, the equilibrium relationship between the mortality rate and population size depends on two main forces: the *primary-employment* effect and the *damage-dilution* effect. The former summarizes the causal link between labor supply and pollution generation: higher population increases total labor inducing higher employment in the primary sector and higher emissions from commodity production. The damage-dilution effect incorporates two distinct mechanisms: dose dilution—that is, the reduction in individual absorption of pollutants when population increases at given total emissions—and emission reduction—that is, the reduction in individual exposure occurring when a larger population causes emissions per capita to fall. Since the primary-employment and damage-dilution effects push mortality in opposite directions, the equilibrium relationship between mortality rates and population size is strongly nonlinear and may be increasing, decreasing, or nonmonotonic—in particular, U-shaped.

Second, under broad conditions the economy converges to a *regular* steady state where the population is constant and income per capita grows due to endogenous R&D-based innovations. Deadly spillovers modify the position of the regular steady state and the path leading to it: equilibrium dynamics are quantitatively different, and pollution-related deaths affect growth and welfare through channels that the existing literature neglects. This result provides new answers to traditional policy questions, like the effects of emission taxes or sectoral subsidies. Deadly spillovers may even create mortality traps, that is, unstable steady states that split the state space in two basins of attraction: one for the regular steady state and one for the *extinction* steady state. If the initial population-resource ratio is below a critical threshold, the population implodes due to increasing mortality despite growing fertility rates. We also show that the existence of deadly spillovers can create regular steady states that would not exist otherwise, that is, endogenous pollution-caused mortality stabilizes the population even in the absence of other well-understood mechanisms.

Third, the demographic development of the economy has first-order effects on its economic development. This follows from the model's Schumpeterian block, which features endogenous R&D-driven innovation that responds to the dynamics of market size. The causal link is that population size is the key driver of market size. Along regular equilibrium paths, population growth expands market size and feeds transitional productivity growth through horizontal innovations that raise the number of firms. In the regular steady state, productivity growth is exclusively driven by the rate of vertical innovations, which is higher the lower the mass of firms relative to population. Deadly spillovers affect both mechanisms because horizontal and vertical innovations depend on population dynamics and thereby on endogenous mortality. In particular, we show that exogenous shocks that increase the long-run population level *and* reduce the long-run mortality rate will typically yield a "double growth dividend": population growth accelerates productivity growth via firms' entry during the transition, while a lower mortality rate increases long-run productivity growth via higher investment in vertical innovations. The fact that deadly spillovers create a channel through which the deep parameters regulating mortality have steady-state growth effects is a novel result in itself since our Schumpeterian framework belongs to a class of models known for the scale invariance of the steady-state growth rate. This is not a manifestation of the traditional scale effect—a causal relationship running from the exogenous supply of labor to growth—but rather a distinctive outcome of our model where the (endogenous) dynamics of the population-resource ratio affect the (endogenous) dynamics of the mortality rate.

Fourth, deadly spillovers matter for environmental policy and the assessment of the effects of resource booms, that is, discoveries of new natural endowments. Taxing polluting primary sectors yields a demographic double dividend: it increases the economy's carrying capacity of people, meaning that a given resource base supports a larger population in steady state, and it also reduces the size of the mortality trap. Also, taxing the polluting sector accelerates transitional productivity growth via horizontal innovations that expand the mass of firms along with population. In the long run, the tax may even yield an economic growth dividend by increasing the steady-state rate of vertical innovations: this will happen if the damage-dilution effect is sufficiently strong to guarantee a lower equilibrium mortality rate in the new steady state. Importantly, subsidies to the primary sector yield opposite effects and may be a recipe for disaster if they are implemented after resource booms.

Fifth, we provide cross-country evidence supporting hypotheses and predictions of the theoretical model. Our regressions use the pollution-attributed deaths estimated by the Lancet Commission for the year 2019 and concentrations of particulate matter (PM) for 180 economies. Our results support the existence of dose dilution (i.e., lower population density is associated to higher excess mortality rates at given emissions and population levels) and of emission-reducing effects (i.e., higher population density leads to lower emissions)—with estimated coefficients compatible with the hypothesis of balanced dilution (i.e., mortality rates depend on pollution per capita)—and support the theoretical

prediction that resource-rich countries tend to have higher excess mortality rates.

Our analysis contributes to several literatures. It contributes to growth economics by providing a full account of demography–economy interactions when all the underlying determinants—fertility, mortality, and innovation—are fully endogenous and produce a finite population. The view that demography matters for macroeconomic performance is well established but rarely implemented in models that produce finite population. The few growth models that do typically focus on Malthusian mechanisms (Brander and Taylor 1998; Eckstein et al. 1988; Galor and Weil 2000) or similar market-based mechanisms where resource scarcity causes relative-price dynamics that eventually bring population growth to a halt (Peretto and Valente 2015; Strulik and Weisdorf 2008).[3] While they provide useful insights, none of these works study pollution-caused mortality.

The few existing theories that link emissions to mortality assume that pollution reduces life expectancy in models of capital accumulation; see Mariani et al. (2010), Varvarigos (2014), and Goenka et al. (2020). In this framework, the average death rate grows with emissions but people can undertake defensive expenditures that mitigate the effect, which can create multiple steady states at different income levels.[4] These conclusions relate to Nelson's (1956) notion of underdevelopment traps: nonlinearities in the returns to investment generate regular high-income and low-income steady states that constitute poverty traps.[5] Our analysis differs from these contributions in two key dimensions. First, we use a Schumpeterian model of endogenous R&D-based innovation where demography and productivity dynamics eventually decouple: with or without pollution, the regular steady state features a constant endogenous population level while income per capita grows at a constant rate. This property yields distinctive predictions—like the impact of demography on innovations and the dividends generated by environmental taxes—that cannot be replicated in one-sector models of the neoclassical type. Second, we specify a mortality function that includes population-exposure interactions in the form of dose dilution, which occurs at the point of contact between humans and pollutants, and emission reduction, which occurs at the point of origin of pollutants as emissions stem from human activity. In our model, the nonmonotonic response of mortality to population is generated by these population-exposure interactions, not by nonlinearity in rates of return. Consequently, our mortality traps are conceptually distinct from the poverty traps discussed in development economics: demographic implosion is triggered by a low population-resource ratio, not by low income levels.

Our result that the emission tax can generate a demographic dividend and an economic growth dividend is a novel contribution to the literature on environmental macroeconomics. The traditional notion of a double dividend is that emission taxes can reduce aggregate efficiency losses by shifting distortionary taxes from clean to dirty inputs (Bovenberg and Goulder 2002). A complementary notion is that emission taxes can encourage innovation (Porter and van der Linde 1995). Neither mechanism works through demography, whereas our result follows entirely from the endogenous demographic response to deadly spillovers. The demographic response to environmental taxes is seldom studied, with a few exceptions (e.g., de la Croix and Gosseries 2012) that contrary to our model consider negative demographic dividends and suggest that limiting population is welfare improving.[6]

Our analysis of subsidies is relevant from a policy perspective because resource-rich developing countries often subsidize polluting primary sectors by invoking the need to boost income via resource rents (Bretschger and Valente 2018). In our model, subsidies reduce the population and push the economy away from the steady state and toward the mortality trap. Our result on the effects of resource booms contributes to the literature on the Resource Curse hypothesis, which studies the mechanisms through which natural resource abundance undermines economic performance (e.g., Mehlum et al. 2006). The idea that a resource curse could actually arise via demography is to our knowledge completely novel and deserves investigation. Also, our analysis is relevant for the macroeconomic models used by researchers and international organizations to forecast future demographic trends and calculate the welfare cost of pollution (e.g., OECD 2016), as we argue in our conclusions (Section 7).

2 | The Model

We study a decentralized economy where the competitive primary sector produces a commodity using labor and a raw natural resource. The monopolistically competitive intermediate sector uses the commodity to produce differentiated goods that the competitive final sector uses to produce a homogeneous consumption good. Endogenous economic growth results from horizontal and vertical innovations in the intermediate sector. The decisions of households facing child-rearing costs drive endogenous fertility. Commodity production and household activities generate harmful pollution that increases mortality.

2.1 | Demography with deadly spillovers

Central to our model is the relation between population and pollution-induced mortality which comprises two main channels. The first channel, discussed in this subsection, is the mortality function whereby population size affects the individual exposure to pollutants at given total emissions. The second channel, discussed in Subsection 2.2, is the pollution generation process which depends on population through multiple mechanisms: emissions increase with primary production—which includes labor as an input—and with household activities entailing a positive "scale effect" from population size and a negative "density effect" from population density.

Time is continuous and indexed by $t \in [0, \infty)$. The dynamics of population, L, is

$$\dot{L}(t) = B(t) - M(t) = [b(t) - m(t)] \cdot L(t), \tag{1}$$

where B is births and M is deaths. For future use, we also specify the dynamics in terms of the birth rate, $b = B/L$, and the death rate, $m = M/L$. The novel ingredient is the function

$$M(t) = \underbrace{\bar{m}L(t)}_{\text{baseline deaths}} + \underbrace{(1-\bar{m})L(t) \cdot D(t)}_{\text{excess deaths caused by pollution, } M_p} \tag{2}$$

that decomposes total deaths in baseline deaths unrelated to pollution, $\bar{m}L$, and excess deaths caused by pollution, M_p. The exogenous constant $\bar{m} > 0$ is the baseline mortality rate that prevails in the absence of deadly spillovers. Deaths from pollution are a fraction D of $(1-\bar{m})L$, the mass of people that survive the baseline causes of death. Drawing on the insights developed in several literatures, we model D as the ratio between the flow of excess deaths due to pollution and the population. The former is the output of a matching process, $f(E,L)$, with two inputs: the population, L, as the measure of the mass of individuals that can potentially absorb harmful pollutants, and aggregate emissions, E.[7] The fraction D, therefore, is the outcome of a process in which individuals and pollutants collide at random. Each collision results in an individual's exposure to and possible absorption of the pollutants, an event that the literature calls *dose absorption*, which can result in the death of the individual. Given this interpretation and its construction, in the language of matching models we call D the *dose-absorption rate*.

Formally, we write the matching process as a differentiable function increasing in each one of its inputs, that is, $f_E(\cdot) > 0$ and $f_L(\cdot) > 0$. We also reasonably assume that each input is essential, that is, $f(0,L) = f(E,0) = 0$. To maximize tractability, we write

$$D(t) = \frac{f(E(t),L(t))}{L(t)} = \mu_0 \cdot E(t)^{\chi} \cdot L(t)^{-\chi\zeta}, \tag{3}$$

where $\chi > 0$ and $0 \leq \zeta < 1/\chi$. With this representation, excess deaths caused by pollution are

$$M_p(t) = (1-\bar{m})L(t) \cdot D(t) = \mu E(t)^{\chi} L(t)^{1-\chi\zeta}, \tag{4}$$

where $\mu = \mu_0(1-\bar{m}) > 0$ collects all the constant terms and $0 < 1-\chi\zeta \leq 1$. For future use, we define the *pollution-caused excess mortality rate*

$$m_p(t) \equiv \frac{M_p(t)}{L(t)} = \mu \cdot \left(\frac{E(t)}{L(t)^{\zeta}}\right)^{\chi}, \tag{5}$$

where the denominator L^{ζ} captures the *dose-dilution effect* , that is, the property that for given emissions E a larger population reduces individual dose absorption. Letting ζ vary between zero and one we obtain three cases of particular interest:

$$\begin{aligned}
&\zeta = 1 && \to M_p = \mu E^{\chi} L^{1-\chi}, && m_p = \mu \cdot (E/L)^{\chi}; && \text{balanced dose dilution}\\
&0 < \zeta < 1 && \to M_p = \mu E^{\chi} L^{1-\zeta\chi}, && m_p = \mu \cdot \left(E/L^{\zeta}\right)^{\chi}; && \text{weak dose dilution}\\
&\zeta = 0 && \to M_p = \mu E^{\chi} L, && m_p = \mu \cdot E^{\chi}. && \text{no dose dilution}
\end{aligned}$$

In the first polar case, $\zeta = 1$, the excess mortality rate depends on emissions per capita. The opposite polar case, $\zeta = 0$, yields no dilution: population size does not affect individual dose absorption and thus the excess mortality rate depends on aggregate emissions. In the intermediate case, $0 < \zeta < 1$, the excess mortality rate depends on aggregate emissions and population size with different elasticities.[8] By generally assuming $\zeta > 0$, we deviate from the practice, often encountered in the climate-change literature, of assuming that each individual is equally harmed by aggregate pollution in a nonrival way (i.e., $\zeta = 0$). We discusses this important point in Section 6.

2.2 | Pollution Generation and Emission-Reducing Effects

Pollution has two sources: commodity production (e.g., mining or generation of energy from fossil fuels) and household or, equivalently, personal activities (e.g., transport services, waste disposal, and residential use of environmental amenities). Pollution generation thus takes the form

$$E(t) = \Gamma(E_{\omega}(t), E_h(t)), \quad \frac{\partial\Gamma(E_{\omega},E_h)}{\partial E_{\omega}} > 0, \quad \frac{\partial\Gamma(E_{\omega},E_h)}{\partial E_h} > 0, \tag{6}$$

where E_{ω} is emissions from commodity production and E_h is emissions from household activities.

Commodity production is $Q = \mathcal{F}(\Omega, L_Q)$ where Q is output, L_Q is the labor input, and $\mathcal{F}$ is a linearly homogeneous function with positive and diminishing marginal productivities for each input. For simplicity, we model the resource input as the constant flow, Ω, of productive services from a fixed endowment.[9] Resource processing generates one unit of emissions per unit of output, that is,

$$E_{\omega}(t) = Q(t) = \mathcal{F}\big(\Omega, L_Q(t)\big). \tag{7}$$

The representation $E_{\omega} = Q$ is not restrictive since further elasticities come into play when we consider the other source of pollution.

Household emissions per person is $E_h/L = \Psi(L)$. In line with the literature, we interpret the argument of this function as population density, that is, population L in a reference geographical area that we normalize to unity (e.g., people per square mile). It would be tempting to assume that a larger population L produces more emissions, $E_h = L \cdot \Psi(L)$, simply because $\Psi'(L) \geq 0$. However, a growing body of literature on urbanization documents density effects that yield $\Psi'(L) < 0$. Empirically, less populated areas tend to exhibit higher emissions per capita and, in some cases, also higher aggregate emissions (Stone 2008). One explanation is that high density allows people to pursue personal activities in less polluting ways (e.g., public transport instead of individual transport) by providing stronger incentives (e.g., congestion effects) or better access to pollution-saving technologies (e.g., infrastructure with strong economies of scale). A second explanation is that pollution abatement activities, private and public, are more likely to take place in high-density areas due to stronger individual awareness and public support for tighter regulations. These and similar arguments suggest that population density reduces the pollution intensity of household activities (see Bork and Schrauth 2021, and the literature cited therein). If this effect is sufficiently strong, population size has a generally ambiguous effect on personal emissions, that is, at least over some range we cannot rule out $dE_h/dL < 0$.

