Mortality Shocks and Growth Cycles

Paul Shea*

Bates College

May 1, 2021

Abstract

We introduce an endogenous morality rate, where life expectancy increases with wealth, into a simple neoclassical growth model. Higher mortality causes agents to discount future utility at a higher rate. Depending on the mortality function, the model can produce either a single stable steady state or multiple stable steady states with different levels of output, consumption, and life expectancy. Mortality shocks may cause the model to shift between neighborhoods of different stable steady states. Anticipated increases in future mortality may shift the model from a high output-life expectancy steady state to a low output-life expectancy steady state. Unexpected increases in mortality, however, can have the opposite effect, moving the model to a steady state with higher output and life expectancy. The latter result provides a new explanation for how the Black Death may have contributed to permanent changes in output in fourteenth century Europe.

JEL Classification: E13, E20, N13, O40.

Keywords: endogenous mortality, growth cycles, multiple equilibria, pandemics.

*pshea@bates.edu

The covid-19 pandemic has generated increased interest on the interaction of macroeconomics and public health. This paper examines the role of endogenous mortality in a simple neoclassical growth model. Mortality is subject to transitory shocks representing events such as pandemics, the introduction of new pathogens, climate shocks, or wars. These shocks may either affect future expected mortality rates or they may be unexpected shocks to the population or capital stock. In addition to being affected by shocks, mortality also endogenously decreases with average wealth. There is ample evidence in support of this assumption, both in pre-industrial societies (Clark and Hamilton (2006)), which our model is designed around, and in modern advanced economies (Chetty, Stepner, and Abraham (2016)).

The model's other main assumption is that higher mortality rates lead agents to more heavily discount the future. The nature of the model's equilibrium then depends on the specific mortality function mortality function. If mortality is relatively insensitive to wealth, then the model yields a unique steady state. In this case, endogenous mortality adds volatility by amplifying and propagating the effect of mortality shocks. When mortality exogenously decreases, agents put more weight on future periods. This leads to an increase in the capital stock which then further increases life expectancy, amplifying the initial exogenous shock.

If mortality is sufficiently sensitive to wealth, however, then the model may exhibit multiple steady stable states. At a low capital steady state, mortality is high, leading agents to heavily discount the future. This induces low capital accumulation, which reinforces high mortality, keeping the model around its low output-high mortality steady state. High output-low mortality steady states may also exist where lower discount rates cause households to maintain a higher capital stock which then allows for longer life expectancy. We present one mortality function that is sensitive to wealth initially, but then reaches a biological limit on life expectancy for a pre-industrial society. This mortality function yields three steady states, with the highest and lowest (in terms of capital) being stable.

We then analyze the impact of mortality shocks on the model. The first type we examine is a stationary shock to upcoming mortality, which may capture the impact of a new pathogen or a worsening of the climate. The most important result is that a temporary decrease in mortality can induce enough capital accumulation to switch the model from the neighborhood of the low output-high mortality steady state to that of the high output-low mortality steady state. A transitory increase in mortality can have the opposite effect. A temporary improvement in climate or health can thus lead to an indefinite improvement in economic conditions. To the best of our knowledge, this result is novel.

We also consider a different type of shock that directly affects capital per-worker. A sudden

decrease in population (or increase in capital) that is unexpected and affects upcoming mortality only through increased wealth per-person, can shift the model from the low output-high mortality steady state to the high output-low mortality steady state. This simulation is a approximation of a catastrophic plague, such as the arrival of the Black Death in Europe in the 1340s, that unexpectedly causes a large reduction in population. Recent scholarship suggests that the Black Death may have permanently increased living standards, especially in northern and western Europe (Pamak (2007)). Our model captures this result.

After a review of the related literature on Section 1, Section 2 presents the model and demonstrates the impact of temporary mortality shocks. Section (4) then shows the impact of unexpected, one-time shocks to the population or capital stock. Finally, Section 4 concludes.

