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Offsetting Behavior and Medical Breakthroughs

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Offsetting Behavior and Medical Breakthroughs

Sam Peltzman

Abstract

Imagine that a medical breakthrough conquers a widespread health risk. The immediate effect is that many lives are saved. But the changed health risk also affects behavior. One effect is that the time, effort and other resources previously devoted to avoiding the conquered risk will now be freed for other activities, including those that have ancillary mortality risk. Thus some of the lives saved by the medical breakthrough will be lost to other risks.

This paper argues that such offsetting risk may be important empirically. It focuses on the history of a great medical breakthrough that nearly conquered infectious diseases – the development of antibiotics and other anti-infective drugs. This advance produced a greatly accelerated decline in mortality over a period from the late 1930s to the mid 1950s. But almost all of this trend-adjusted progress evaporated in the subsequent 10 to 15 years.

Cross-age group behavior is consistent with an offsetting-behavior interpretation of those aggregate trends. Age groups that benefited most from antibiotics show the worst subsequent deterioration in mortality. That deterioration is especially visible in mortality from accidents, suicides and homicides, where, arguably, mortality can respond promptly to behavioral change.

Offsetting Behavior and Medical Breakthroughs

Sam Peltzman

Medical research is sometimes viewed as a series of battles against specific maladies. Progress is often incremental but occasionally punctuated by breakthroughs that make substantial inroads on the enemy in a short period of time. Economists and others also understand that the enemy is not faceless: human behavior also affects health outcomes. Thus, holding access to medical technology constant, a better educated wealthier individual will tend to be healthier. So far, however, the interaction between the technology and the behavior has been mainly ignored.

That interaction is the subject of this paper. Specifically, I explore how medical progress, especially breakthroughs, might change the choices people make in ways that affect their health. The number of choice margins affected by a significant technical advance is too large to permit unambiguous predictions about this. Accordingly, I will focus on one specific margin – the allocation of risky activities. I will show that, even at this level of detail, theory can at best highlight opposing possibilities.

The paper is essentially an empirical exploration of one of the possibilities: that risk is reallocated in ways that blunt the health benefits of medical technology. I find that this is a possibility that deserves to be taken seriously. Much of the support for that finding comes from examining mortality data before, during and after one of the great medical breakthroughs of history: the discovery of anti-bacterial drugs. The data are crude, but they suggest a surprisingly large offsetting behavioral response to this innovation.

The next section outlines some theoretical considerations motivating a behavioral response to medical advances. It is followed by the analysis of mortality data over the 60 or so years from the beginning of the anti-bacterial drug revolution.

I. Theoretical Considerations

As far as I know, the only attempt to model a behavioral response to medical advance is in Dow, Philipson and Sala-I-Martin (DPS) (1999). It is useful here as a way of bracketing the possibilities. DPS focus on the sequential nature of health risks and the complementarities that arise because of this in risk-mitigation activities. Thus, while there are many potential causes of death, an individual can die only once. This elemental fact, according to DPS, leads the individual to invest most heavily in mitigating the most currently salient risk. For example, a young person might spend relatively little on mitigating the debilities of old age and more on exercise. If the individual survives, he or she will then shift resources to mitigating risks that become more prominent as people age.

Now suppose a medical advance reduces a mortality risk that is important to the young. They still face many kinds of mortality risk in the future. According to DPS, individuals will then shift resources no longer needed to mitigate the risk that has been reduced by technology toward coping with the myriad of remaining risks.

In fact DPS – as will I – couch everything in terms of individual choice between consumption and protective expenditures. And their comparative static exercise involves a price subsidy to the latter rather than a technological advance. But I follow them in adopting a broad view of the resources being allocated. Their model has a clear

implication: if one kind of mortality risk (to other than the oldest) is reduced, there will be a favorable effect on other kinds of mortality of risk.

I get the opposite implication by emphasizing a link between mortality risk and consumption choices (and by suppressing the sequential nature of health risk.) That is, mortality can be affected by the size and composition of the consumption bundle as well as by health spending. So all three are in principle chosen simultaneously. To illustrate how a link between mortality and consumption can act to blunt the impact of medical advance on mortality, I begin with the simplest case I can think of: the individual wants to maximize expected utility (E) over a single period. To obtain utility the individual has to survive the whole period. So I ignore any utility obtained or agony endured up to the date of death. Thus the maximand is just

$$(1) \quad E = \text{probability of surviving} \times \text{Utility, given survival.}$$

As in DPS, the probability of surviving is increased by devoting resources (S) to health and safety. But the resources (X) devoted to utility-producing consumption reduce the survival probability. Total resources (S+X) are fixed.