These considerations suggest that the effect of population size on aggregate emissions comprises three main channels:

$$\frac{dE}{dL} = \underbrace{\frac{\partial\Gamma(E_{\omega},E_h)}{\partial E_{\omega}} \cdot \frac{dE_{\omega}}{dL}}_{\text{labor supply} > 0} + \underbrace{\frac{\partial\Gamma(E_{\omega},E_h)}{\partial E_h} \cdot \Psi(L)}_{\text{scale} > 0} + \underbrace{\frac{\partial\Gamma(E_{\omega},E_h)}{\partial E_h} \cdot L \cdot \Psi'(L)}_{\text{density} < 0}.$$

This decomposition identifies two empirically relevant cases. Weak emission reduction occurs when a larger population raises aggregate emissions but reduces emissions per capita. This requires $0 < (dE/dL)(L/E) < 1$. Strong emission reduction occurs when the larger population reduces emissions in both aggregate and per capita terms. This requires $(dE/dL)(L/E) < 0$. The recent empirical literature estimates the elasticity $(dE/dL)(L/E)$ using population density and ground-level concentrations as the measures of L and E. These estimates suggest that strong emission reduction holds for ozone while weak emission reduction appears to hold for air pollution.[10]

We work with the functional form $E = E_\omega^{\upsilon} E_h^{1-\upsilon} = Q^{\upsilon}(L\Psi(L))^{1-\upsilon}$, with $0 < \upsilon < 1$ and $\Psi(L) = L^{-(1+\xi)}$, where $\xi \geqslant -1$. If $\xi < 0$, density effects are weaker than scale effects in household emissions and E_h increases with the population. The reverse occurs if $\xi > 0$. Next, we define the elasticity of commodity output with respect to population size

$$\varepsilon_{Q,L} \equiv \frac{dQ}{dL_Q} \cdot \frac{L_Q}{Q} = \left(\frac{\partial Q}{\partial L_Q} \cdot \frac{L_Q}{\mathcal{F}}\right) \cdot \left(\frac{dL_Q}{dL} \cdot \frac{L}{L_Q}\right). \tag{8}$$

Note that this definition allows for the general equilibrium dependence of primary employment, L_Q, on labor supply, L, and is thus a macroeconomic object. This structure yields the elasticity

$$\frac{dE}{dL} \cdot \frac{L}{E} = \underbrace{\upsilon \cdot \varepsilon_{Q,L}}_{\text{labor supply}} - \underbrace{\xi(1-\upsilon)}_{\text{emission reduction}} \tag{9}$$

which shows that aggregate emissions depend on population through two main channels: labor supply as the driver of primary employment, and the balance between scale and density effects in household emissions. Expression (9) identifies parametric conditions for weak or strong emission reduction that depend on the properties of the production technology of the primary sector.

In our theory, we abstract from defensive investments and/or pollution abatement expenditures in order to keep the model tractable. These elements would be relevant because they affect net emission generation and thereby mortality outcomes. There is evidence that economic development brings about reductions in per capita emissions through the defensive channel (e.g., Ordás Criado et al. 2011) and we stress that endogenous reactions to pollution are a major reason behind the negative pollution elasticities to real GDP per capita that we find in our cross-country regressions (see Subsection 6.3).

2.3 | Mortality, Employment, and Damage Dilution

From (2), (3), and (6), the crude mortality rate equals

$$\underbrace{m(t)}_{\text{crude}} = \underbrace{\bar{m}}_{\text{baseline}} + \underbrace{\mu \cdot Q(t)^{\chi\upsilon} \cdot L(t)^{-\chi[\zeta+\xi(1-\upsilon)]}}_{\text{excess death rate } m_p(t)}. \tag{10}$$

Given primary production, Q, population size, L, reduces the excess death rate via dose dilution at given total emissions and via emission reduction from population density. We can thus define the overall *damage-dilution effect* of larger population at given primary production as

$$\chi[\zeta + \xi(1-\upsilon)] = \underbrace{\text{damage intensity} \cdot [\text{dose dilution} + \text{emission reduction}]}_{\text{per capita damage reduction (given } Q)}. \tag{11}$$

The sign of the damage-dilution effect is a priori ambiguous but it can be positive under a variety of plausible circumstances because dose dilution and emission reduction operate independently and can substitute for each other. Considering pollutants for which dose dilution is substantial, like water-contaminating elements, damage dilution may be positive even if population density does not yield substantial emissions reduction, that is, $\zeta > 0$ with $\xi(1-\upsilon)$ negligible. Symmetrically, pollutants for which population density induces substantial emissions reduction, like ozone, exhibits positive damage dilution even if we treat O3 doses as nonrival, that is, $\zeta = 0$ with $\xi(1-\upsilon)$ positive. For intermediate cases where dose dilution and emissions reduction can be positive but moderate, like PM, damage dilution can still be positive. Total differentiation of (10) yields

$$\frac{dm_p}{dL} \cdot \frac{L}{m_p} = \underbrace{\chi\upsilon\varepsilon_{Q,L}}_{\text{primary-employment effect}} - \underbrace{\chi[\zeta + \xi(1-\upsilon)]}_{\text{damage-dilution effect}}. \tag{12}$$

This expression shows that larger population increases or decreases the mortality rate depending on the difference of two effects that fully summarize the several channels identified above. This property drives our analysis of the equilibrium path of the economy.

2.4 | Consumption and Reproduction Choices

We use the Peretto and Valente (2015) extension of the textbook formulation of fertility theory (see, e.g., Barro and Sala-i-Martin 2004, Ch. 9). The extension gives full control over expenditure per child to the household and allows for a "quantity–quality" trade-off with no additional complexity. Specifically, a representative household maximizes the dynastic utility function

$$U_0 = \int_0^\infty \mathrm{e}^{-\rho t} \ln u(C_L(t), C_B(t), L(t), B(t))dt, \quad \rho > 0, \tag{13}$$

where ρ is the individual discount rate, C_L is consumption of the adults, C_B is consumption of the children, L is the stock of adults, and B is the instantaneous flow of newly born children per unit of time. Instantaneous utility is

$$u(C_L, C_B, L, B) = \left(\frac{C_L}{L}\right)^{\alpha} \left(\frac{C_B}{B}\right)^{1-\alpha} \left(L^{\alpha} B^{1-\alpha}\right)^{\psi}, \quad 0 < \alpha < 1, \quad 0 < \psi < 1. \tag{14}$$

In this structure, agents obtain utility from the consumption and presence of adults and from the consumption and presence of children with weights, respectively, α and $1-\alpha$.[11] The parameter ψ regulates the trade-off between the individual consumption of the members of each group (adults and children) and the size of each group.[12]

Household expenditure is $Y = p_c C_L + p_c C_B$, where p_c is the price of the final good. The fertility choice is thus characterized by a trade-off between the utility benefit from reproduction and expenditure on the children's consumption. The price-taking

household supplies the services of labor and of the natural resource inelastically. The household's budget is

$$\dot{A}(t) = r(t)A(t) + w(t)L(t) + p_{\omega}(t)\Omega + S(t) - Y(t), \tag{15}$$

where r is the rate of return on financial assets, A is asset holdings, w is the wage, p_{ω} is the per-unit resource royalty, and Ω is the natural resource endowment over which the household has full property rights. The household chooses the time paths of C_L, C_B, and B to maximize (13) subject to (15) and (1). The household takes the path of the mortality rate as given because private agents are unable to internalize the effects of emissions on mortality. Nonetheless, the household internalizes the intertemporal trade-off caused by population growth: a larger mass of adults expands the dynasty's consumption possibilities via additional labor income but, at the same time, reduces individual consumption possibilities via dilution effects.

The solution to the household problem is described in the Appendix. The conditions for utility maximization are the familiar Euler equation for consumption growth

$$\frac{\dot{Y}(t)}{Y(t)} = r(t) - \rho \tag{16}$$

and the associated equation for the birth rate,

$$\frac{\dot{b}(t)}{b(t)} = \frac{b(t)}{(1-\alpha)(1-\psi)}\left[\psi + \frac{w(t)L(t) - Y(t)}{Y(t)}\right] - \rho. \tag{17}$$

Equation (16) determines the growth rate of household consumption expenditure according to the traditional trade-off: the marginal benefit of asset accumulation versus the marginal cost of sacrificing current consumption. Equation (17) says that the birth rate increases over time when the anticipated rate of return from generating future adults exceeds the utility discount rate, ρ. The term in square brackets shows the components of this rate of return: the gross elasticity of utility to the mass of adults, ψ, plus their contribution to asset accumulation, given by the difference between labor income and consumption expenditure.

2.5 | Producers: Final and Intermediate Sectors

Final sector. The final sector is competitive and produces with the technology

$$C(t) = \left(\int_0^{N(t)} x_i(t)^{\frac{\epsilon-1}{\epsilon}} di\right)^{\frac{\epsilon}{\epsilon-1}}, \quad \epsilon > 1, \tag{18}$$

where C is output, N is the mass of intermediate goods, x_i is the quantity of good i, and ϵ is the elasticity of substitution between pairs of intermediate goods. Final producers maximize profits taking as given the mass of intermediate goods and the price, p_{x_i}, of each intermediate good. The solution to this problem yields the demand schedule

$$x_i(t) = \frac{Y(t)}{\int_0^{N(t)} p_{x_i}(t)^{1-\epsilon} di} \cdot p_{x_i}(t)^{-\epsilon} \tag{19}$$

for each intermediate good, where $P_C \equiv \left[\int_0^{N(t)} p_{x_i}(t)^{1-\epsilon} di\right]^{\frac{1}{1-\epsilon}}$ is the price index of intermediate goods.

Intermediate sector: incumbents. Each intermediate good is supplied by a monopolist that operates the production technology

$$x_i(t) = z_i(t)^{\theta} \cdot Q_{x_i}(t)^{\gamma}\left(L_{x_i}(t) - \phi\right)^{1-\gamma}, \quad 0 < \theta < 1, \quad 0 < \gamma < 1, \tag{20}$$

where x_i is output, Q_{x_i} is the commodity input, L_{x_i} is production labor, and $\phi > 0$ is a fixed operating cost.[13] The productivity term z_i^{θ} is Hicks-neutral with respect to the rival inputs, labor, and the commodity, and depends on the stock of firm-specific knowledge z_i. The firm's cost minimization problem yields the total cost function

$$TC_i\left(x_i(t); w(t), p_q(t)\right) = w(t)\phi + \gamma^{-\gamma}(1-\gamma)^{-1+\gamma} p_q(t)^{\gamma} w^{1-\gamma} z_i(t)^{-\theta} x_i(t) \tag{21}$$

and the associated conditional factor demands

$$L_{x_i}(t) = (1-\gamma)\frac{\epsilon-1}{\epsilon} \cdot \frac{p_{x_i}(t)x_i(t)}{w(t)} + \phi; \tag{22}$$

$$Q_{x_i}(t) = \gamma\frac{\epsilon-1}{\epsilon} \cdot \frac{p_{x_i}(t)x_i(t)}{p_q(t)}. \tag{23}$$

The firm accumulates firm-specific knowledge according to the technology

$$\dot{z}_i(t) = \kappa \cdot \left[\int_0^{N(t)} \frac{1}{N(t)} z_j(t) dj\right] \cdot L_{z_i}(t), \quad \kappa > 0, \tag{24}$$

where L_{z_i} is R&D labor, κ is an exogenous parameter, and the term in bracket is the stock of public knowledge that accumulates as a result of spillovers across firms: when one firm develops a new idea, it also generates nonexcludable knowledge that benefits the R&D of other firms. The firm's instantaneous profit is

$$\pi_i(t) = \left[p_{x_i}(t) - \gamma^{-\gamma}(1-\gamma)^{-1+\gamma} p_q(t)^{\gamma} w^{1-\gamma} z_i(t)^{-\theta}\right] x_i(t) - w(t)\phi - w(t)L_{z_i}(t). \tag{25}$$

The value of the firm is

$$V_i(t) = \int_t^{\infty} \pi_i(v) \exp\left(-\int_t^v (r(s) + \delta) ds\right) dv, \quad \delta > 0, \tag{26}$$

where δ is an exit shock. (To avoid confusion with the death rate of people, m, we refer to δ as the exit rate.) At time t, the firm chooses the paths $\{p_{x_i}, L_{z_i}\}$ that maximize (26) subject to (19) and (24). The solution to this problem (see the Appendix) yields the maximized value of the firm given the time path of the mass of firms, $N(t)$. Under the assumption $z_i(t) = z(t)$, that is, all incumbent firms start with the same stock of knowledge, the equilibrium is symmetric. That is, at each instant t each monopolist charges the same price $p_{x_i} = p_x$ and produces the same quantity $x_i = x$. Combining this result with the final producer's behavior, we obtain

$$p_x(t)x(t) = \frac{Y(t)}{N(t)}. \tag{27}$$

This equation says that aggregate intermediate sales equal consumption expenditure, Y, and that each monopolist captures a share, $1/N$, of the market.

Intermediate sector: entrants. Entrepreneurs hire labor to develop new intermediate goods and set up firms to serve the market. Denoting the typical entrant i without loss of generality, and denoting L_{N_i} the amount of labor required to start the new firm that enters the market with knowledge $z_i(t)$ equal to the industry average, the cost of entry is $wL_{N_i} = w\beta L/N$, where $\beta > 0$ is a parameter representing technological opportunity.[14] The entrant anticipates that once in the market the new firm solves an intertemporal problem identical to that of the generic incumbent and therefore that the value of the new firm is the maximized value $V_i(t)$ defined in (26). Free entry then requires

$$V_i(t) = w(t)L_{N_i}(t) = w(t)\beta L(t)/N(t) \tag{28}$$

for each entrant.

2.6 | Primary Sector

Since the intermediate sector is symmetric, we write the quantity of the commodity demanded by intermediate producers as $Q = NQ_{x_i}$, with Q_{x_i} given by (23). A representative competitive firm produces the commodity by combining the resource with labor under constant returns to scale. The firm maximizes profit

$$\Pi_q = p_q(t)Q(t)(1-\tau) - p_\omega(t)\Omega - w(t)L_Q(t) \tag{29}$$

subject to the technology (7) taking all prices and the tax rate as given. To simplify the exposition, we work with the CES specification of (7)

$$Q(t) = \mathcal{F}\left(\Omega, L_Q(t)\right) = \left[\eta \cdot \Omega^{\frac{\sigma-1}{\sigma}} + (1-\eta)\cdot L_Q(t)^{\frac{\sigma-1}{\sigma}}\right]^{\frac{\sigma}{\sigma-1}},$$
$$\sigma \geq 0,\ \eta \in (0,1), \tag{30}$$

where σ is the elasticity of input substitution and η governs the input shares. The resource and labor are complements if $\sigma < 1$ and substitutes if $\sigma > 1$. Letting $\sigma \to 1$ we obtain the Cobb–Douglas case $Q = \Omega^{\eta} L_Q^{1-\eta}$. Let $\Theta(w, p_\omega) \equiv \eta^\sigma p_\omega^{1-\sigma} + (1-\eta)^\sigma w^{1-\sigma}$ denote the unit cost function associated to the technology (30). The profit-maximizing decisions of the commodity producer yield

$$p_q = \frac{\Theta(w, p_\omega)}{1-\tau} \tag{31}$$

and the resource *cost-share* function (see the Appendix)

$$\mathrm{Y}(t) \equiv \frac{d\ln\Theta(w(t), p_\omega(t))}{d\ln p_\omega(t)} = \frac{p_\omega(t)\Omega}{p_\omega(t)\Omega + w(t)L_Q(t)}$$
$$= \frac{\eta^\sigma p_\omega(t)^{1-\sigma}}{\eta^\sigma p_\omega(t)^{1-\sigma} + (1-\eta)^\sigma w(t)^{1-\sigma}}. \tag{32}$$

The resource cost share Y is the ratio between royalties paid by firms to resource owners and the firm's total expenditures on inputs. In the Cobb–Douglas case, $\sigma \to 1$, the cost share is constant, $\mathrm{Y} \to \eta$. In the other cases, a higher resource price reduces (increases) the resource cost share when primary inputs are substitutes (complements). These *cost-share effects* determine the equilibrium response of household income and consumption expenditure to changes in the relative scarcity of the resource, as we show below.

3 | Equilibrium and the Mortality Rate

This section summarizes the key interactions taking place in equilibrium between demographic and economic variables. Expenditures per capita reflect the response of income to changes in resource scarcity, while mortality responds to changes in the population-resource ratio according to precise relationship among the equilibrium mortality rate, per capita emission damages, and the allocation of labor to commodity production.