1 Related Literature

Several other papers endogenize the mortality rate in macroeconomic models. The closest paper to ours is Chakraborty (2004). That paper also assumes that higher mortality rates lead to greater discounting. There, higher mortality and discounting leads to reduced investment in public health. This reinforces higher mortality and leads to lower growth rates. If capital's share of income is greater than $\frac{1}{2}$, then the model may become stuck in a low output steady state, an example of a development trap. Another closely related paper is Castelló-Climent and Doménech (2008). That paper also uses endogenous mortality to generate multiple steady states. The mechanism is quite distinct from the present paper. There, households with higher mortality have less incentive to invest in education. Lower education levels then reinforce higher mortality trapping the model in a low education steady state. If households can achieve high education levels, however, then there is a preferable steady state where high education and low mortality reinforce each other.

Other related papers include Blackburn and Cipriani (2002), who assume that mortality depends on parents' human capital in a model where agents choose their consumption, education, and fertility. Their model also yields multiple steady states where low mortality incentivizes lower fertility and more education, thus maintaining the low mortality rate. The model can also, however, become stuck in a suboptimal equilibrium defined by high mortality, high fertility, and low educational investment. Gori and Sodini (2020) constrict a model containing both private and public health expenditures. If these are compliments, then the model may exhibit both multiple steady states and expectational indeterminacy.

In contrast to the preceding papers, this paper uses a very simple mechanism to generate

multiple steady states with endogenous mortality. All that it entails is: 1) a discount rate that depends on mortality and 2) a mortality rate that initially depends sharply on wealth but where the relationship ceases to exist once mortality hits a biological minimum.

A major contribution of the present paper is to show how mortality shocks can lead to the model switching between between steady states. Growth models with multiple stable steady states often do not examine switching dynamics and we are unaware of another with this result. Other papers do, however, examine the dynamics of switching between steady states in different settings. One prominent example is Evans, Honkapohja, and Romer (1998). They design a model with multiple stable steady states by assuming both complimentary capital goods and increasing returns to scale. Expectational indeterminacy then allows the model to switch between high and low growth states. Comin and Gertler (2006) show similar switching between steady states in an endogenous growth model. Unlike those papers, the present paper considers a pre-industrial economy where the steady states differ by output level,s and not growth rates. These related papers also do not include endogenous mortality.

2 Model

The model is embedded in a simple neoclassical growth framework. Households solve a standard optimization problem:

$$Max_{C_t} \quad \sum_{i=0}^{\infty} \beta ln(C_{t+i}) \tag{1}$$

s.t.

$$K_{t+1} = (1-\delta)K_t + N_t^{1-\alpha}K_t^{\alpha} - C_t \tag{2}$$

Optimization then yields a standard Euler Equation.

$$\frac{1}{C_t} = \frac{\beta(1-\delta+\alpha N_t^{1-\alpha}K_t^{\alpha-1})}{C_{t+1}} \tag{3}$$

We now add our first novel assumption: that the discount factor, β, is simply the probability of being alive in the next period, which is just one less the mortality rate.[1] Furthermore, the rate is itself a decreasing function of wealth, which consists exclusively of capital in the model.[2]

[1] We also considered a version of the model where the discount factor included intrinsic impatience as well as the probability of death. This version produced no noteworthy differences and the present model thus excludes intrinsic impatience.

[2] Another version of the model included a fixed stock of land as additional input. This did not affect the

$$\beta_t = 1 - l(k_t)\mu_t \tag{4}$$

where

$$\mu_t = \mu_{t-1}^{\rho} e_t \tag{5}$$

and $ln(e_t)$ is mean-zero white noise.

There is substantial evidence showing that better economic conditions lead to reduced mortality, although the evidence does not strongly suggest whether the link depends primarily on capital, income, or consumption. Chetty, Stepner, and Abraham (2016) find a strong link between higher economic status and life expectancy exists in the contemporary United States. This assumption is also common in the literature on Malthusian economics. Steinmann, Prskawetz, and Feichtinger (1998), for example, develop a model where higher levels of human capital both increase the birth rate and decrease the death rate. Empirical evidence supports the basic Malthusian connection between economic performance and population growth in pre-industrial economies. Clark and Hamilton (2006) finds that increased wealth led to improved reproductive fitness in England between 1585-1638.

Mortality also depends on an exogenous AR(1) shock, μ_t. Positive values reflect higher than normal mortality which may result from wars, pandemics, famines etc. Lower values may reflect improved climate or other factors that temporarily improve life expectancy. We assume rational expectations where agents fully observe the shock and understand the stochastic process that governs μ_t.