The composites, S and X, include specific goods and services. So X would include something like driving a car, which produces both utility and a safety risk. And S would include medical expenses, which are valued mainly because they prevent illness. But they include more: time and effort can be allocated to activities that entail more risk, or to mitigating risk; life-styles can be chosen to emphasize more or less risk; government policy can emphasize medical research or building roads; etc.

One way to grasp the breadth of the relevant tradeoff is to think of what would be in X if survival were certain. There would, of course, be more of it, since there would be

no need for medical expenses. But the composition and manner of consumption of X would also change. For example, food would be richer, exercise would be less common and traffic restraints would be unnecessary. We forgo some of these pleasures because survival is in fact uncertain. Accordingly, S should be thought of as including a monetary equivalent of the utility forgone to enhance survival.

With total resources fixed, the problem boils down to picking X to maximize

$$(2) E = F(X, S) \times U(x),$$

where,

F=survival probability,

U= utility function, and

$$F_x < 0, F_s > 0, U_x > 0.$$

The first-order condition for this problem can be written:

$$(3) [(F_x - F_s)/F] + U_x/U = 0,$$

where the percentage gain in utility from an extra X just balances the percentage loss in survival probability that arises both from consuming the extra X and from having less to spend on S.

Now, suppose there is a medical advance that, for simplicity, increases the level of F. The sign of the effect of this change on the equilibrium X is the opposite of the sign of the bracketed term in (3); that is X increases. The intuition here is straightforward: the advance has reduced the percentage survival loss from the marginal X and thereby induces a balancing reduction of the marginal utility of X. The increase in X needed to

achieve the new equilibrium will engender some reduction in survival probability that offsets part of the effect of the medical advance.

The only point of this exercise is to highlight one aspect of the response to medical advances – the inducement to (risky) utility-seeking that occurs because the advance substitutes for some of the individual's own risk-reducing expenditure. Accordingly, much is being left out here. For example, by analyzing a parametric increase in survival probability, I implicitly ignore the kind of complementarities emphasized by DPS. In terms of my set up, those would translate into medical advances that raised the marginal product of S . And such advances would, all else the same, induce substitution toward more, not less, private risk mitigation expenses.

Also, the model is too sketchy and general to use as a guide to magnitudes. For the specific case analyzed above, the model does imply that the behavioral response offsets no more than half the parametric change in survival probability. But that quantitative precision is easily lost when the model or the presumed nature of the medical advance is embellished.

Finally, since there is only one source of risk, the model cannot easily handle a medical breakthrough in a specific type of risk. To do that, I expand the model to include two sources of risk. Thus, suppose there are two activities that involve consuming X and Y respectively. As before, the activities produce utility but also entail survival risk. For relevance to subsequent empirical work, think of the X -activity as producing exposure to infectious diseases (before antibiotics were discovered), while the Y -activity engenders all other risks. For example, X could involve much social interaction, while driving a car in solitude would illustrate Y . Realistically, any consumption bundle would produce

some of both risks, but for simplicity, I treat X and Y as mutually exclusive, separable activities.

As before, safety expenditures can mitigate the two kinds of risks. But here I assume that each dollar of safety expenditures affect both kinds of survival risk. Some safety expenditures undoubtedly have this general effect. For example, acquiring good habits and knowledge about health can reduce the risk of both infectious diseases and heart attacks. However, the reason I do not assume two mutually exclusive kinds of safety expenditures is pedagogical. It is simpler and permits me to emphasize a specific tradeoff.

The two-risk version of equation (2) is

$$(4) E = F(X,S)G(Y,S)U(X,Y),$$

where F is now the probability of surviving infectious diseases, G is the probability of surviving other kinds of risk, and FG is the total probability of surviving and thereby obtaining utility. So the choice problem is now to pick X and Y and, implicitly, S to maximize expected utility.

The first-order conditions for this problem can be expressed as:

$$(5) \frac{F_S - F_X}{F} = \frac{U_X}{U} - \frac{G_S}{G}$$

and

$$(6) \frac{F_S}{F} = \frac{U_Y}{U} + \frac{G_Y - G_S}{G}.$$

As before, these come from balancing the favorable effects from spending more on S – the direct effect on survival and the indirect effect from spending less on X and Y – on

the loss in utility. The favorable marginal effects of S on surviving infectious diseases have been collected on the left-hand sides of (5) and (6).