3.1 | Output and Input Markets

To determine the general equilibrium of the economy, we impose several market clearing conditions. The resource market clears when supply by the representative household equals demand by the representative commodity producer, that is, when

$$p_\omega(t)\Omega = \mathrm{Y}(t)\cdot p_q(t)Q(t)(1-\tau). \tag{33}$$

This equation says that the commodity producer spends on the resource a fraction Y of the after-tax value of its sales, where Y is the cost-share function defined in (32). The commodity market clears when supply by the commodity producer equals demand by intermediate firms. Using (23) and (27), we obtain

$$p_q(t)Q(t) = \gamma\frac{\epsilon-1}{\epsilon}\cdot Y(t). \tag{34}$$

The labor market clears when $L = L_X + L_Z + L_N + L_Q$, where L is labor supply, $L_X + L_Z = N\left(L_{x_i} + L_{z_i}\right)$ is labor demand by intermediate producers (for production and in-house R&D), $L_N = \dot{N}L_{N_i}$ is labor demand by entrants, and L_Q is labor demand by the primary sector. Finally, the financial market clears when the value of the household's portfolio equals the value of the securities issued by firms, $A = NV_i$. The free-entry condition (28) then yields

$$A(t) = \beta w(t)L(t). \tag{35}$$

In the remainder of the analysis, we normalize the wage, $w(t) \equiv 1$. This choice of numeraire implies that expenditure on final output, Y, is an index of the value added of labor services.[15] Also, we let $y \equiv Y/L$ denote *consumption expenditure per capita* and $\ell \equiv L/\Omega$ denote the ratio of labor supply (population) to resource supply, henceforth *population-resource ratio* for short. High ℓ represents relative abundance of labor or, equivalently, relative scarcity of the resource.

3.2 | Expenditure and Resource Use

Two relationships between consumption expenditure and resource income characterize the intratemporal equilibrium of the economy (see the Appendix for the derivation). The first

follows from combining the household's budget constraint (15) and the Euler equation (16) with the equilibrium condition of the assets market (35). It reads

$$y(t) = \frac{1 + \beta\rho + \frac{p_\omega(t)}{\ell(t)}}{1 - \tau\gamma\frac{\epsilon-1}{\epsilon}} \tag{36}$$

and says that consumption expenditure per capita, y, is a constant fraction of income per capita, the sum of the wage, $w = 1$, asset income per capita, $\rho A/L = \beta\rho w = \beta\rho$, and resource income per capita, $p_\omega\Omega/L = p_\omega/\ell$. The presence of the commodity tax at the denominator is due to the balanced-budget assumption and captures the positive effect of public transfers on household expenditure. The second relationship follows from (33) and (34). It reads

$$\frac{p_\omega(t)}{\ell(t)} = \left[(1-\tau)\cdot \mathrm{Y}(p_\omega(t))\cdot\gamma\frac{\epsilon-1}{\epsilon}\right]\cdot y(t) \tag{37}$$

and says that resource income per capita is a fraction (in brackets) of consumption expenditure per capita. We call this fraction the *royalty share*.

The royalty share depends on the technological parameters of all production sectors and on the commodity tax. The tax reduces the royalty share despite the lump-sum rebate because it distorts the use of the commodity in primary production and thus generates a traditional deadweight loss. With $w = 1$, the resource cost share defined in (32) is a function $\mathrm{Y} \equiv \mathrm{Y}(p_\omega)$ of the resource price only. Therefore, Equations (36) and (37) form a system of two equations in three variables (y, p_ω, ℓ). To characterize the interaction of the resource market equilibrium with household consumption-saving decisions, we solve for the resource price p_ω and expenditure per capita y as functions of the population-resource ratio ℓ.

Proposition 1. *Given population-resource ratio $\ell(t) > 0$, at each instant $t \in [0, \infty)$ the solution of Equations (36)–(37) yields a unique equilibrium pair*

$$\{p_\omega^*(\ell(t)), y^*(\ell(t))\}$$

with the following properties. The resource price is monotonically increasing in the population-resource ratio, that is, $\mathrm{d}p_\omega^*(\ell)/\mathrm{d}\ell > 0$ for all $\ell > 0$. The effect of the population-resource ratio on expenditure per capita, instead, depends on the elasticity of substitution between inputs in commodity production. In terms of elasticity,

$$\frac{d\ln y^*(\ell)}{d\ln\ell} = (1-\tau)\gamma\frac{\epsilon-1}{\epsilon}\ell\, y^*(\ell)\cdot\frac{d\mathrm{Y}(p_\omega(\ell))}{d\ell},$$

where

$$\frac{d\mathrm{Y}(p_\omega(\ell))}{d\ell} = \begin{cases} <0 & \text{if } \sigma > 1 \\ =0 & \text{if } \sigma = 1 \\ >0 & \text{if } \sigma < 1. \end{cases}$$

Using Equation (31), the equilibrium commodity price is

$$p_q^*(\ell) \equiv \frac{1}{1-\tau}\Theta(1, p_\omega^*(\ell)) \quad \text{with} \quad \frac{dp_q^*(\ell)}{d\ell} = \begin{cases} <0 & \text{if } \sigma > 1 \\ =0 & \text{if } \sigma = 1 \\ >0 & \text{if } \sigma < 1. \end{cases}$$

Proof. See the Appendix. □

The effects of the population-resource ratio, ℓ, on expenditure per capita, y, are a direct consequence of the *cost-share effects* discussed earlier. When ℓ rises, the resource becomes relatively more scarce and its price, p_ω, rises. When labor and the resource are substitutes (complements), an increase in the resource price reduces (increases) the resource cost share in primary production and thereby reduces (increases) resource royalties per capita.[16] The important insight of Proposition 1 is thus that the cost-share effects push expenditure per capita in the same direction as resource income per capita. Under substitutability, $\sigma > 1$, we have $\partial y^*(\ell)/\partial\ell < 0$ because the quantity channel at the denominator of p_ω/ℓ dominates as the resource price falls less than one-for-one with ℓ. With $\sigma < 1$, instead, we have $\partial y^*(\ell)/\partial\ell > 0$ because the price channel at the numerator of p_ω/ℓ dominates as the resource price falls more than one-for-one with ℓ. In the Cobb–Douglas case, changes in ℓ leave resource income per capita and expenditure per capita unchanged.

3.3 | The Equilibrium Mortality Rate

Expressions (10) and (12) yield the relationship between mortality and the population-resource ratio. We stress that our definition $\ell = L/\Omega$ implies that comparative-statics statement concerning ℓ qualitatively apply to L as well. Therefore, in the following, one can use "population-resource ratio" and "population" interchangeably. The next proposition provides a full characterization of the response of the equilibrium mortality rate to population and emphasizes the crucial role played by the primary sector's technology.[17]

Proposition 2. *The equilibrium mortality rate is a function of the population-resource ratio, that is, $m = m^*(\ell)$. In the Cobb–Douglas case, $\sigma \to 1$, we have*

$$m = m^*(\ell) \equiv \bar{m} + \tilde{\mu}\cdot\ell^{\chi\{\upsilon(1-\eta)-[\zeta+\xi(1-\upsilon)]\}}, \tag{38}$$

where

$$\tilde{\mu} \equiv \mu\left(\frac{1-\eta}{\eta}\cdot\frac{(1+\beta\rho)(1-\tau)\eta\gamma\frac{\epsilon-1}{\epsilon}}{1-\tau\gamma(1-\eta)\frac{\epsilon-1}{\epsilon}-\eta\gamma\frac{\epsilon-1}{\epsilon}}\right)^{\chi\upsilon(1-\eta)}\Omega^{\chi\upsilon-\chi[\zeta+\xi(1-\upsilon)]}$$

is constant over time. Under substitutability or complementarity, $\sigma \lesseqgtr 1$, we have

$$m = m^*(\ell) \equiv \bar{m} + \bar{\mu}\cdot\mathrm{Y}(\ell)^{\frac{\sigma}{1-\sigma}\chi\upsilon}\cdot\ell^{-\chi[\zeta+\xi(1-\upsilon)]}, \tag{39}$$

where $\bar{\mu} \equiv \mu\eta^{\chi\upsilon\frac{\sigma}{\sigma-1}}\Omega^{\chi\{\upsilon-[\zeta+\xi(1-\upsilon)]\}} > 0$ is constant over time and $\mathrm{Y}(\ell) \equiv \mathrm{Y}(p_\omega^*(\ell))$ is the equilibrium cost share of resource use with

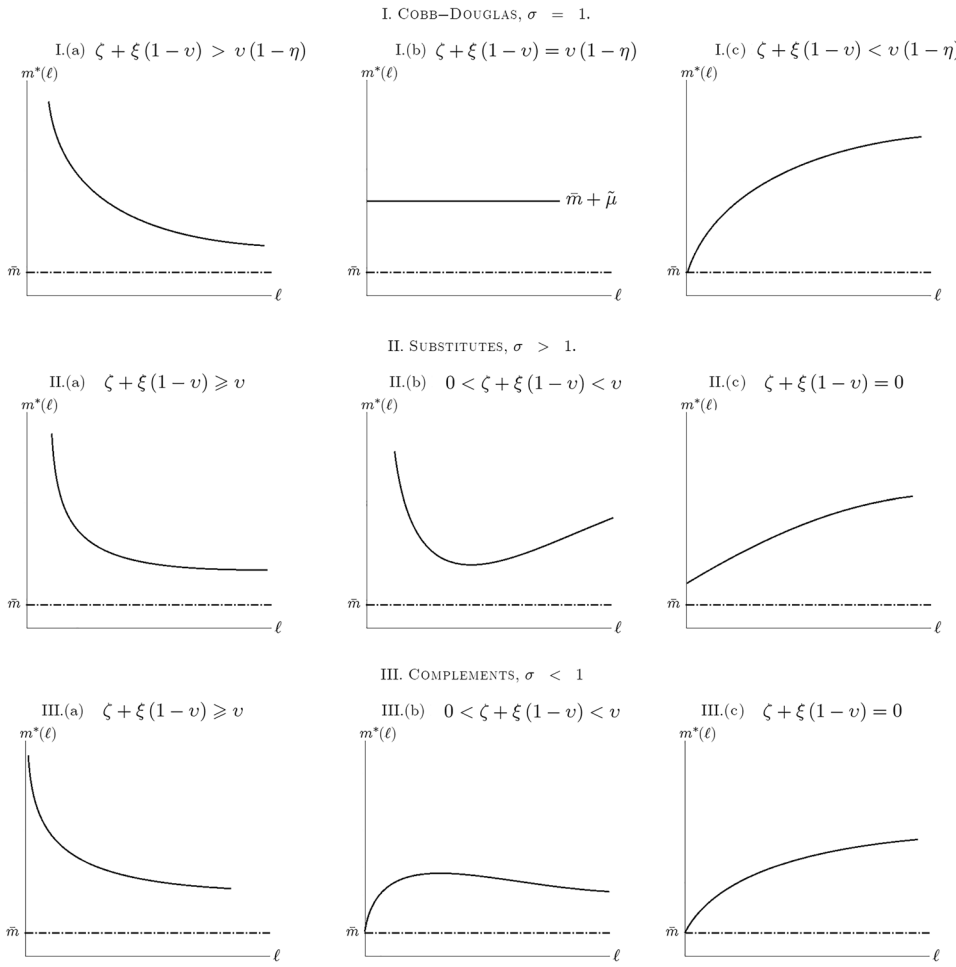

FIGURE 1 | Equilibrium mortality rates as functions of the population-resource ratio, $m = m^*(\ell)$. Without deadly spillovers ($\mu = 0$), the mortality rate would coincide with the baseline case $m = \bar{m}$ in all cases.

the property:

$$\begin{aligned} \sigma > 1 \;\rightarrow\; & \frac{d\Upsilon(\ell)}{d\ell} < 0, \quad \lim_{\ell \to 0^+} \Upsilon(\ell) = 1, \quad \lim_{\ell \to \infty} \Upsilon(\ell) = 0; \\ \sigma < 1 \;\rightarrow\; & \frac{d\Upsilon(\ell)}{d\ell} > 0, \quad \lim_{\ell \to 0^+} \Upsilon(\ell) = 0, \quad \lim_{\ell \to \infty} \Upsilon(\ell) = 1. \end{aligned} \tag{40}$$

Proof. See the Appendix. □

Figure 1 illustrates the equilibrium mortality rates defined in Proposition 2. The mortality response to larger population-resource ratio is ambiguous and often nonmonotonic. In the Cobb–Douglas case, the mortality rate responds to ℓ monotonically, but in different directions depending on the underlying parameters. Under substitutability and complementarity, $m^*(\ell)$ can be nonmonotonic because it depends on the resource cost share, $\Upsilon(\ell) \equiv \Upsilon(p^*_\omega(\ell))$, which affects the strength of the labor-supply channel. We prove in the Appendix all the subcases appearing in Figure 1. In this subsection, we emphasize the intuition behind the results for the Cobb–Douglas and substitutability cases, which are particularly relevant for our results.

Cobb–Douglas. For $\sigma \to 1$, the employment share of the primary sector is an exogenous constant and we obtain $\varepsilon_{Q,L} = 1 - \eta$. Therefore, the response of the mortality rate to ℓ obeys a simple knife-edge condition. When $\upsilon(1-\eta) < \zeta + \xi(1-\upsilon)$, the damage-dilution effect dominates the primary-employment effect and the mortality rate is decreasing in ℓ. When $\upsilon(1-\eta) > \zeta + \xi(1-\upsilon)$, instead, the primary-employment effect dominates: as ℓ grows, the damage-dilution effect does not compensate for higher emissions and the mortality rate increases. The special case $\upsilon(1-\eta) = \zeta + \xi(1-\upsilon)$ yields $m = \bar{m} + \tilde{\mu}$, that is, the mortality rate is invariant to ℓ.

Substitutability. When $\sigma > 1$, the labor-supply effect is weak for small ℓ and strong for large ℓ. In particular (see Appendix),

$$\lim_{\ell \to 0^+} \varepsilon_{Q,L} = 0 \text{ and } \lim_{\ell \to \infty} \varepsilon_{Q,L} = 1. \tag{41}$$

To grasp the intuition for (41), note that a decline in population reduces ℓ because it reduces labor supply. Given $\sigma > 1$, as labor becomes relatively scarce, its relative price rises and the primary sector substitutes labor with the primary resource. As ℓ keeps falling, this process continues until the labor cost share in the primary sector, and thus the elasticity $\varepsilon_{Q,L}$, converges to zero. The same mechanism in reverse explains why the primary-employment effect becomes stronger when the population-resource ratio increases. As a result of these forces, the mortality response to ℓ is generally ambiguous and possibly nonmonotonic. If we rule out damage dilution setting $\zeta + \xi(1-\upsilon) = 0$, the

mortality rate increases with ℓ via the primary-employment effect. Allowing for damage dilution, $\zeta + \xi(1-\upsilon) > 0$, makes the mortality response nonmonotonic in ℓ. Moreover, the mortality rate explodes as ℓ becomes very small.

Lemma 1. *With substitutability, $\sigma > 1$, and damage dilution, $\zeta + \xi(1-\upsilon) > 0$, the mortality rate approaches infinity as the population-resource ratio approaches zero:*

$$\sigma > 1 \rightarrow \lim_{\ell \to 0^+} m^*(\ell) = \lim_{\ell \to 0^+} \bar{m} + \bar{\mu} \cdot \ell^{-\chi[\zeta+\xi(1-\upsilon)]} = +\infty. \quad (42)$$

Proof. See the Appendix. □

The intuition for this result follows from the fact that the elasticity of commodity output with respect to employment, $\varepsilon_{Q,L}$, approaches zero as $\ell \to 0$. When ℓ decreases because population declines, primary producers substitute labor with resource use at increasing rates. This implies that while primary production declines, emissions per worker increase and the resulting excess deaths caused by deadly spillovers eventually explode. In this scenario, a small population is bad for mortality because emissions per capita become very high and the resulting damage cannot be relieved by dose dilution and/or emissions reduction from population density.