It is straightforward to convert the model to a per-capita basis.

$$k_{t+1} = (1 - \delta)k_t + k_t^{\alpha} - c_t \tag{6}$$

$$\frac{1}{c_t} = \frac{(1 - l(k_t)\mu_t)(1 - \delta + \alpha k_t^{\alpha - 1})}{c_{t+1}} \tag{7}$$

The model thus consists of one state variable, k_t, one control variable, c_t, and one shock, μ_t. We now turn our attention to analyzing the different types of solutions that the model may exhibit. The nature of equilibrium depends primarily on the functional form of $l(k)$, the mortality rate:

model's main results.

Assumption 1: $l(0) < 1$, $l^{'}(k) \leq 0$, and there exists a minimum level of steady state mortality, $\bar{l}$ that is attained at some level of k.

Assumption 1 ensures that mortality cannot equal one, preventing the extinction of the species. It assumes that mortality is initially decreasing in wealth, but that there is some biological minimum on steady state mortality. Later, we show that Assumption 1 ensures the existence of at least one generally stable steady state.

Besides the mortality function, the model has only a few variables to calibrate. We set $\alpha = \frac{1}{3}$, the standard variable for capital's share of income. The depreciation rate is set at 10%, suggesting annual data. For now, we assume a constant population. This requires that the birth rate equals the mortality rate so that each deceased agent is replaced by a new household who takes over their capital. This is in contrast to the Malthusian literature which instead assumes that the birth rate itself is an increasing function of wealth.

Case I: A Unique Steady State

The model's steady states may be evaluated using just (7):

$$\frac{1}{1 - l(k)} = 1 - \delta + \alpha k_t^{\alpha - 1} \tag{8}$$

The left hand side of (8) is the inverse of the discount factor, while the right hand side is one plus the real interest rate. Assumption 1 ensures that, for low enough values of k, the right hand side is greater than the left hand side. Our assumption of a minimum mortality rate ensures that there is at least one point where the right hand side crosses from above, which typically results in a stable equilibrium. It is easy to design a mortality function which yields just a single stable steady state which generally occurs when $\frac{dl(k)}{dk}$ is sufficiently small. One such example that yields a steady state life expectancy of forty years is:

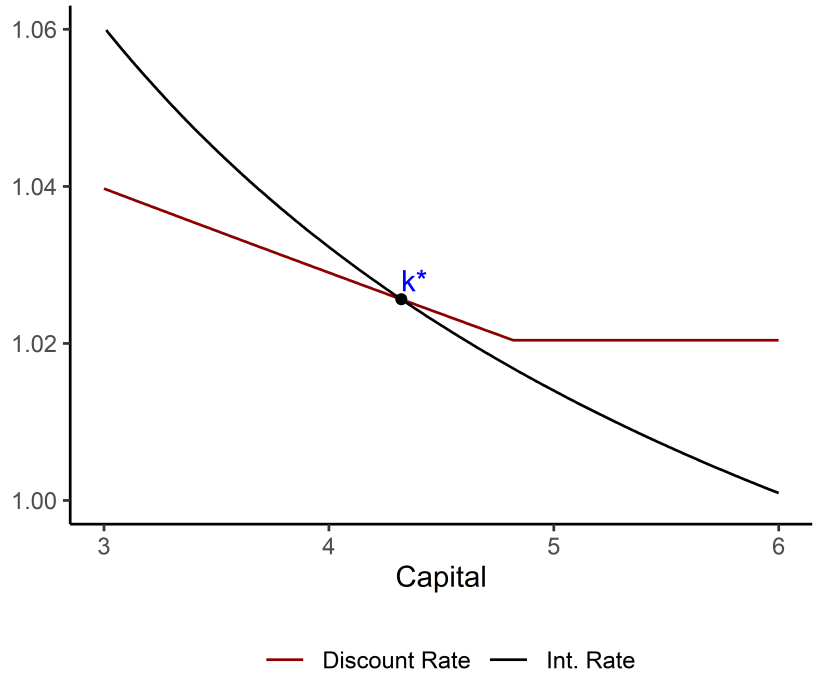
$$l(k_t) = max[0.02, 0.068 - 0.01k_t] \tag{9}$$

Figure 1 illustrates the steady state by plotting the LHS of (8) (labeled "Discount Rate") and the RHS (labeled "Int. Rate").