The reason for doing this is to help us think about the effects of a medical breakthrough in treating infectious diseases. Specifically, imagine an innovation that very nearly eliminates that risk. It pushes F close to 1, and thereby causes the marginal consumption hazards and payoffs to precautions in $F(\cdot)$ to vanish. This is approximately what happened after antibiotics were discovered. Mathematically, the positive expressions on the left-hand sides of (5) and (6) would both go to zero. The kind of adjustment required to restore equilibrium would entail diverting resources away from S toward X and Y – to reduce the marginal consumption utilities and raise the remaining health risk.

It is this last adjustment that I will focus on. It is not mathematically required that Y increase or increase enough to matter empirically. And allowing for different types of safety expenditures would only heighten that ambiguity. But one of the incentives created by the breakthrough is to divert resources from precaution to utility enhancing risk taking. The next section shows why we may need to take that incentive seriously when thinking about the effects of medical breakthroughs.

II. The Anti-bacterial Drug Revolution and Mortality

Infectious diseases have been a major source of mortality for most of human history. At least in the developed world, these scourges have now receded into the background. Today only pneumonia and influenza remains as one of the 10 leading causes of death in the United States, and it is mainly an affliction of the elderly. The

contribution of medical technology to control of these diseases goes back at least 200 years, to Jenner's discovery of smallpox vaccine. But there was a major acceleration of progress in the two decades from the mid 1930s to the mid 1950s.

That period began with the commercial introduction of sulfa drugs, the first broadly effective anti-bacterial drug. This was followed by the introduction of penicillin and other antibiotics in the 1940s and 1950s as well as the development of effective anti-influenza vaccines in the mid 1940s. These had the effect of dramatically reducing mortality, especially premature mortality, from formerly dreaded diseases like tuberculosis, scarlet fever, syphilis, etc. These drugs also contributed to advances in surgery by controlling secondary infections.

Figure 1 summarizes this history. It shows the United States age-adjusted death rate¹ over the first half or so of the twentieth century. Prior to the advent of antibiotics² there had been a remarkably steady decline in mortality interrupted only by the influenza pandemic around 1918. The effect of antibiotics is evident in the lower panel of Figure 1. This shows actual mortality for 1940-1955 and a linear projection of the pre-1940 trend. There is a palpable acceleration of progress in this period: the rate of decline in mortality doubles – from around 1 per cent annually to around 2 per cent. By 1955 actual mortality is nearly 20 per cent below the extrapolated pre-1940 trend.

At this point, however, progress essentially stops. The next 15 years – from 1955 to 1970- are shown in the top panel of Figure 2. Here actual mortality is essentially trendless until the very end of the period. By the late 1960s it is almost as if the antibiotic revolution hadn't occurred, in that actual and extrapolated- pre-antibiotic era mortality nearly coincide. No previous 15-year period in the century (excluding the years of the

influenza epidemic) saw as little progress as this one. That sudden dramatic swing from above average to below-average progress is a clear motivation to this paper.

That aberrant period ends around 1970. As shown in the bottom panel of Figure 2, progress subsequently resumes. It does so at a rate slightly greater than the pre-antibiotic area. Accordingly, at the end of the century, most of the mid 1950s gap between actual mortality and the extrapolated pre-antibiotic-era mortality is restored.

A. Cross-Age Group Correlations

These aggregate trends may suggest an important role for risk-substitution. But to say more it is necessary to look behind the aggregates. Specifically, the antibiotic revolution had uneven effects across demographic groups. The most important difference was a greater relative impact on younger age groups than the elderly. In 1938 infectious diseases accounted for around 10 per cent of deaths of those over 45. But for those younger, 25 to 65 per cent (depending on the specific age group) of mortality came from these diseases. The leading cause of death for those aged 20 to 40 was tuberculosis, which accounted for over one sixth of all deaths in this age group. Antibiotics essentially wiped out that disease.

These differences in the impact of antibiotics imply different risk-responses, if risk substitution is important. For example, we might doubt the importance of risk-substitution if most of the post 1955 retrogression is due to worsening mortality among the elderly, because they were the least benefited by antibiotics. However, the experience across age groups – summarized in Table 1 and Figure 3 – tends to dispel such doubts.