Lemma 1 implies that countries with small population and/or abundant primary resources may exhibit very high mortality rates. What happens for large ℓ, instead, depends on parameter values. Since $\varepsilon_{Q,L}$ approaches one as $\ell \to \infty$, we have the cases in Figure 1. If $\zeta + \xi(1-\upsilon) \geqslant \upsilon$, the mortality rate is *L-shaped*, that is, $m^*(\ell)$ is monotonically decreasing in ℓ because the labor-supply effect is weaker than the damage-dilution effect for all ℓ. If $0 < \zeta + \xi(1-\upsilon) < \upsilon$, the mortality rate is *U-shaped*, that is, $m^*(\ell)$ reaches a minimum and then increases with ℓ, because a large ℓ combined with a high elasticity $\varepsilon_{Q,L}$ makes the primary-employment effect strong enough to dominate the damage-dilution effect.[18]

4 | Population Dynamics

This section characterizes the equilibrium dynamics of fertility, mortality, and population in a self-contained subsystem describing the demography block of our economy. The property that makes our model this tractable is the scale invariance of the Schumpeterian model of endogenous innovation that provides the industry block of our economy.

4.1 | Demography–Scarcity Interactions

Since the resource endowment Ω is fixed, the population-resource ratio, $\ell = L/\Omega$, grows at the same rate as population, that is,

$$\frac{\dot{\ell}(t)}{\ell(t)} = b(t) - m^*(\ell(t)), \quad (43)$$

where $m^*(\ell)$ is the equilibrium mortality rate characterized in Proposition 2. The Euler equation for the birth rate (17) yields

$$\frac{\dot{b}(t)}{b(t)} = \frac{b(t)}{(1-\alpha)(1-\psi)}\left[\frac{1-(1-\psi)\cdot y^*(\ell(t))}{y^*(\ell(t))}\right] - \rho, \quad (44)$$

where $y^*(\ell)$ is the equilibrium expenditure per capita characterized in Proposition 1. Equations (43) and (44) form a 2D dynamic system that fully determines the equilibrium interactions between fertility, resource scarcity, and mortality. Since the system can generate multiple steady states, we distinguish between stable and unstable cases with the following definition.

Definition 1. A *regular steady state is a* point (ℓ_{ss}, b_{ss}) in (ℓ, b) space such that the values (ℓ_{ss}, b_{ss}) are positive and finite and satisfy $\dot{b} = \dot{\ell} = 0$. Moreover, the point exhibits (at least local) stability, that is, there is a thick set of initial conditions $\ell(0) > 0$ starting from which the equilibrium trajectory $(\ell(t), b(t))$ converges to (ℓ_{ss}, b_{ss}) and population converges to the finite value $L_{ss} = \ell_{ss}\Omega > 0$.

Our notion of regular steady state is conventional in the sense that, being a stable rest point, it represents the long-run attractor of the dynamics when certain initial conditions hold. The distinctive property is that (ℓ_{ss}, b_{ss}) features constant population size in the long run, L_{ss}, while per capita income grows via innovation. In this light, it is worth noting that our approach makes two distinct contributions to the existing analytical framework. First, if a regular steady state exists independently of pollution, deadly spillovers modify its position and the path that leads to it. While the qualitative properties of the dynamics in the two models are similar, their *quantitative* properties are obviously different and potentially very much so. Second, deadly spillovers can create steady states that would not otherwise exist, and such steady states may be regular or not. In other words, deadly spillovers change the *qualitative* properties of the dynamics rather drastically. To highlight this feature, we first summarize the predictions of the model with no deadly spillovers (Subsection 4.2) and then analyze the model with deadly spillovers (Subsection 4.3).

4.2 | Special Case With Exogenous Mortality

We set $\mu = 0$ in (10) to obtain the special case with exogenous mortality nested in our model. The steady-state loci are, respectively, $\dot{\ell} = 0 \rightarrow b = \bar{m}$ and

$$\dot{b} = 0 \rightarrow b = \frac{(1-\alpha)(1-\psi)\rho}{y^*(\ell)^{-1} - (1-\psi)}. \quad (45)$$

This special case delivers the following results (see the Appendix for details and proofs).

First, combining the two steady-state equations yields

$$\bar{m} = \frac{(1-\alpha)(1-\psi)\rho}{y^{-1} - (1-\psi)} \rightarrow y_{ss} = \frac{(1-\psi)\bar{m}}{(1-\alpha)\rho - \bar{m}}.$$

This result says that steady-state expenditure depends only on preference parameters and demography via the exogenous mortality rate. It thus has a strong Malthusian flavor. It differs from the standard Malthusian result, however, because y_{ss} is

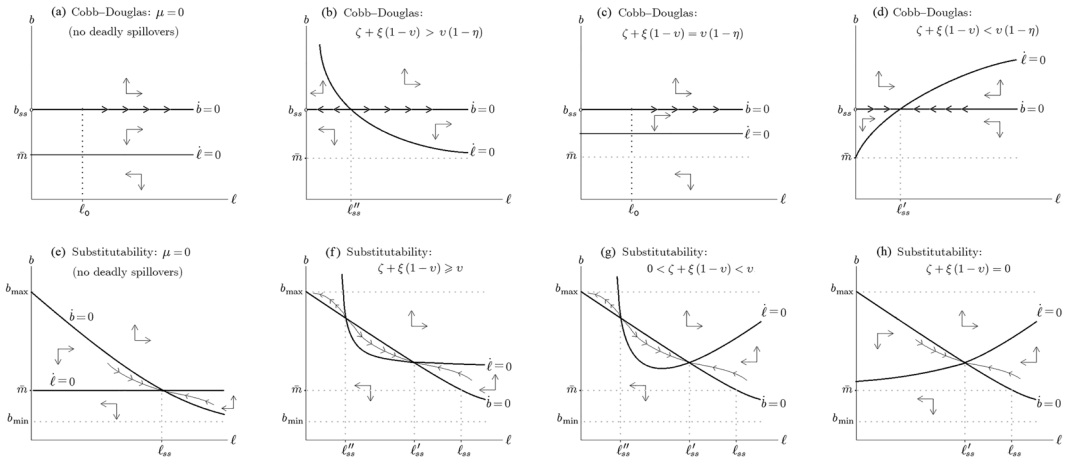

FIGURE 2 | Phase diagrams of system (43)–(44) under exogenous and endogenous mortality with $\sigma \geqslant 1$. Diagrams (a) and (e) exclude deadly spillovers ($\mu = 0$). Diagrams (b)–(d) and (f)–(g) assume deadly spillovers under different combinations of parameters. See the Appendix for the case of complementarity.

consumption expenditure per capita, not real consumption per capita, and therefore it is not a measure of living standards. As stated, in our model constant expenditure per capita is associated to constant growth of consumption per capita via innovations that reduce the price of consumption.[19]

Second, when the primary sector's technology is Cobb–Douglas, $\sigma = 1$, population grows or declines at a constant rate because the stationary loci are horizontal straight lines that in general do not coincide. In Figure 2, phase diagram (a) shows the case in which the equilibrium birth rate exceeds $\bar{m}$, implying a constant and positive population growth rate.

Third, under substitutability, $\sigma > 1$, there exists a regular steady state (ℓ_{ss}, b_{ss}) that is saddle-point stable (see phase diagram (e) in Figure 2). If the economy starts with $\ell(0) < \ell_{ss}$, the equilibrium path features *positive population growth* with a *declining fertility rate* until b reaches $\bar{m}$ and stabilizes the population. The reason for these dynamics is that, with $\sigma > 1$, expenditure per capita declines with ℓ because the rising resource scarcity yields lower resource income per capita. This mechanism produces the negative slope of the $\dot{b} = 0$ locus, which is the key to the stability of the process. In fact, in the opposite case of complementarity, the income response to ℓ is reversed and the steady state (ℓ_{ss}, b_{ss}) becomes unstable: with $\sigma < 1$, the economy follows diverging paths, leading to either population explosion or human extinction depending on the initial level of the population-resource ratio (see the Appendix for details).

The main takeaway of this analysis is that $\sigma \geqslant 1$ deserves special emphasis. The Cobb–Douglas case is interesting because the prediction of exponential population growth rests on a knife-edge hypothesis about technology: only for $\sigma = 1$ no steady state exists unless the two stationary loci are on top of each other. Substitutability, $\sigma > 1$, is even more relevant because it generates a plausible path of demographic development: assuming $\ell(0) < \ell_{ss}$, population converges to a finite size because resources per worker and births per adult shrink over time. This is consistent with the well-known fertility decline observed throughout the industrialized world and with the widely shared idea that population growth cannot outstrip the finite natural resource base. Introducing deadly spillovers in this context identifies how pollution changes at the margin the steady state and thus the equilibrium path of the economy. We thus focus on $\sigma \geqslant 1$ in the remainder of the analysis.[20]

4.3 | Dynamics With Endogenous Mortality

The analysis of the previous subsection allows us to study the dynamical system with endogenous mortality (43)–(44) in a straightforward manner. The $\dot{b} = 0$ locus is still given by expression (45). Equation (43), instead, yields

$$\dot{\ell} = 0 \quad \rightarrow \quad b = m^*(\ell) \equiv \begin{cases} \bar{m} + \tilde{\mu} \cdot \ell^{\chi\{\upsilon(1-\eta)-[\zeta+\xi(1-\upsilon)]\}} & \text{if } \sigma = 1, \\ \bar{m} + \tilde{\mu} \cdot \Upsilon(\ell)^{\frac{\sigma}{1-\sigma}\chi\upsilon} \cdot \ell^{-\chi[\zeta+\xi(1-\upsilon)]} & \text{if } \sigma \gtrless 1. \end{cases} \tag{46}$$

This expression shows that the shape of the $\dot{\ell} = 0$ locus matches the shape of the equilibrium mortality rate defined in Proposition 2. A property of note is that we no longer obtain a simple analytical solution for expenditure per capita because the mortality rate is endogenous. Combining the $\dot{b} = 0$ and $\dot{\ell} = 0$ equations yields that ℓ is the solution of an implicit equation, that is,

$$\ell_{ss} = \arg\text{solve}\left\{ m^*(\ell) = \frac{(1-\alpha)(1-\psi)\rho}{y^*(\ell)^{-1} - (1-\psi)} \right\}. \tag{47}$$

We then obtain

$$y_{ss} = y^*(\ell_{ss}) = \frac{(1-\psi)m^*(\ell_{ss})}{(1-\alpha)\rho - m^*(\ell_{ss})}. \tag{48}$$

Figure 2 shows the resulting phase diagrams for the Cobb–Douglas case and for substitutability. Both cases deliver novel results.

Cobb–Douglas. With $\sigma = 1$, the gross fertility rate determined by (45) is constant but deadly spillovers generally affect population growth via the mortality rate: whenever the primary-

employment effect does not match exactly the damage-dilution effect, $\upsilon(1-\eta) \neq \zeta + \xi(1-\upsilon)$, deadly spillovers create a steady state that would not exist otherwise. The steady state (ℓ_{ss}, b_{ss}) can be stable or unstable depending on the relative strength of the primary-employment and damage-dilution effects.

Proposition 3. (Cobb–Douglas). For $\sigma = 1$ and $\upsilon(1-\eta) \neq \zeta + \xi(1-\upsilon)$, deadly spillovers create a steady state (ℓ_{ss}, b_{ss}), which may be stable or unstable: it is a regular steady state for $\upsilon(1-\eta) > \zeta + \xi(1-\upsilon)$; it creates a mortality trap for $\upsilon(1-\eta) < \zeta + \xi(1-\upsilon)$. For $\sigma = 1$ and $\upsilon(1-\eta) = \zeta + \xi(1-\upsilon)$, there is no steady state and deadly spillovers permanently reduce the constant population growth rate.

Proof. See the Appendix. □

The top panel of Figure 2 shows the phase diagrams for the Cobb–Douglas commodity production technology. It is worth stressing that, except for specific knife-edge cases, the main message of the Cobb–Douglas technology is that pollution-caused mortality creates steady states that would not exist otherwise and thus delivers novel qualitative results. In diagram (b), $\upsilon(1-\eta) < \zeta + \xi(1-\upsilon)$ yields the cases where the damage-dilution effect dominates the primary-employment effect. In this configuration, $m^*(\ell)$ is decreasing in ℓ, the $\dot{\ell} = 0$ locus is decreasing in ℓ, and the steady state (ℓ''_{ss}, b_{ss}) is unstable. The population-resource ratio ℓ''_{ss} is thus an *extinction threshold*: if labor is initially abundant relative to the resource, $\ell_0 > \ell''_{ss}$, the economy experiences sustained population growth whereas in the opposite situation, $\ell_0 < \ell''_{ss}$, the economy is in a mortality trap characterized by a vicious circle of ever-declining population and ever-increasing mortality. In this scenario, population must be initially large enough, relative to the resource endowment, to generate positive population growth at time zero and thereafter. In diagram (d), $\upsilon(1-\eta) > \zeta + \xi(1-\upsilon)$ yields the case in which the primary-employment effect dominates the damage-dilution effect. In this configuration, $m^*(\ell)$ is decreasing in ℓ and deadly spillovers create a stable steady state (ℓ'_{ss}, b_{ss}). Starting from $\ell_0 > \ell'_{ss}$, population increases at a declining rate due to pollution-induced mortality until population growth becomes zero. We thus have the insight that introducing pollution-caused mortality in a model that would otherwise feature exploding population is sufficient to produce a finite population. In other words, deadly spillovers are the only force that stabilizes the population in the long run. Diagram (c) considers the knife-edge case $\upsilon(1-\eta) = \zeta + \xi(1-\upsilon)$ that does not feature steady states and may predict opposite dynamics depending on the strength of pollution-caused mortality. In this scenario, Equation (45) yields $b_{ss} > \bar{m}$ and the equilibrium mortality rate determined by (46) is $m^* = \bar{m} + \tilde{\mu}$. If $\tilde{\mu}$ is relatively small, that is, if pollution induces moderate excess mortality, we obtain positive constant population growth, $b_{ss} - \bar{m} - \tilde{\mu} > 0$, like in diagram (c). If, instead, $\tilde{\mu}$ is sufficiently large, we obtain $b_{ss} - \bar{m} - \tilde{\mu} < 0$ and deadly spillovers reverse the sign of the constant population growth rate from positive to negative.[21]

The bottom panel of Figure 2 shows the phase diagrams under substitutability, $\sigma > 1$, which delivers further interesting results. First, even if damage dilution is positive, deadly spillovers reduce the steady-state size of the population: while the model without pollution exhibits a regular steady state (ℓ_{ss}, b_{ss}), the model with deadly spillovers generates a regular steady state (ℓ'_{ss}, b'_{ss}) with $\ell'_{ss} < \ell_{ss}$. Second, recalling Lemma 1, substitutability makes the mortality rate explode for small ℓ when damage dilution is positive, $\zeta + \xi(1-\upsilon) > 0$. Phase diagrams (f) and (g) in Figure 2 illustrate this mechanism: deadly spillovers shift the $\dot{\ell} = 0$ locus up and bend it upward as ℓ approaches zero. If deadly spillovers are extremely strong, the regular steady state disappears.[22] More generally, when the regular steady state exists, the mortality effect of pollution at low population-resource ratio creates an additional, unstable steady state that yields a mortality trap.