The local stability of the model is evaluated by linearizing (5), (6), and (7):

$$X_t = AX_{t-1} + e_t \tag{10}$$

Figure 1: Unique Steady State, $l(k) = max[0.02, 0.068 - 0.01k]$



where $X_t = [\tilde{c}_t, \tilde{k}_t, \tilde{\mu}_t]'$, with the *tildes* indicating the percentage deviations from the steady state for each variable. The model's reduced form shock, e_t, directly impacts consumption. Stability then depends on the eigenvalues of A. One is equal to ρ the AR(1) parameter for the mortality shock. If one other eigenvalue is outside the unit circle, then there is one saddle condition to pin down the model's only control variable, c_t. If, however, there are two such eigenvalues, then no solution exists and the steady state is unstable. Were all three eigenvalues inside the unit circle, then sunspot equilibria would exist. We do not encounter any such cases, however, when analyzing the model.

For all calibrations that we considered where there is exactly one steady state, that steady state is stable.

The most interesting aspect of the calibration with a unique steady state is to isolate the impact of endogenous mortality. To do so, we compare the model to another version where $l(k)$ is fixed at 0.025, which yields an identical steady state life expectancy of 40 years. The standard deviation of shocks to mortality is set to 0.005 and we set ρ, the AR(1) paramter for mortality shocks equal to 0.9. Table 1 compares the results; the volatilities of consumption and capital are reported as percentage deviations form the steady state while that of the mortality

rate is reported in absolute terms.

Table 1: Standard Deviations of Consumption, Capital, and mortality

	Endogenous Mortality	Exogenous Mortality
k	0.0353	0.0186
c	0.0133	0.0086
l(k)μ	0.0012	0.0005

Consider a shock that increases mortality. Agents now discount future periods more heavily and thus increase their current consumption. This results in a smaller future capital stock, which, with endogenous mortality, further increases the mortality rate. The mortality shock is thus amplified and the model exhibits more volatility for all three endogenous variables.

Case II: Multiple Steady States

We now consider a different mortality function that yields multiple steady states:

$$l(k) = max[0.02, .07 - .0024k^2] \tag{11}$$

The key feature of this mortality function is that, prior to reaching its biological minimum, mortality is more sensitive to changes in the capital stock than from the unique steady state case. This yields three distinct steady states as shown in Figure 2:

The local stability of each steady state is again evaluated using the eigenvalues of A from Equation (10). For this mortality function, the low and high capital steady states from Figure (2) are stable, while the intermediate steady state is not. Numerical simulations show, however, that this intermediate steady state is approximately at the boundary between the basins of attractions for the two stable steady states.[3]

[3]To assess the basins of attractions, we simulate the non-linear model by assuming that agents form expectations by fitting mortality and consumption to simple AR(1) processes using a rolling window of past observations. This is similar to constant-gain learning, a common approach to choosing among multiple equilibria. For details on constant-gain learning, see Evans and Honkapohja (2001).

Figure 2: Multiple, Stable Steady States, $l(k) = max[0.02, .07 - .0024k^2]$

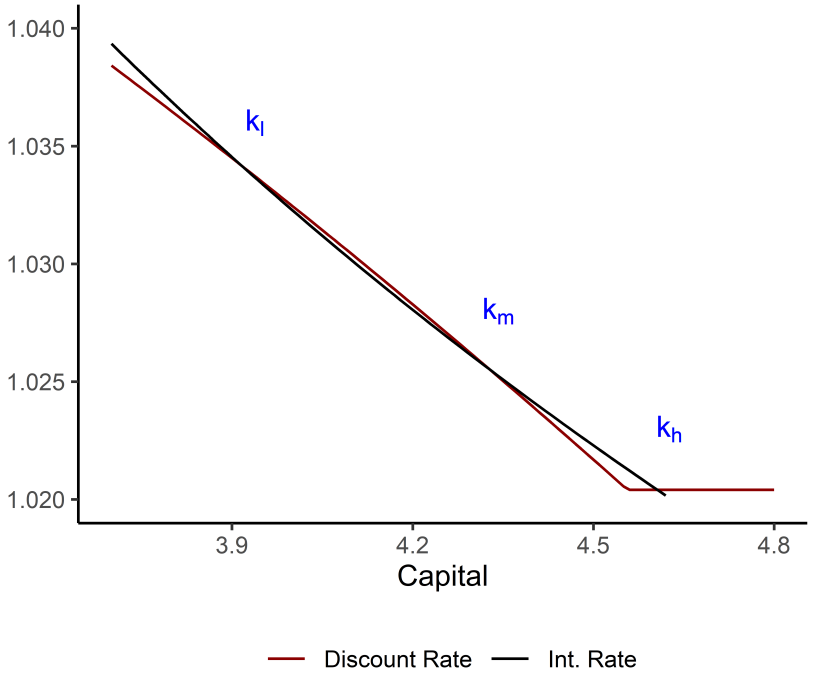


Table 2 reports the characteristics of each steady state from Figure 2.

Table 2: Steady State Properties

	k_l	k_m	k_h
k	3.918	4.322	4.606
l(k)	0.33	0.25	0.02
Life Exp.	30	40	50
Stable	Yes	No	Yes
c	1.185	1.197	1.203
r	3.41%	2.56%	2.04%

The high capital steady state corresponds to higher income, lower interest rates, and slightly higher consumption than the low capital steady state. Higher life expectancy (50 versus 30 years) ensures that the high capital steady state is preferable to the low capital steady state.

We now simulate a stochastic version of the model. We add a mortality shock to the model, although similar results can be be obtained through either a preference shock or a productivity shock:

$$\frac{1}{c_t} = E_t \left[\frac{(1 - l(k_t)\mu_t)(1 - \delta + \alpha k_t^{\alpha-1})}{c_{t+1}} \right] \tag{12}$$

Because numerical simulations show that the boundary between the basins of attraction is close to the medium capital steady state, we simulate the model by linearizing around both the low and high capital steady states. If the capital stock is less than that of the medium capital steady state, then we use the former linear approximation. If not, then we use the latter.

The key result is that the mortality shock can shift the model between steady states. A pandemic that increases mortality can increase capital per worker and shift the model from the low capital steady state to the high capital steady state. Likewise, a temporary shock that directly reduces mortality can lead to higher life expectancy indefinitely. The model thus provides a novel explanation for rapid reversals in longevity observed in the data.[4]

To simulate the model, we continue to set $\rho = 0.9$ and the standard deviation of shocks to mortality at 0.005. Larger values for either parameter cause more frequent shifts between steady states. Figure 3 shows a sample simulation including several switches. A series of sufficiently high mortality shocks can cause the model to switch from the high-capital and life expectancy steady state to the neighborhood of the steady state with low capital and life expectancy.