Proposition 4. *(Substitutability). Assume $\sigma > 1$. With deadly spillovers, the regular steady state (ℓ'_{ss}, b'_{ss}) has smaller population-resource ratio, $\ell'_{ss} < \ell_{ss}$, than the regular steady state (ℓ_{ss}, b_{ss}) of the model without pollution. In addition, if $\zeta + \xi(1-\upsilon) > 0$, deadly spillovers create a second, unstable steady state (ℓ''_{ss}, b''_{ss}) with $b''_{ss} > b'_{ss}$ and $\ell''_{ss} < \ell'_{ss}$. The interval $(0, \ell''_{ss})$ is the mortality trap caused by deadly spillovers. If $\ell(0) > \ell''_{ss}$, the economy converges to the regular steady state. If $\ell(0) < \ell''_{ss}$, the equilibrium path exhibits $\lim_{t\to\infty} \ell(t) = 0$.*

Proof. See the Appendix. □

Figure 2 illustrates the two main results delivered by Proposition 4. First, deadly spillovers *reduce the population* by modifying the position of the regular steady state: as the economy converges to (ℓ'_{ss}, b'_{ss}), the long-run population-resource ratio is lower because of higher mortality. This conclusion, which holds regardless of the damage-dilution effect, is self-evident in Figure 2: with respect to the case with no pollution, diagram (e), deadly spillovers reduce ℓ'_{ss} in all cases, even when no mortality trap arises like in diagram (h). More generally, endogenous mortality due to pollution affects the whole equilibrium path of the economy and, as we shall see, has substantial consequences for welfare through multiple channels, including firms' incentives to innovate since these depend on the anticipated dynamics of the size of the market.

The second result is that deadly spillovers can create the mortality trap, the region $(0, \ell''_{ss})$ of the state space where implosive population dynamics prevail. The unstable steady state (ℓ''_{ss}, b''_{ss}) is an extinction threshold: if population is initially too small relative to the resource endowment, $\ell(0) < \ell''_{ss}$, the economy does not converge to the regular steady state (ℓ'_{ss}, b'_{ss}) and follows, instead, an equilibrium path leading to zero population. Such population implosion *does not* result from falling fertility. Rather, starting from $\ell(0) < \ell''_{ss}$, the transition exhibits increasing fertility as well as increasing mortality. The reason is that the fertility rate is constrained by household income, whereas the mortality rate is unbounded: as population shrinks, growing deadly spillovers lead to exploding mortality while households may only raise the fertility rate up to $b_{\max}$, the highest birth rate consistent with their budget constraint. The economy escapes the mortality trap and converges to the regular steady state only if the initial population-resource ratio is sufficiently high, $\ell(0) > \ell''_{ss}$. This result delivers specific insights for less populated, resource-rich economies. Diagrams (f)–(g) in Figure 2 show that economies that are closer to the mortality trap feature a *low* population-resource ratio and a *high* birth rate. Given resource abundance,

economies with a small population tend to be ceteris paribus closer to the mortality trap even though they may exhibit higher birth rates. By the same token, exogenous shocks that reduce population push the economy toward the trap. A similar though not identical mechanism applies to resource abundance and exogenous shocks expanding the endowment (e.g., discoveries of new stocks of natural resources): given population, a larger resource base can push the economy toward the trap not only by reducing the current population-resource ratio, but also by expanding the mortality trap itself by pushing ℓ''_{ss} to the right. We discuss these and related points in the next section.

5 | Growth, Emission Taxes, and Resource Booms

In this section, we derive the equilibrium paths of consumption, innovation rates, income growth, and utility. We then study the effects of emission taxes, subsidies to the primary sector, resource booms, and discuss the framework's implications for empirical analysis and policymaking.

5.1 | Consumption, Growth, and Utility

The model's measure of gross domestic product is final output, C. Since household expenditure on consumption is Y, we have (see the Appendix)

$$\frac{C(t)}{L(t)} = \frac{y(t)}{p_c(t)} = y(t) \cdot \frac{z(t)^{\theta} N(t)^{\frac{1}{\epsilon-1}}}{(1-\gamma)^{-(1-\gamma)} \gamma^{-\gamma} \frac{\epsilon}{\epsilon-1} p_q(t)^{\gamma}}. \tag{49}$$

This expression says that GDP per capita equals consumption expenditure per capita divided by the price index of intermediate goods. The price index, in turn, depends on the endogenous components of technology, product variety, and firm-specific knowledge, and on the relative price of the commodity. For clarity, we separate the role of endogenous technology from that of the vertical production structure. In the last term of (49), the numerator is a reduced-form representation of *total factor productivity* (TFP), which we henceforth denote as $T \equiv z^{\theta} N^{\frac{1}{\epsilon-1}}$. The denominator is an index of how markup-pricing and the cost of inputs drive the price of intermediates.

Differentiating (49) with respect to time, we obtain

$$g(t) \equiv \frac{\dot{C}(t)}{C(t)} - \frac{\dot{L}}{L} = \frac{\dot{T}(t)}{T(t)} + \frac{\dot{y}(t)}{y(t)} - \gamma \frac{\dot{p}_q(t)}{p_q(t)}. \tag{50}$$

The first term is the growth rate of TFP, which in turn equals a weighted sum of the rates of vertical innovation, $\dot{z}/z$, and horizontal innovation, $\dot{N}/N$. The second term is expenditure per capita growth. The third term is the standard *scarcity drag* of models with finite natural resources. Recalling Proposition 1, the equilibrium commodity price is $p_q^*(\ell) = \frac{1}{1-\tau}\Theta(1, p_\omega^*(\ell))$ and its growth rate over time thus reads

$$\frac{\dot{p}_q(t)}{p_q(t)} = \frac{d \ln \Theta(w, p_\omega^*(\ell(t)))}{d \ln p_\omega^*(\ell(t))} \frac{\dot{\ell}(t)}{\ell(t)} = \Upsilon(t) \frac{\dot{\ell}(t)}{\ell(t)}, \tag{51}$$

where Υ is the resource cost share defined in (32). Therefore, using (51) and the results in Proposition 1, we can write the growth rate of income per capita as

$$g(t) = \frac{\dot{T}(t)}{T(t)} + \gamma \left[(1-\tau) \frac{\epsilon-1}{\epsilon} \ell y^*(\ell) \underbrace{\frac{d\Upsilon(p_\omega(\ell))}{d\ell}}_{-\text{ for } \sigma>1} - \Upsilon(t) \right] \frac{\dot{\ell}(t)}{\ell(t)}. \tag{52}$$

The right-hand side decomposes the growth rate g into a first component representing TFP growth, $\dot{T}/T$, and a second term representing the transitional effects that operate only when ℓ changes over time. Under substitutability, the term in square brackets is surely negative due to the scarcity drag—that is, resource income per capita and expenditure per capita react negatively to increasing resource prices. Holding TFP growth constant, the scarcity drag makes transitional growth slower. Therefore, along an equilibrium path with positive net fertility, $\dot{\ell} > 0$, the growth rate of income per capita is smaller than that of TFP.

Expression (52) shows that growth in income per capita is positive only if TFP growth more than compensates for scarcity-drag effects. Importantly, when the condition for $g > 0$ is satisfied, the conventional equilibrium path with growing population—that is, the path converging to the regular steady state "from the left"—exhibits growing income per capita, $g > 0$, and a falling birth rate over time, $\dot{b} < 0$, during the whole transition.[23] This negative comovement between income per capita and the fertility rate is consistent with the negative income–fertility relationship that characterized the development of many advanced economies in the past century (see, e.g., Doepke 2004).

When the population-resource ratio becomes constant, $\dot{\ell} = 0$, the only source of economic growth is innovation. More precisely, if the economy converges to a regular steady state (ℓ_{ss}, b_{ss}), the only source of economic growth is *vertical* innovation: firm-specific knowledge grows at a constant rate while the mass of firms is constant, $N(t) = N_{ss}$. The mechanism driving this property is that vertical and horizontal innovation exhibit a negative comovement during the transition: entry of new firms reduces the profitability of firm-specific knowledge investment through market fragmentation while investment in firm-specific knowledge slows down entry by diverting labor away from horizontal R&D. As we show in the Appendix, these comovements eventually bring the economy to a steady state where the mass of firms is constant and the engine of growth is firm-specific knowledge accumulation.

Proposition 5. Assume

$$\frac{\frac{\epsilon-1}{\epsilon} \kappa \theta \left(\phi - \frac{\rho+\delta}{\kappa} \right) y_{ss}}{\frac{1-\theta(\epsilon-1)}{\epsilon} y_{ss} - \beta(\rho+\delta)} > \rho + \delta$$

and let the economy converge to the steady state (ℓ_{ss}, b_{ss}). Then, the mass of firms is

$$N_{ss} = \frac{\frac{1-\theta(\epsilon-1)}{\epsilon} y_{ss} - \beta(\rho+\delta)}{\phi - \frac{\rho+\delta}{\kappa}} \cdot L_{ss} > 0, \tag{53}$$

firm-specific knowledge grows at rate

$$\left(\frac{\dot{z}}{z}\right)_{ss} = \frac{\frac{\epsilon-1}{\epsilon}\kappa\theta\left(\phi - \frac{\rho+\delta}{\kappa}\right)y_{ss}}{\frac{1-\theta(\epsilon-1)}{\epsilon}y_{ss} - \beta(\rho+\delta)} - \rho - \delta > 0, \tag{54}$$

and final output grows at rate

$$g_{ss} = \theta\left(\frac{\dot{z}}{z}\right)_{ss}.$$

Proof. *See the Appendix.* □

This proposition highlights an important property of our model. While the model belongs to a class known for the scale invariance of the steady-state growth rate, deadly spillovers create a novel channel through which the deep parameters regulating pollution-induced mortality have steady-state growth effects. To see this, note that Equation (54) contains steady-state expenditure per capita, y_{ss}, which according to Equation (48) is a function of the steady-state resource-population ratio, ℓ_{ss}. The economic intuition and the direction of the key relationships is the following. First, a higher steady-state mortality rate $m^*(\ell_{ss})$ implies a higher steady-state expenditure per capita $y^*(\ell_{ss})$ via the pesudo-Malthusian relationship (48)—an effect that we label as the *mortality-expenditure channel*. Second, a higher steady-state expenditure per capita $y^*(\ell_{ss})$ reduces steady-state growth g_{ss} because higher expenditure per capita expands the size of the market and this attracts entry: each firm captures a smaller market share and thus reduces in-house R&D efforts[24]—an effect that we label as the *expenditure-innovation channel*. These two mechanisms imply that the steady-state growth rate is a function of the steady-state population-resource ratio ℓ_{ss} as a result of endogenous mortality. Importantly, this is not a scale effect linking population size to economic growth because population L_{ss} is endogenous, and the relationship between L_{ss} and $m^*(\ell_{ss})$ has a generally ambiguous sign. In fact, the relation that we obtain is between growth and the model's deep parameters characterizing the generation and propagation of pollution through the population, with the resulting effect on mortality, and not a relation between growth and the size of a particular endowment. The sign of steady-state growth effects induced by an exogenous shock ultimately depends on the origin of the shock, which may affect the shape of the mortality function $m^*(\cdot)$ and the steady-state input ratio ℓ_{ss} at the same time—a case in point is the analysis of tax changes in the next subsection.

The model's key measure of living standards is individual utility in Equation (14), which evaluated at the equilibrium reads (see the Appendix)

$$\ln u = \underbrace{\bar{\alpha} + \ln T + \ln y^*(\ell) - \gamma \ln p_q^*(\ell)}_{\text{Economic channel}} + \underbrace{\ln L^{\psi} b^{-(1-\psi)(1-\alpha)}}_{\text{Demographic channel}}, \tag{55}$$

where $\bar{\alpha} \equiv \ln \alpha^{\alpha}\gamma^{\gamma}(1-\alpha)^{1-\alpha}(1-\gamma)^{1-\gamma}\frac{\epsilon-1}{\epsilon}$. Equation (55) allows us to distinguish the different components of instantaneous utility. The economic channel shows how the components of economic activity affect utility at each point in time. The demographic channel summarizes the effects of population level and birth rate on utility: it combines direct effects, that is, the household's preference for adults and children, and the indirect effects of family composition on the allocation of consumption among adults and children. Differentiating (55) with respect to time yields

$$\frac{\dot{u}(t)}{u(t)} = g(t) + (\psi+1)\frac{\dot{L}(t)}{L(t)} - (1-\psi)(1-\alpha)\frac{\dot{b}(t)}{b(t)}, \tag{56}$$

where g is the growth rate computed in (52). Equation (56) shows the distinct contribution of economic and demographic channels to the dynamics of utility. The model's dynamics, worked out in detail in the Appendix and briefly discussed above, show that in response to a permanent expansion of the market for intermediate goods both firm-specific knowledge growth and net entry accelerate until they revert to $(\dot{z}/z)_{ss}$ and N_{ss}. Changes in fundamentals therefore modify the dynamics of (ℓ, b) and affect welfare through the underlying components of utility, namely, the consumption expenditure channel, $\ln y^*(\ell)$, the commodity price channel, $-\gamma \ln\left(p_q^*(\ell)\right)$, and the demographic channel, $\ln L^{\psi} b^{-(1-\psi)(1-\alpha)}$. We next provide concrete examples by studying the effects of the commodity tax and of a resource boom.

5.2 | Commodity Tax

We consider the scenario in which substitutability and deadly spillovers create a regular steady state and a mortality trap (Proposition 4). The following proposition provides the comparative-statics effects of τ on both the regular steady state and the size of the mortality trap.

Proposition 6. (Commodity Tax). Assume $\sigma > 1$ and $\zeta + \xi(1-\upsilon) > 0$. The increase in the commodity tax, τ, shifts the $\dot{\ell} = 0$ locus down and the $\dot{b} = 0$ locus up. Therefore, it yields a higher regular-steady-state population-resource ratio, $d\ell'_{ss}/d\tau > 0$, as well as a smaller mortality-trap threshold, $d\ell''_{ss}/d\tau < 0$.

Proof. See the Appendix. □

To understand the mechanism driving these comparative-statics effects, start holding the resource-population ratio constant at the initial steady state. The increase in τ reduces the demand for the resource, triggering a reduction in the resource price and an increase in expenditure per capita. Given substitutability, the lower resource price raises the resource cost share and thus drives down the mortality rate via the primary-employment effect. In graphical terms, there is an upward shift of the expenditure schedule $y^*(\ell)$ that yields an upward shift of the $\dot{b} = 0$ locus and a downward shift of mortality schedule, $m^*(\ell)$, that yields a downward shift of the $\dot{\ell} = 0$ locus (see Figure 3a–c). The consequence of these shifts is a widening gap between the two steady states, with a higher regular-steady-state population-resource ratio, ℓ'_{ss}, and a lower mortality-trap threshold, ℓ''_{ss}.