Figure 3: Transitions Between Steady States

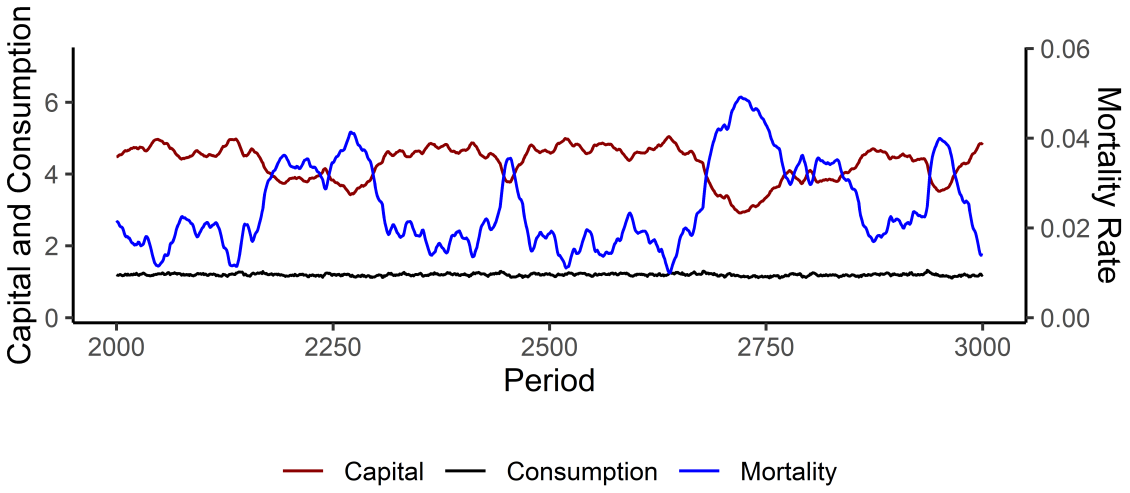


Table 3 reports the model's dynamics around each steady state.

For this calibration, the model switches between steady states on average every 219 years. In addition to yielding higher consumption and life expectancy, the high capital steady state is also far more stable than the low capital steady state. This is partly because around the high

[4]Bhattacharya and Qiao (2007) document these reversals. To explain them, they develop a model where households invest in private health which compliments public health expenditures. The model exhibits highly volatile and sometimes chaotic life expectancy.

Table 3: Behavior in Each Steady State

	k_l	k_h
Freq	0.484	0.516
stdev($\tilde{k}$)	0.0942	0.0376
stdev($\tilde{c}$)	0.0311	0.0238
stdev(mort)	0.0063	0.0035

capital steady state, the mortality function is flat, suppressing the added volatility brought about because of endogenous mortality.

In this model, an epidemiological or climate shock that increases mortality leads to a direct reduction in economic performance by making households less patient. This is in addition to any direct decline in productivity from such a shock, a feature that is not explicitly included in the model.[5] In addition, however, this type of shock, if sufficiently large or sustained, can also switch the model to the lower steady state, leading to an indefinite decline in economic performance even after the effects of the shock have fully worn off.

3 Backward Looking Shocks

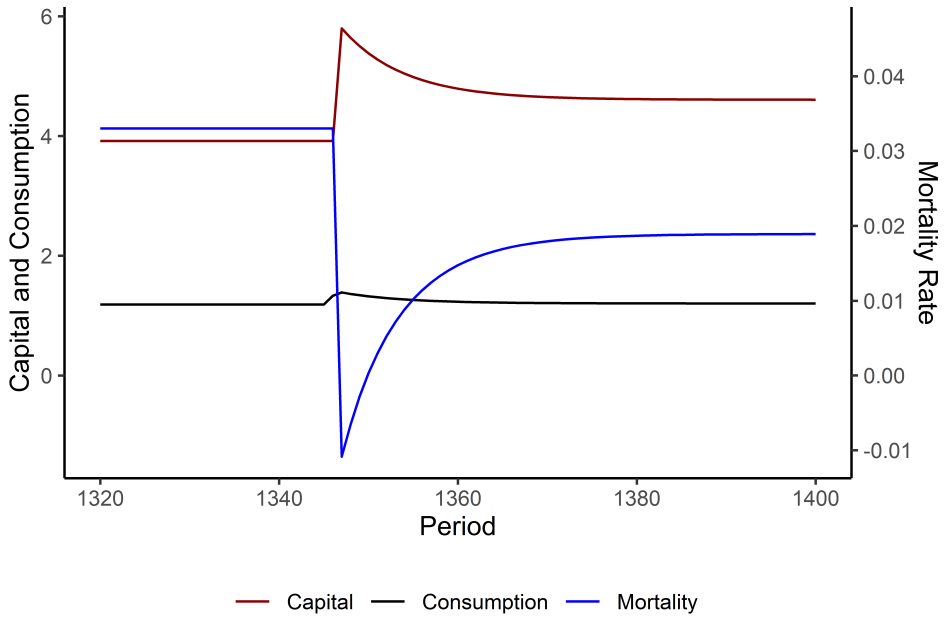
So far we have considered shocks which affect future mortality. Under rational expectations, households respond to higher mortality by more heavily discounting the future. Higher mortality thus deters capital accumulation and reduces wealth. We now consider a different type of shock: an unexpected, one time change to k either through a loss of capital or population. By construction, these shocks do not impact future mortality and therefore the discount rate. They do, however, impact the model in a different but straightforward way. By changing capital per worker, they can also result in a transition between steady states.