As an example of the forces at play, assume that the tax change is relatively small so that the initial steady state remains in the basin of attraction of the regular steady state. The phase diagram shows that the population-resource ratio, ℓ, increases monotonically over time as the economy converges to the new regular steady state. The commodity tax increase, therefore, triggers a permanent, monotonic expansion of the population. The birth rate, b,

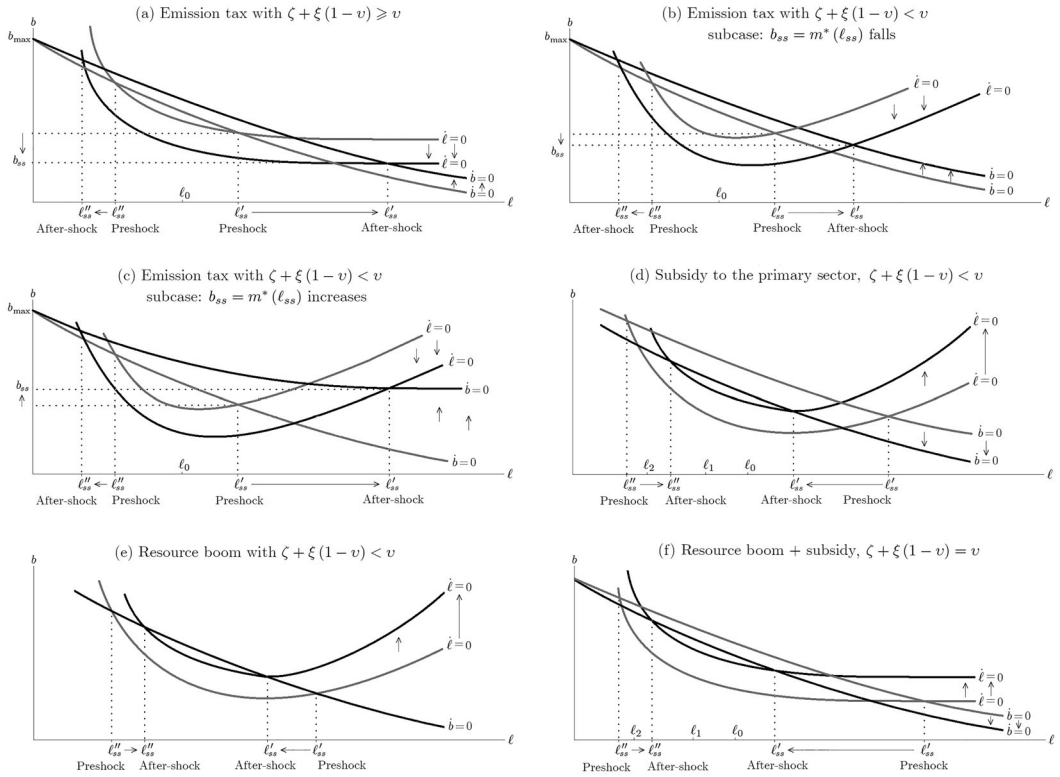

FIGURE 3 | Exogenous shocks under substitutability, $\sigma > 1$, and $\zeta + \xi(1-\upsilon) > 0$. See the main text for detailed descriptions.

in contrast, exhibits *overshooting*: it jumps up on the new saddle path and then declines gradually and monotonically during the transition, converging from above to the new steady state. The position of the new steady state on the vertical axis, however, can be either above or below the old one. Before discussing this property, we note that the permanent expansion of the population causes a permanent expansion of the market for intermediate goods. This means that initially TFP growth accelerates because of both more net entry and more investment by incumbent firms. Along the transition, the expansion of the mass of firms weakens the incentive of incumbents to invest, causing a slowdown of firm-level productivity growth that counteracts the expansion of product variety. This negative comovement between mass of firms and firm growth is at the heart of the Schumpeterian model that we use. As discussed, absent deadly spillovers, this mechanism would produce scale invariance, which in this context would yield that steady-state TFP growth is invariant to the commodity tax. However, with deadly spillovers, steady-state TFP growth responds to the steady-state population-resource ratio. This means that the commodity tax has an effect on steady-state TFP growth that has the opposite sign of the effect on the mortality rate. We stress once again that this channel for growth effects is solely and entirely due to the endogeneity of the mortality rate.

As mentioned, the birth rate overshoots: it jumps up on the new saddle path and then declines along the transition toward a new steady-state value that can be larger or smaller than the old one because in steady state the birth rate must equal the mortality rate. We have two cases:

- If $\zeta + \xi(1-\upsilon) \geqslant \upsilon$, damage dilution is sufficiently strong to guarantee that the mortality rate does not increase despite the larger ℓ and higher aggregate emissions at the new steady state.[25] This implies the new steady-state birth rate is not higher than the old one. This case is in Figure 3a. Note that because the new steady state has a lower mortality rate, it has a higher growth rate.

- If $\zeta + \xi(1-\upsilon) < \upsilon$, damage dilution is weaker and thus in the new steady state the mortality rate may be higher or lower than the initial one because it is subject to opposing forces: the downward shift of the schedule, $m^*(\ell)$, that reduces mortality for given ℓ; the larger ℓ that can result in higher aggregate emissions that dominate damage dilution.[26] If the net effect is a lower or unchanged mortality rate, the conclusions are the same as for the previous case (see Figure 3b). If, instead, the net effect is a higher mortality rate, like in Figure 3c, the new steady state has a lower growth rate.

One takeaway of this analysis is that the commodity tax yields a double *demographic* dividend: it expands the size of the population *and* it reduces the size of the mortality trap by pushing the mortality threshold ℓ''_{ss} to the left. Associated with these gains there is an *economic growth* dividend because the lower mortality rate yields a higher TFP growth rate. The same mechanism in reverse, that is, reducing the commodity tax, yields a double loss, namely, a lower population and a larger mortality trap. A large enough cut of the commodity tax can actually put the economy in the mortality trap, as shown in Figure 3d. If the initial population-

resource ratio is ℓ_2, the economy converges to the regular steady state under the old tax rate but falls in the mortality trap with the new tax rate, following a path that eventually leads to extinction. This scenario offers a sobering lesson for less populated resource-rich countries that implement low commodity and/or emission taxes and/or subsidize their primary sectors. In an economy with population-resource ratio close to the mortality trap, subsidizing the primary sector is functionally equivalent to introducing a negative emission tax. Empirical evidence suggests that many real-world economies face such a situation, in particular oil-exporting countries where subsidies to the extractive industry are pervasive and high (Gupta et al. 2002; Metschies 2005). Below, we pursue this argument further by showing that the combination of subsidies to the primary sector and new discoveries of the resource can be a recipe for disaster.

5.3 | Resource Booms

A *resource boom* is an exogenous increase at time $t = 0$ of the resource endowment, Ω. By definition, therefore, it reduces the resource-population ratio, $\ell(0) = L(0)/\Omega$. All else equal, this immediate effect brings the economy closer to the mortality trap. But the shock may further increase the threat of population implosion by expanding the mortality trap depending on the value of the damage elasticity. Note, moreover, that, as in the case of the commodity tax, the resource boom has an effect on steady-state TFP growth of the opposite sign of its effect on the mortality rate. The following proposition summarizes the demographic effects of the boom.

Proposition 7. *(Resource boom). Assume $\sigma > 1$ and $\zeta + \xi(1-\upsilon) > 0$. An increase in the resource endowment, Ω, affects the equilibrium mortality function $m^*(\ell)$ as follows:*

$$\frac{dm^*(\ell)}{d\Omega}\begin{cases} >0 & \text{if } \upsilon > \zeta + \xi(1-\upsilon) \\ =0 & \text{if } \upsilon = \zeta + \xi(1-\upsilon) \\ <0 & \text{if } \upsilon < \zeta + \xi(1-\upsilon) \end{cases} \text{for any } \ell > 0. \tag{57}$$

When $\upsilon > \zeta + \xi(1-\upsilon)$, a resource boom enlarges the mortality trap, $(0, \ell''_{ss})$.

Proof. See the Appendix. □

The mechanism driving this result is that the emission damage incorporated in the mortality function (39) depends on the resource endowment Ω with elasticity $\upsilon - [\zeta + \xi(1-\upsilon)]$. If this elasticity is positive, the increase in Ω raises the mortality rate associated with the regular steady state. This phenomenon is a type of *resource curse* seldom recognized in the literature. Figure 3e describes the effect of the resource boom assuming $\upsilon > \zeta + \xi(1-\upsilon) > 0$. As the endowment increases from Ω_0 to Ω_1, the $\dot{\ell} = 0$ locus shifts up and yields a lower population-resource ratio in the regular steady state, ℓ'_{ss}, and a higher mortality-trap threshold, ℓ''_{ss}. At the same time, the population-resource ratio at time zero moves from the preshock level $\ell_0 = L_0/\Omega_0$ to the lower after-shock level $\ell_1 \equiv \ell_1(0) = L_0/\Omega_1$. The welfare effects of these shocks are generally ambiguous. Moreover, the shock itself may drive the economy into the mortality trap, yielding drastically opposite results: if $\ell_1 < \ell''_{ss}$, the population decline deletes and eventually overturns the consumption gains, while both the demographic components of utility—adult population and flow of children—yield net losses both in the transition and in the long run as the mortality rate grows.

In the case $\upsilon \leqslant \zeta + \xi(1-\upsilon)$, the resource boom does not expand the mortality trap but this does not mean that the trap is less threatening: even when the mortality trap, $(0, \ell''_{ss})$, shrinks or remains the same, the increase in Ω reduces the population-resource ratio. With $\upsilon < \zeta + \xi(1-\upsilon)$, the $\dot{\ell} = 0$ locus shifts down but the initial resource-population ratio can fall more than the mortality threshold ℓ''_{ss}. Moreover, with $\upsilon = \zeta + \xi(1-\upsilon)$, the resource boom surely moves the economy closer to population implosion because ℓ falls instantaneously while the steady-state levels of ℓ and b do not change.

5.4 | Resource Booms and Subsidies

Consider the polar case $\upsilon = \zeta + \xi(1-\upsilon)$ under substitutability, $\sigma > 1$, with a linear damage function, $\chi = 1$, and assume the following scenario: the economy experiences a resource boom and the government decides to subsidize the primary sector by reducing the commodity tax rate, τ, below zero. Policies of this kind are frequently implemented in resource-rich countries. In graphical terms, the effect of the resource boom is a displacement to the left of the current population-resource ratio with no change in the steady states (ℓ'_{ss}, b'_{ss}) and (ℓ''_{ss}, b''_{ss}). The subsidy, instead, modifies the positions of both the regular steady state and the extinction threshold by reducing ℓ'_{ss} and increasing ℓ''_{ss}. Figure 3f shows that the combination of resource boom and subsidies move the economy closer to the extinction threshold for two independent reasons: while the larger resource endowment reduces the *current* population-resource ratio ℓ, the lower tax rate τ shifts the mortality-trap threshold ℓ''_{ss} to the right. Both these effects push the economy away from the preshock regular steady state and, if they are strong enough, may even derail the economy from the regular path and push it into the mortality trap. Using Figure 3f, suppose the preshock level of the population-resource ratio is ℓ_0 so that the economy is initially converging to the preshock regular steady state. If the resource boom is relatively small, the postshock population-resource ratio may fall to a moderately lower level like $\ell_1 > \ell''_{ss}$, which still guarantees convergence to the (new, postshock) regular steady state. If the increase in Ω is substantial, instead, the postshock population-resource ratio may fall down to $\ell_2 < \ell''_{ss}$ and trigger population implosion: the fertility rate jumps up and keeps growing but never reaches the exploding mortality rate.[27] These and the previous considerations make our general conclusion evident: labor-poor countries with abundant polluting resources face larger mortality traps. If the governments of these countries respond to new resource discoveries with higher subsidies to the primary sector—a policy often justified with the need to escape underdevelopment traps—the possibility of falling into a different trap characterized by ever-growing mortality should be taken seriously.

6 | Pollution-Attributed Deaths and Cross-Country Evidence

The Lancet Commission estimated the pollution-attributed mortality rates—the empirical counterpart of m_p in our model—for

more than 200 countries in 2019. The mapping between the damage functions used by the Lancet Commission and our mortality function (5) hinges on the distinct concepts of dose, concentration, and rival absorption (Subsection 6.1). We combine the estimates of pollution-attributed deaths with World Bank (2024) data on PM concentrations for more than 180 countries to perform cross-country regressions. Our empirical analysis focuses on the shape of the mortality function (Subsection 6.2), the generation of pollution (Subsection 6.3), and the role of resource abundance in mortality outcomes (Subsection 6.4).

6.1 | Pollution Absorption and Dose-Dilution Effects

Dose, exposure, and concentration. According to conventional definitions, *dose* is the amount of the pollutant that crosses one of the body's boundaries and reaches the target tissue, *exposure* refers to any (outer or inner) contact between a contaminant and the human body, *concentration* is the amount of pollutant per unit volume, for example, micrograms of "PM" per cubic meter of air.[28] The empirical literature typically uses concentration per cubic meter as a measure of individual doses (see, e.g., Burnett and Cohen 2020). This is an understandable way to circumvent the problem of measuring actual individual doses, but it is an approximation that neglects the distinction between pollution—a physical characteristic of the environment at a certain place and time—and individual dose—the result of the interaction between the environment and a specific individual. The distinction must be made in a dynamic model like ours where pollution and population are endogenous and change over time. We capture this aspect by identifying E with *aggregate pollution*—for example, concentration of PM over the whole land area of the economy where population L operates—and defining the individual dose D as the outcome of a matching process with well-defined characteristics. Assuming *concentration* = *exposure* = *dose*, as the empirical literature does, is a way to bypass the need to estimate dose-dilution effects. In fact, there have been no attempts, to our knowledge, to estimate the effect of population levels on individual absorption at given concentration. This leaves open the empirical question of which value of ζ would allow our mortality function to match the pollution-attributed mortality rates estimated by the Global Burden Disease Study (IHME 2024).

Empirical methodologies. The estimates of pollution-induced mortality in Global Burden Disease studies are based on the formula

$$\text{PAF} = 1 - \left(\text{RR}_{(d,d_{\min})}\right)^{-1}, \tag{58}$$

where the left-hand side is the *pollution-attributed fraction* (PAF) of total deaths and $\text{RR}_{(d,d_{\min})}$ is a relative risk function built on the concept of odds ratio, namely, the probability of death if the average person absorbs d units divided by the probability of death if the person absorbs $d_{\min}$ units, where $d_{\min}$ is the counterfactual with zero deaths from pollution. Relative risk functions can take many different shapes, but in all cases they must be increasing in d and satisfy $\text{RR}_{(d,d_{\min})} \geqslant 1$, with equality when $d = d_{\min}$ (see, e.g., Burnett and Cohen, 2020). We now derive the explicit relation between these objects and our model. First, we solve Equation (58) for RR and note that in our model $d_{\min} = 0$. This gives us $\text{RR}_{(d,0)} = (1 - \text{PAF})^{-1}$. Next, we note that PAF is the empirical counterpart of the ratio M_p/M in our model. We thus use Equations (2) and (4) to write

$$\text{RR}_{(d,0)} = 1 + \frac{1-\bar{m}}{\bar{m}} \cdot D(E,L), \tag{59}$$

which shows that our representation of excess deaths is consistent with empirical models: the right-hand side of (59) satisfies the necessary properties of relative risk functions identified in empirical studies. The difference between our theory and the empirical literature is that the latter assumes that "concentration per cubic meter" is a good proxy for individual dose absorption whereas, in our specification, the individual dose is positively related to aggregate pollution (i.e., concentration over the economy's land area) and negatively related to total population (i.e., residents living in the land area).

Pollutants and humans. Consider a group of L identical individuals located in a volume of space. Inject in that space a quantity E of a pollutant and denote by E_P cumulative exposure, that is, the sum of individual exposures (units of pollutant making contact with the body of an individual) so that average exposure equals E_P/L. Next, we follow the literature and write the individual dose, or absorption rate, as an increasing and concave function of average exposure, that is,

$$D = h\left(\frac{E_P}{L}\right). \tag{60}$$

The mapping between the damage functions assumed in the related literature and our mortality function hinges on whether the pollutant is nonrival or rival across individuals.

Nonrival pollutants. A pollutant is nonrival if multiple individuals can absorb it simultaneously with no reduction of its effect on each individual's health. An example of a process that meets this definition is climate change: rising temperature is viewed as affecting every individual simultaneously and equally. The literature on climate change specifies the individual damage as an increasing function of temperature, and temperature is an increasing function of aggregate measures of greenhouse gasses (see, e.g., Bressler 2021). Since every individual is equally exposed to total pollution, cumulative exposure is $E_P = E \cdot L$, average exposure coincides with E, and the dose-absorption rate (60) reads $D = h(E)$. Our mortality function captures this type of process in the no-dilution case, $\zeta = 0$, which yields $m_p = \mu E^{\chi}$. However, our focus in this paper is neither climate change nor rising temperature. The most relevant risks for individual health come from PM and water pollution: these pollutants cause noncommunicable diseases through absorption of individual doses that are rival since PM units inhaled (or contaminated water consumed) by an individual cannot be absorbed by other individuals.