Figure 4 considers a scenario where the model is initially at the high mortality steady state and is then hit with an isolated shock that kills one-third of the population. The survivors are left with a higher capital stock per-worker and thus exhibit higher incomes and lower mortality. The impact of this shock then fades over time. The economy, however, converges to the low mortality steady state and workers thus experience indefinite improvements in income and life expectancy.

This example sheds light on the Black Death which reached Europe around 1346. The exact mortality rate of the plague has been the subject of intense debate, but one-third is well within

[5]Allowing such a shock to directly impact the productivity function does not affect our results.

Figure 4: Black Death Response Function



the range of existing estimates.[6] The plague did return in subsequent outbreaks for the next several centuries, allowing it to have some impact on the forward-looking mortality rate. But clearly its main impact was as an extraordinary abrupt decline in the population.

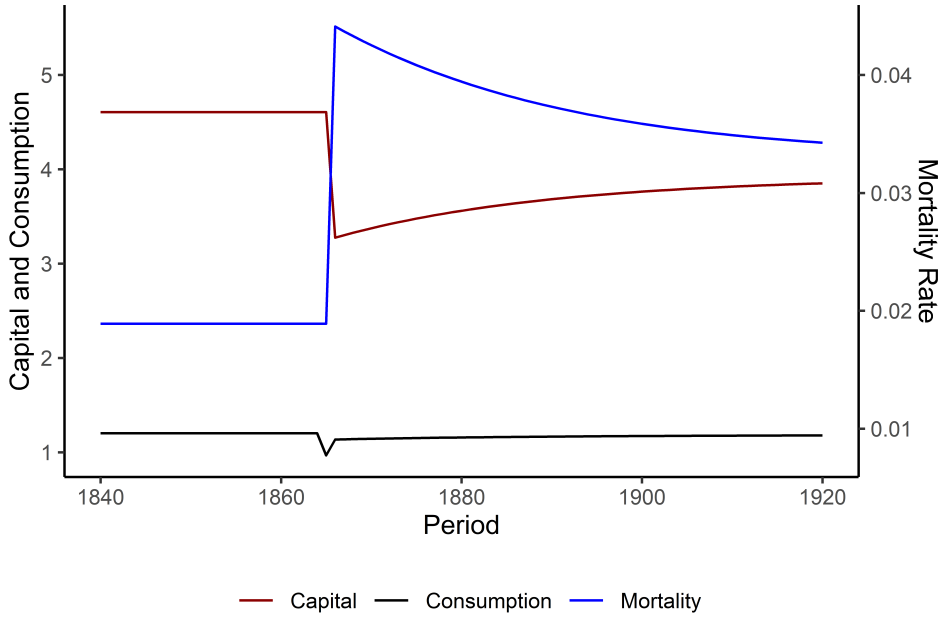
The improvement in living conditions after the plague in Europe is well-documented. Wages, especially among peasants, increased for centuries afterwards.[7]. DeWitte (2014) digs up some evidence from English cemeteries showing that, as predicted by the model, the population was healthier in the aftermath of the Black Death. Most interestingly, it fits with evidence that the Black Death may have led to permanent economic change in at least parts of Europe. Epstein (2000) argues that the Black Death, and not the industrial revolution, may have been the event that moved Europe to a higher growth path. Pamak (2007) argues that whereas northern and western Europe lagged southern Europe prior to the plague, the event may have reversed this, suggesting that northern and western Europe may have moved to the model's more prosperous steady state.

Events that reduce the capital stock relative to the population can have the opposite effect. Figure 5 illustrates a one-time, backwards looking shock which reduces capital per-worker by one-third. Now, the capital stock declines and mortality increases. As the model transitions

[6]See Megson (1998) for an examination of the evidence. She puts mortality at 35% in London.
[7]See Scheidel (2017) for a recent treatment

to its new steady state, these never fully recover from the shock. The most natural examples are wars that lead to more capital destruction than death. Feigenbaum, Lee, and Mezzanotti (2019) provide one such example, Sherman's March in 1864. They show that this event left caused parts of the U.S. South to remain relatively behind the rest of the country as late as the 1920s.

Figure 5: Sherman's March



The results of this section demonstrate that mortality shocks can also lead to beneficial economic changes if they are unexpected and do not affect upcoming mortality. Actual mortality shocks may, of course, have features of both types of shock. The Black Death, for example, not only killed much of the population unexpectedly, but may also have disincentivized capital accumulation if medieval household's feared it return. We leave simulations of such shocks to future research.

4 Conclusion

This paper introduces a new mechanism where pandemics and other mortality shocks may cause the economy to cycle between high and low output steady states. The model is, however, simple and does not include other important features of many growth models. It does not, for example, consider how this type of endogenous mortality might interact with households'

decisions on health and education spending. The model's steady states also differ in terms of output levels and the model does not include steady state growth, Including its mechanism in endogenous growth settings would be a worthwhile extension.

This paper's model also shares some similarities with Malthusian economics by connecting mortality to economic conditions. The model is not, however, Malthusian because the birth rate is assumed to make up for any morality shocks in order to keep the population constant. Future research will extend the model to allow for birth rates to endogenously respond to changes in consumption and mortality.

References

Bhattacharya, J., and X. Qiao. 2007. "Public and private expenditures on health in a growth model." *Journal of Economic Dynamics and Control*, Vol. 31(8): 2519-2535.

Blackburn, K. and G.P. Cipriani. 2002."A model of longevity, fertility and growth." *Journal of Economic Dynamics and Control*, Vol. 26: 187-204.

Castelló-Climent, A., and R. Doménech. 2008. "Human Capital Inequality, Life Expectancy and Economic Growth." *Economic Journal*, Vol. 118 (528): 653-677.

Chakraborty, S. 2004. "Endogenous lifetime and economic growth." *Journal of Economic Theory*, Vol. 116: 119-137.

Chetty, R., Stepner, M., and S. Abraham. 2016. "The Association Between Income and Life Expectancy in the United States, 2001-2014." *Journal of the American Medical Association*, Vol. 315(6): 1750-1766.

Clark, G. and G. Hamilton. 2006. "Survival of the Richest: The Malthusian Mechanism in Pre-Industrial England." *Journal of Economic History*, Vol. 66(3): 707-736.

DeWitte. 2014. "Mortality Risk and Survival in the Aftermath of the Medieval Black Death." *PLoS ONE 9(5)*, e96513.

Epstein, S.R. 2000. *Freedom and Growth: The Rise of States and Markets in Europe, 1300-1750.* London: Routledge.

Evans, G., and S. Honkapohja. *Learning and Expectations in Macroeconomics.* Princeton, New Jersey; Woodstock, Oxfordshire: Princeton University Press, 2001.

Evans, G., Honkapohja, S., and P. Romer. 1996. "Growth Cycles." *American Economic Review*, Vol. 88(3): 495-515.

Feigenbaum, J., Lee, J., and F. Mezzanotti. 2019. "Capital Destruction and Economic Growth: The Effects of Sherman's March, 1850-1920." *NBER Working Paper #25392.*

Comin, D. and M. Gertler. 2006. "Medium-Term Busienss Cycles." *American Economic Review*, Vol. 96(3): 523-551.

Gori, L., and M. Sodini. 2020. "Endogenous labour supply, endogenous lifetime and economic development." *Structural Change and Economic Dynamics*, Vol. 52: 238-259.

Megson, B. 1998. "Mortality among London Citizens in the Black Death." *Medieval Prosopography,* Vol. 19: 124-133.

Pamuk, S. 2007. "The Black Death and the Origins of the 'Great Divergence' Across Europe, 1300-1600." *European Review of Economic History,* Vol. 11(3) 289-317.

Scheidel, W. 2017. "The Great Leveler." *Princeton University Press,* Princeton, NJ.

Sharp, P., Strulik, H., and J. Weisdorf. 2012. "The Determinates of Income in a Malthusian Equilibrium." *Journal of Development Economics*, Vol. 97: 112-117.

Steinmann, G., Prskawetz, and G. Feichtinger. 1997. "A Model on the Escape from the Malthusian Trap." *Journal of Population Economics*, Vol. 11: 535-550.