Rival pollutants and dose dilution. We henceforth label as "idle," the units of the pollutant that do not make contact with any individual. By construction, the number of idle units is $E - E_P$. If we assume no idle units of pollutant (i.e., each unit of the pollutant makes contact with an individual), pure rivalry implies that cumulative exposure is $E_P = E$, average exposure is E/L, and the dose-absorption rate is $D = h(E/L)$. Our mortality function

describes this scenario in the balanced-dilution specification where $\zeta = 1$ yields $m_p = \mu (E/L)^{\chi}$. However, if we relax the hypothesis of no idle units, the dilution effect of population is generally not proportional. Consider a preshock situation in which the initial population level implies $E - E_P > 0$ idle units of the pollutant. Assume an increase dL of the mass of individuals holding pollution E constant. The increase in population can affect average and cumulative exposure in radically different ways. At one extreme is the scenario where cumulative exposure E_P does not change and average exposure falls because the dL newcomers only make contact with nonidle units (i.e., units that would have made contact with other individuals in the preshock situation): in this case, a larger population dilutes linearly individual exposure. At the opposite end is the scenario where average exposure does not change and cumulative exposure E_P increases because all the dL newcomers only make contact with previously idle units: in this case there is no dose dilution. Between the two polar cases, there is a continuum of scenarios where the dL newcomers absorb some idle and some nonidle units, implying that the increase in population dilutes average exposure less than linearly. We can think of these scenarios as cases with intermediate degrees of rivalry and represent them by the function $D = h(E, L) = \mu_0 E^{\chi} L^{-\chi\zeta}$, where parameter ζ determines the degree of dose dilution. Our main hypothesis is $\zeta > 0$ because albeit perfect rivalry might not apply, most pollutants are rival to some degree since they are physical objects. Pure nonrivalry only applies in special cases like temperature. We can think of $h(E, L)$ more generally as a convenient reduced form capturing the threshold, congestion, and saturation effects of chemical and physiological processes whose dilution laws do not obey simple proportionality rules.

6.2 | Mortality Function

Consider the cross-country regression equation

$$\ln m_{p,i} = \beta_0 + \beta_1 \ln[\text{agg. pollution}_i] + \beta_2 \ln[\text{population}_i] + \beta_3 \ln \check{x}_i + \epsilon_i, \quad \text{(R.1)}$$

where i is the country index, $\check{x}_i$ is an additional country-specific regressor, and ϵ_i is an error term. Estimating (R.1) allows us to check whether population can explain the cross-country variation in excess mortality rates *at given pollution levels*. A negative coefficient $\beta_2^{\text{est}} < 0$ will suggest the existence of dose dilution. Since the mortality function in our model reads

$$\ln m_p = \ln \mu + \chi \ln(E) - \chi\zeta \ln(L),$$

we can interpret the coefficients of (R.1) as $\beta_1 = \chi$ and $\beta_2 = -\chi\zeta$ and define the implicit dose-dilution coefficient as $\zeta^{\text{est}} = -\beta_2^{\text{est}}/\beta_1^{\text{est}}$. Recalling our definitions in Subsection 2.3, the case $\zeta = 0$ excludes dose dilution, whereas $\zeta = 1$ implies *balanced dilution*, that is, the mortality rate depends on pollution *per capita*. We will investigate these points by providing confidence intervals for the ratio ζ^{est} and by performing robustness checks.

The data we use for $m_{p,i}$ are the pollution-attributed mortality rates in 2019 publicly available from the World Bank (2024).[29] On the right-hand side of (R.1), we calculate "aggregate pollution" as PM-concentration times land area,[30] while "population" is the number of residents in 2019.[31] The large data set naturally implies heterogeneity in technologies and stages of development across countries. We control for stage of development by including PPP-adjusted real GDP per capita as the additional regressor $\check{x}_i$. The expected sign is $\beta_3 < 0$ because given the same pollution level, less (more) developed countries tend to have higher (lower) risk of death due to lower (higher) quality of health care and reduced (enhanced) access to risk-reducing technologies. The top panel in Table 1 reports the estimation results for (R.1). Aggregate pollution always exhibits a significant positive coefficient $\beta_1^{\text{est}} > 0$. The second column shows that pollution and population have strongly significant coefficients of opposite sign and suggests not only the existence of dose dilution, $\beta_2^{\text{est}} < 0$, but also the possibility of balanced dilution since $\zeta^{\text{est}} = -\beta_2^{\text{est}}/\beta_1^{\text{est}} = 0.96$ is very close to unity. The third column shows that both $\beta_2^{\text{est}} < 0$ and $\zeta^{\text{est}} \approx 1$ still hold after controlling for GDP per capita, with the new coefficient β_3^{est} being significant and negative, that is, pollution kills more in less developed countries at given pollution and population levels. These conclusions are confirmed if we repeat the estimations in the smaller sample that only includes high-income countries[32]: see columns (4)–(6) in the top panel of Table 1.

The results for (R.1) reported in Table 1 include calculations of confidence intervals for the ratio ζ^{est} using the standard method (second-last row of the upper panel) and the alternative Fieller method (last row), both at the 95% level.[33] The lower bounds are substantially higher than zero in all cases, supporting the *existence* of dose dilution. Concerning the *intensity* of dose dilution, the results obtained using Fieller's method show that a unit coefficient ratio remains firmly in the midrange of each interval in all cases, implying that balanced dilution is by no means unreasonable: assuming $\zeta = 1$ in the theoretical model appears fully compatible with empirical evidence. We investigated both points further by performing two robustness checks. First, regarding the existence of dose dilution, we ran alternative regressions specified in terms of PM-concentration and population density, obtaining results that confirm strongly significant coefficients of opposite sign: *given the pollution level, less populated economies exhibit higher excess mortality rates from pollution than more populated countries*. Second, we tested the balanced-dilution hypothesis by specifying an alternative regression in terms of pollution per capita: the results support the hypothesis $\zeta = 1$ and suggest an elasticity of the pollution mortality rate to pollution *per capita* between 0.09 and 0.25. The Appendix reports detailed estimation results for both robustness checks.

6.3 | Pollution Generation

We assess empirically the process that generates pollution with the regression equation

$$\ln[\text{agg. pollution}_i] = \gamma_0 + \gamma_1 \ln[\text{industry output}_i] + \gamma_2 \ln[\text{population}_i] + \gamma_3 \ln[\text{pop.density}_i] + \gamma_4 \check{x}_i + \epsilon_i, \quad \text{(R.2)}$$

where "industry output" is a real index of total manufacturing production and "pop.density" is the number of residents divided by the country's land area. Equation (R.2) is comprehensive in the sense that it does not exclude economies without natural resources, it can be estimated for all countries in the sample. Recalling the discussion in Section 2.2, the expected signs of the

TABLE 1 | Regression results, see Section 6 for description.

Regression (R.1)	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Depend.var.: m_p	**Full sample**	**Full sample**	**Full sample**	**High income**	**High income**	**High income**
Aggregate pollution	0.101***	0.249***	0.088***	0.084***	0.223***	0.111***
	(0.029)	(0.050)	(0.030)	(0.031)	(0.048)	(0.041)
Population		−0.238***	−0.093***		−0.236***	−0.119***
		(0.066)	(0.039)		(0.065)	(0.054)
GDP per capita			−0.706***			−0.820***
			(0.038)			(0.107)
Constant	−8.833***	−7.226***	−0.525	−9.133***	−7.422***	0.684
	(0.439)	(0.615)	(0.504)	(0.451)	(0.638)	(1.172)
Observations	177	177	177	108	108	108
R^2	0.064	0.129	0.712	0.066	0.169	0.471
Implicit dose dilution ζ^{est}		0.96	1.07		1.06	1.07
Confidence interval (95%)		[0.66,1.26]	[0.58,1.55]		[0.71,1.40]	[0.51,1.63]
Fieller method (95%)		[0.60,1.26]	[0.39,1.72]		[0.66,1.43]	[0.22,1.91]

Regression (R.2)	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**
Depend.var.: Aggregate pollution	**Full sample**	**Full sample**	**Full sample**	**Full sample**	**Full sample**
Industry output	0.777***	1.043***	1.428***	0.436***	
	(0.061)	(0.042)	(0.245)	(0.095)	
GDP per capita		−1.509***	−1.929***	−0.757***	
		(0.092)	(0.279)	(0.108)	
Population			−0.434	0.616***	1.108***
			(0.272)	(0.105)	(0.021)
Population density				−0.925***	−0.984***
				(0.027)	(0.032)
Constant	−7.524***	−0.835	−0.967***	3.643***	1.364***
	(1.742)	(1.180)	(1.178)	(0.455)	(0.374)
Observations	183	183	183	183	183
R^2	0.470	0.787	0.790	0.972	0.956

Note: Standard errors in parentheses. ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$.

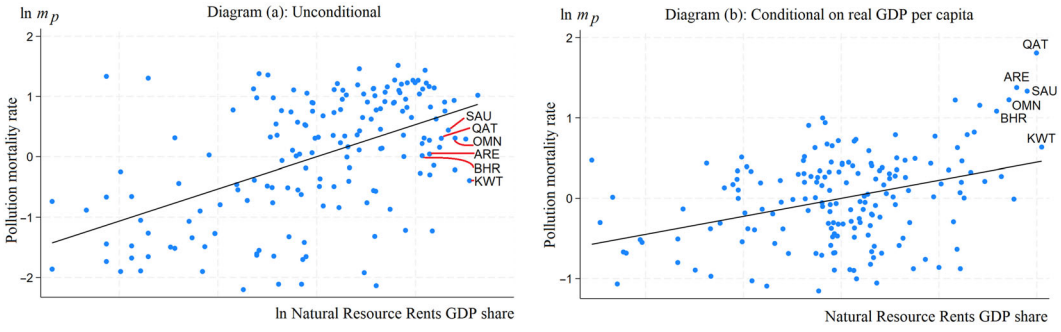

FIGURE 4 | Diagram (a) reports pollution-attributed mortality rates (2019 estimates) versus GDP shares of total resource rents (2010–2019 average), the regression line corresponds to column (1) of regression (R.3) in Table 2. Diagram (b) reports fitted values after controlling for stage of development (real GDP per capita in 2019), the regression line corresponds to column (2) of regression (R.3) in Table 2.

TABLE 2 | Regression results, see Section 6 for description.

Regression (R.3)	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Depend.var.: m_p	**WB data**	**WB data**	**IEA data**	**IEA data**	**WB data**	**WB data**
GDP per capita		−0.649***		−0.803***		−0.724***
		(0.038)		(0.120)		(0.051)
Total resource rents (GDP share)	0.266***	0.112***				
	(0.033)	(0.022)				
Oil reserves per capita			−0.009	0.119**		
			(0.060)	(0.046)		
Oil rents (GDP share)					0.117***	0.093***
					(0.036)	(0.021)
Constant	−7.509***	−1.326***	−7.486***	−0.007	−7.408***	−0.459
	(0.071)	(0.365)	(0.242)	(1.130)	(0.090)	(0.489)
Observations	172	172	45	45	114	114
R^2	0.278	0.734	0.001	0.517	0.008	0.678
Regression (R.4)	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Depend.var.: Aggregate pollution	**WB data**	**IEA data**	**WB data**	**WB data**	**IEA data**	**WB data**
Industry output				0.247***	0.769**	0.458***
				(0.118)	(0.313)	(0.166)
GDP per capita	−0.180***	−0.552***	−0.297***	−0.471***	−1.218***	−0.770***
	(0.034)	(0.098)	(0.044)	(0.143)	(0.286)	(0.176)
Population	1.035***	1.046***	1.035***	0.774***	0.324	0.575***
	(0.020)	(0.053)	(0.028)	(0.126)	(0.297)	(0.168)
Population density	−0.875***	−0.766***	−0.866***	−0.873***	−0.817***	−0.865***
	(0.030)	(0.063)	(0.034)	(0.030)	(0.063)	(0.033)
Total resource rents (GDP share)	0.142***			0.109***		
	(0.022)			(0.027)		
Oil reserves per capita		0.177***			0.105***	
		(0.045)			(0.052)	
Oil rents (GDP share)			0.110***			0.067***
			(0.018)			(0.024)
Constant	3.677***	6.433***	4.952***	3.541***	2.323	3.564***
	(0.449)	(1.393)	(0.670)	(0.450)	(2.124)	(0.821)
Observations	171	45	114	171	45	114
R^2	0.967	0.941	0.954	0.967	0.949	0.957

Note: Standard errors in parentheses, ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$.

unknown coefficients are $\gamma_1 > 0$, $\gamma_2 > 0$, and $\gamma_3 < 0$: aggregate pollution is supposed to increase with industry output and with the population level due to scale effects on household-generated emissions, whereas population density should give rise to emission-reducing effects. In estimating (R.2), aggregate pollution data are the same as in regression (R.1); we construct the real index "industry output" by multiplying the industry share of total GDP by the level of PPP-adjusted aggregate GDP at constant prices. Using this index of *aggregate industrial GDP* as the main control for firms' emissions of PM allows us to keep *per capita overall GDP* as the additional regressor $\check{x}_i$ that controls for country i's stage of economic development. The hypotheses are $\gamma_1 > 0$ and $\gamma_4 < 0$ as long as both regressors are included: aggregate industrial output raises aggregate pollution, but more (less) developed economies have enhanced (reduced) access to low-emission production technologies, stronger (weaker) environmental regulation, and higher (lower) capacity for spending in pollution abatement. In other words, we expect $\gamma_4 < 0$ due to the "defensive effect" of economic development (cf. Subsection 2.2).

The lower panel in Table 1 reports the results for regression (R.2): all the estimated coefficients have the predicted signs. Industry output has a significant positive coefficient (first column) which is confirmed after controlling for real GDP per capita (second column). Adding population does not immediately yield a significant elasticity (third column), but simultaneously controlling for population level and population density yields $\gamma_2 > 0$ and $\gamma_3 < 0$, that is, a positive scale effect of the population level on total emissions, and an emission-reducing effect of population density. The coefficients remain strongly significant when all the regressors are considered simultaneously (fourth column). The results on population level and density, $\gamma_2 > 0$ and $\gamma_3 < 0$, also hold in isolation from the effects of industry output and per capita GDP (fifth column).

6.4 | Resource Abundance, Mortality Outcomes, and the Pollution Channel

Our theory shows that if the overall damage-dilution rate defined in (11) is sufficiently high, the equilibrium mortality rate m_p is higher the smaller the population-resource ratio ℓ: resource abundance leads to higher excess mortality rates caused by pollution. Instead, when the overall damage-dilution rate is relatively weak, m_p is increasing in ℓ so that excess mortality falls with resource abundance. All the possible subcases are documented in Figure 1 with the associated parameter restrictions. From an empirical standpoint, our large sample will likely include subsets of countries displaying opposite comovements between mortality and resource abundance. Still, the prevailing trend that we see in our data set appears to be a positive relationship between m_p and resource abundance—arguably, because resource-rich economies tend to produce more PM and are thus prone to experience higher excess mortality induced by pollution. As a first step, consider a basic regression between m_p and an index of resource abundance

$$\ln m_{p,i} = \delta_0 + \delta_1 \ln [\text{res. abundance}_i] + \delta_2 \ln \check{x}_i + \epsilon_i, \qquad \text{(R.3)}$$

where we deliberately exclude pollution from the right-hand side because resource abundance is supposed to affect mortality through emission generation (see below). In order to measure resource abundance, we can use two conceptually different proxies. The first is "total natural resource rents as a share of GDP," a comprehensive index of resource specialization which includes all types of resources and for which available data cover 172 countries. The second option is to focus on specific resources by means of a physical index, like "oil proven reserves per capita," that is conceptually close to $\Omega/L = \ell^{-1}$ in our theoretical model but for which the available IEA data only cover 45 countries (International Energy Agency 2024). A possible compromise is to measure oil abundance as "oil rents as a share of GDP" which guarantees a much larger sample—data cover 114 countries—but is still an index of specialization.[34] The upper panel of Table 2 shows the estimation results for Equation (R.3). Total rents and oil rents are significantly positively correlated to excess mortality with and without controlling for GDP per capita, $\check{x}_i$. Oil reserves per capita display a positive significant coefficient after controlling for GDP per capita.[35]

To be consistent with the theory, these positive correlations must originate in a positive contribution of resource abundance to pollution generation. We test this channel by extending Equation (R.2) to include resource abundance as an additional regressor. The bottom panel of Table 2 labels the results for these estimations as "Regression (R.4)." The first three columns exclude "industry GDP" from the regression, whereas columns (4)–(6) include it. In all cases, each index of resource abundance exhibits a positive and significant coefficient without affecting our previous results on population level and density effects. The conclusion is that the cross-country relationship between m_p and resource abundance appears to be positive because resource-rich economies tend to generate ceteris paribus more PM. Further inspection of data and regression results shows that oil-producing countries in the Middle East actually form a cluster exhibiting very high pollution and associated mortality: despite the fact that United Arab Emirates, Qatar, Kuwait, Saudi Arabia, Oman, and Bahrain are solidly high-income economies, their pollution-attributed mortality rates are among the highest in the whole sample even *without* controlling for GDP per capita: see diagram (a) in Figure 4. Controlling for GDP per capita makes this group of countries stand out as the undoubted top cluster in the sample—see diagram (b). We can see this as a real-world example of the "decoupling prediction" in our model whereby resource-rich countries may be exposed to high mortality from pollution even at advanced stages of development.

7 | Conclusion

In stark contrast to the magnitude of pollution-induced mortality reported in the empirical literature, there is little to no recognition of such an important phenomenon in macroeconomic models of growth and development. Filling this gap requires tractable models in which economic growth, fertility, and mortality are simultaneously endogenous and interconnected via equilibrium relationships. Our model suggests the long-run consequences of interactions between pollution and population dynamics on economic growth and can stimulate new empirical studies on the relationship among pollution, the mortality rate (or population dynamics), and innovation.

We have shown that unlike conventional pollution externalities, deadly spillovers affect welfare through multiple channels and that the response of the equilibrium mortality rate to population size is generally ambiguous and often nonmonotonic. This relationship between mortality and population reflects not only the emission intensity of primary production but also damage-dilution effects induced by population size/density and labor reallocation effects caused by technology. Under parameterizations that yield empirically plausible paths—prominently, a transitional fertility decline leading to a finite endogenous population level—deadly spillovers modify potential population in the long run, productivity growth in both the short and the long run, and may even create mortality traps. From a growth perspective, our framework shows that exogenous shocks that (a) increase long-run population capacity accelerate TFP growth during the transition via net entry of firms, while shocks that (b) reduce long-run mortality rates increase TFP growth in the long run via faster rates of vertical innovations. Under certain conditions, policy-induced shocks—like an increase in environmental taxes in the presence of substantial damage dilution—can simultaneously produce both outcomes, (a) and (b). More generally, our model

suggests that emission taxes may yield double dividends by increasing long-run population capacity, whereas subsidies to primary production reduce long-run population capacity and may increase the risk of population implosion. Subsidizing commodity production during a resource boom can have disastrous consequences if the primary sector's technology does not change. These considerations suggest that some novel thinking is called for in the debate on the prospects of many developing countries where discoveries of natural resources are accompanied by (implicit or explicit) subsidies designed to foster their exploitation.

Our analysis is also relevant for the macroeconomic models used by researchers and international organizations to forecast demographic trends and to calculate the welfare cost of pollution (e.g., OECD 2016). These works typically assume a mortality function that only depends on pollution concentration, estimate the relevant elasticity by means of regressions between past pollution levels and pollution-attributed deaths, and apply the resulting mortality function to model-generated projections of future pollution levels in order to obtain projections of future deaths. Our results suggest that (i) the mortality function should include population as a second argument with negative elasticity, and (ii) the model generating projections of future emissions should incorporate the effects that population level and population density exert on pollution generation. The resulting overall rate of damage dilution can in fact be a crucial determinant of mortality rates and of the associated welfare costs. Estimating such relationships and including them in the calculations of demographic trends and pollution costs is our main suggestion for this important body of research.

Acknowledgments

We thank conference participants at the Royal Economic Society, European Economic Association, SURED, and various seminars for useful comments. We are grateful to Gunnar Bårdsen for useful suggestions. The usual disclaimer applies.

Data Availability Statement

The data that support the findings of this study are openly available in openICPSR at https://www.openicpsr.org/openicpsr/project/213062/version/V1/view reference number: openicpsr-213062

Endnotes

[1] In the 2022 update, the Lancet Commission emphasized the change in the composition of total deaths. The number of deaths attributable to types of pollution associated with extreme poverty have declined, but this reduction is offset by deaths attributable to air and chemical pollution which have risen by 7% since 2015 and by over 66% since 2000 (Lancet Planet Health 2022). Ritchie and Roser (2020a, 2020b) show that all four main causes of death—heart disease, cancer, respiratory diseases, and infections—exhibit a strong relationship with air pollution. See also Schlenker and Walker (2016) and the literature cited therein.

[2] Demographers forecast a leveling off of world population within the next century (e.g., United Nations 2019).

[3] Non-Malthusian theories of the population level are even rarer. Brunnschweiler et al. (2021) propose a different approach where population is stabilized by the dilution of financial wealth in an OLG economy populated by disconnected generations.

[4] A high rate of pollution-reducing technical change is a precondition for sustainable long-term growth (Brock and Taylor 2005). Models of optimal pollution control study whether the sustainability condition is satisfied ex post under endogenous investment in clean technologies and show that poverty traps induced by pollution are indeed a possible outcome (Smulders and Gradus 1996; Xepapadeas 1997).

[5] An interesting application of this reasoning is in models with endogenous lifetime (Blackburn and Cipriani 2002; Chakraborty 2004) where households optimize over finite horizons and longevity rises with income. The interaction produces a stable steady state with high income but also a poverty trap with low income and short lifetime.

[6] In de la Croix and Gosseries (2012), emission taxes prompt agents to allocate more time to tax-free activities such as reproduction so that pollution control leads to larger population and lower output per capita. The normative implication is that imposing population caps would actually increase welfare by increasing production per capita.

[7] We treat pollution as a flow in order to obtain neat analytical results. Nothing of substance would change in our analysis by modeling pollution as a stock-flow process—which would create an additional state variable in the model and require a substantial amount of extra algebra.

[8] We omit the case $\zeta < 0$ because we are interested in dose dilution, not its opposite. Allowing for $\zeta < 0$ makes m_p increasing in population size, which yields general equilibrium results that are qualitatively the same as in the no-dose dilution case.

[9] We assume that one unit of the endowment provides one unit of services. Thus, Ω denotes the endowment as well. We considered extensions where the natural resource is either renewable or exhaustible (Ω is an endogenous state variable) and noted that they complicate the analysis substantially without adding insight. We thus decided to focus on the simpler case of a fixed endowment.

[10] Bork and Schrauth (2021) obtain −0.14 for O3 ground-level concentrations and 0.08 for PM in Germany; Carozzi and Roth (2023) estimate an elasticity for PM of 0.14 in the United States; Chen et al. (2020) find a negative elasticity for PM of −0.26 in Chinese cities after controlling for city fixed effects in a panel specification. According to Ahlfeldt and Pietrostefani (2019), the elasticity for PM ranges from 0.08 to 0.15 in advanced western economies.

[11] Assumption (14) does not include the disutility from deaths because nothing substantial would change in the analysis if it were explicitly incorporated.

[12] The restriction $0 < \psi < 1$ implies that for each group the elasticity of utility with respect to individual consumption exceeds the elasticity of utility with respect to the size of the group. Moreover, as we show in the Appendix, the maximization problem of the household is well defined only if the condition $\psi(1-\alpha) < 1-\alpha$ holds.

[13] The fixed operating cost, ϕ, ties product proliferation to population growth, as explained in detail in Peretto and Connolly (2007). Since variety expansion entails the replication of fixed operating costs, a growing number of product lines puts pressure on the economy's resources: in the long run, the mass of firms is bound by labor availability and, hence, by population size.

[14] See Peretto and Connolly (2007) for the microfoundations of this assumption and a discussion of alternatives that deliver the same qualitative results. In the present model where $wL_{N_i} = w\beta L/N$, satisfying the free-entry condition (28) compatibly with total labor supply requires $\dot{N}L_{N_i} = \dot{N} \cdot (\beta L/N) < L$, where N is endogenous. In a free-entry equilibrium where new firms enter the market, this inequality holds automatically because the flow of new entrants $\dot{N}$ adjusts instantaneously to satisfy the labor market clearing condition—that is, employment in entry does not exceed the labor supply: $L_N = \dot{N}L_{N_i} = \dot{N} \cdot (\beta L/N) < L$. In an no-entry equilibrium where $\dot{N} = 0$, we likewise have $L_N = 0 < L$.

[15] With the wage set at $w = 1$, p_c is the price of the final good in units of labor. Therefore, the real wage, w/p_c, grows when $\dot{p}_c/p_c < 0$ and a long-run equilibrium featuring constant expenditure Y and growth of the physical variables $c_L \equiv C_L/L$ and $c_B \equiv C_B/B$ is characterized by $\dot{c}_L/c_L = \dot{c}_B/c_B = -\dot{p}_c/p_c$, that is, growth comes from the rate of decline of the relative price of the final good.

[16] Expression (32) yields $\partial \mathrm{Y}(p_\omega)/\partial p_\omega < 0$ if $\sigma > 1$, $\partial \mathrm{Y}(p_\omega)/\partial p_\omega = 0$ if $\sigma = 1$, and $\partial \mathrm{Y}(p_\omega)/\partial p_\omega > 0$ if $\sigma < 1$.

[17] Proposition 2 characterizes the equilibrium relations among endogenous variables: to avoid confusion, we drop the time argument unless necessary.

[18] Figure 1 shows that for any value of σ, there are cases in which $m^*(\ell)$ is decreasing at least locally. Decreasing mortality occurs less under complementarity, $\sigma < 1$, because the cost-share effects underlying result (41) are reversed. This makes the primary-employment effect stronger for small population.

[19] A version of the model with a time cost of reproduction delivers the same qualitative results as the one presented here. It follows that this property of our approach does not depend on the cost of reproduction being in units of the consumption good.

[20] The analysis of the dynamic system with deadly spillovers and strict complementarity, $\sigma < 1$, is in the Appendix for completeness.

[21] The case $b_{ss} - \bar{m} - \tilde{\mu} < 0$ is like diagram (c) but with the $\dot{\ell} = 0$ locus lying above b_{ss}. This reverses the direction of the arrows along the $\dot{b} = 0$ locus and delivers persistent population decline.

[22] The case with no steady states looks like Figure 2, graphs (f)–(g), but with the $\dot{\ell} = 0$ locus so high that there is no intersection with the $\dot{b} = 0$ locus.

[23] See Figure 2 (bottom panel). Assuming an initial condition $\ell(0) < \ell'_{ss}$ where ℓ'_{ss} is the regular steady state, the equilibrium path converging to ℓ'_{ss} features positive population growth, $\dot{\ell} > 0$, and falling birth rates, $\dot{b} < 0$, under substitutability. The fact that $\dot{b} < 0$ along such paths is due to the negative slope of saddle path. The negative slope of the saddle path reflects the negative response of expenditure per capita to rising resource prices under substitutability.

[24] This mechanism is analyzed in detail in Peretto and Connolly (2007). Since the entry cost is proportional to the population–firms ratio, L/N, the long-run mass of firms relative to the population N_{ss}/L_{ss} responds positively to expenditure per capita $y^*(\ell_{ss})$ because as expenditure per capita rises, the size of the market expands more than proportionally to the entry cost and thus attracts entry. Entry, in turn, dilutes firms' market shares inducing each firm to reduce their in-house R&D efforts.

[25] In graphical terms, $\zeta + \xi(1-\upsilon) \geqslant \upsilon$ implies that the $\dot{\ell} = 0$ is L-shaped, so that the combined shifts of the $\dot{b} = 0$ locus (upward) and of the $\dot{\ell} = 0$ locus (downward) imply a steady state with lower mortality rate $m^*(\ell_{ss}) = b_{ss}$.

[26] In graphical terms, $\zeta + \xi(1-\upsilon) < \upsilon$ implies that the $\dot{\ell} = 0$ is U-shaped, and the regular steady state is likely to be found in the part of the curve with positive slope. Hence, the combined shifts of the $\dot{b} = 0$ locus (upward) and of the $\dot{\ell} = 0$ locus (downward) may imply either a lower or higher mortality rate $m^*(\ell_{ss}) = b_{ss}$ in the regular steady state, depending on the curvature and on the extent of the shift of the $\dot{\ell} = 0$ locus.

[27] Note that in the alternative case with weaker damage dilution, $\upsilon > \zeta + \xi(1-\upsilon)$, the increase in Ω would further reduce the gap between the two steady states. In graphical terms, $\upsilon > \zeta + \xi(1-\upsilon)$ would imply one downward shift of the $\dot{b} = 0$ locus and *two* upward shifts of the $\dot{\ell} = 0$ locus: one due to the subsidy and one due to the resource boom.

[28] See National Research Council (1988) for a detailed discussion of concepts and conventional definitions.

[29] See the Appendix for detailed data description.

[30] The Lancet Commission estimates PAFs on the basis of individual risk factors among which the prominent pollution index is the concentration of PM expressed as micrograms of PM25 per cubic meter—which is what we call "PM-concentration" in the main text. The economy-wide presence of PM25 is thus captured by multiplying PM-concentration by the size of the country's territory, that is, land area expressed in square meters.

[31] Both types of data are publicly available from the World Bank (2024), which reports PM-concentration for most countries over the years 2010–2019. We have calculated variants of "aggregate pollution"—one by using the level observed in 2019, the other one by using the 2010–2019 average levels—obtaining the same results. We report results based on the 2010–2019 average.

[32] In our estimation of (R.1), the high-income subsample is obtained by setting a threshold on PPP-adjusted real GDP per capita (our variable $\check{x}_t$) of USD 10,000 in the year 2019, were the currency unit is 2017-chained dollars. The set of countries is essentially the same as in the World Bank definition of high-income economies (i.e., those with a GNI per capita of USD 13,846 or more in the year 2022).

[33] A relevant difference between the standard method, also known as "delta method," and Fieller's (1954) approach is that the former calculates symmetric confidence intervals around the reference ratio—here represented by ζ^{est}—whereas Fieller's method generally yields asymmetric intervals. Lye and Hirschberg (2018) discuss the practical implications of the different properties of the two methods and recommend using both in order to draw robust conclusions.

[34] Resource rents and physical indices are not alternative proxies for the same variable, but they both allow us to test the model's predictions on the effects of per capita resource abundance on mortality and emissions. Physical indices, like oil reserves per capita, are a proxy for the *per capita* endowment Ω/L. The GDP share of resource rents, instead, is a proxy for the effects of the resource cost share $\mathrm{Y}(\ell)$, which depends on the labor-resource ratio $\ell = (\Omega/L)^{-1}$ and thus incorporates the effects of resource abundance *per capita* via input markets. In particular, under substitutability, the GDP share of resource rents increases with per capita resource abundance because $\mathrm{Y}'(\ell) < 0$. See Appendix E for details.

[35] We obtain the same results by replacing oil reserves per capita with 2019 oil production (barrels per day) divided by population. The reason is likely to be that oil is a fundamental energy source and its abundance per capita is strongly positively correlated with GDP per capita, which makes the oil abundance variable capturing part of the negative relationship between emissions and GDP per capita unless we control for the latter as an index of stage of development.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.