Panel A. of the table describes the experience across age groups within various demographic categories in the periods that Figures 1 and 2 suggest are interesting – the

antibiotic ‘shock’ to mortality from the late 1930s to the mid 1950s, the ‘response’ (?) of little progress to the late 1960s and a resumption of progress thereafter. The data here are unweighted averages across the age groups conventionally used to summarize US vital statistics (<1, 1-4, then each 10 years beginning 5-14). There are 9 age groups in each sample.³

The main message of panel A. is that the trends evident in aggregate mortality are broadly similar across demographic groups and characterize most age groups. There is some variety – the shock is greater for females and non-whites, for example. But every group’s mortality improves, then falls back and finally, around 1970, resumes to some degree its pre-antibiotic progress. However, the variety in panel A is not suggestive of risk-substitution – e.g., there is no difference between male and female average responses that correspond to the differences in average shocks.

The more intriguing data are in panel B, which shows correlations between the antibiotic shock and subsequent behavior across the 9 age groups. The first three columns focus on behavior from 1955-68, or just after the shock had ended. The correlations are almost all negative, often significantly so in spite of the small sample. That is, age groups with the most favorable effects from antibiotics tended to have the most unfavorable experience subsequently.

Moreover, the relevant magnitudes are substantial. For example, the first entry in panel B (-.78) implies a response-shock elasticity only a little below -1 . This would suggest that most of any unusually favorable antibiotic shock is offset in the ensuing period. Figure 3 depicts this particular correlation, and it reveals that only 15-24 year olds depart substantially from the fitted relationship.

The other important regularity in panel B is the lower correlations in column (2) than in (1). For example, for males the two correlations differ by a factor of 2 (-0.73 v. -0.37). This says that the male response across ages is more highly correlated with the antibiotic shock for the whole population than with the antibiotic shock for males alone. The (correct) inference is that there is a negative partial correlation between the female shock and the male response. While the small samples warrant caution, the pattern seems too persistent to ignore.

That pattern either argues against a risk-substitution interpretation or embellishes it. If we think of gender/race groups as immutable states-of-nature, then males, for example, should not respond to the female shock. An alternative is that the female shock conveys information about consequences of a variety of life-style choices that are also available to men (and vice versa). For example, in the pre-1955 period 'female' connotes less 'blue-collar' type work than does 'male.' This alternative implies that a sub-group's response would be sensitive to other sub-group shocks, which is generally what we find in the first two columns of panel B, Table 1.

A similar argument applies to age groups. Responses to a medical breakthrough can entail life-style changes that do not affect mortality immediately. Column (3), panel B, table 1 tries to grapple with this possibility by averaging the antibiotic shock over three age groups. Specifically, this column uses an age group's 'average shock', which is just the average of the shock for that group and the two preceding.⁴ However, the correlations in column (3) are not distinguishable from those in (1).

Finally, column (4) of panel B, table 1 shows that the response patterns persist into the post-1970 period, when aggregate mortality resumes its progress. Thus, the

resumption of progress is more like (a series of) general advances that have roughly similar effects across age groups. The altered age structure of mortality progress evident in figure 3 persists to this day.

B. Are the Cross-Age Group Correlations Biased?

Any bias in the correlations in Table 1 would appear to be upward. To obtain the shock and response variables, I deduct a common counterfactual trend from two different trends. If everything but this common counterfactual were noise, the correlation between shock and response would be positive, not negative.

There is, however, a potential negative bias that arises from the history of mortality over the relevant period. The counterfactual I have used is the trend in total mortality up to around 1940. Importantly, in that period, progress in infectious disease mortality already exceeded that in other areas. Thus the advent of antibiotics did not initiate progress against infectious diseases. But it did very nearly eliminate them as a significant source of mortality. This means that groups for which infectious disease mortality is more important will tend to have absolutely larger counterfactual trends, shocks and responses, and this could induce a negative shock-response correlation that has nothing to do with behavior. The correlation would be rooted in the historical difference in the importance of infectious diseases plus the convergence of mortality rates after infectious diseases are conquered.⁵

This possibility is addressed in Table 2. It repeats parts of Table 1, but it eliminates infectious disease mortality trends from the post-shock counterfactual. That is, to estimate a group's response to the antibiotic shock, the counterfactual I use is the pre-antibiotic trend of non-infectious-disease mortality. Panel A of Table 2 shows that this

counterfactual is, as expected, smaller algebraically than the Table 1 counterpart, by around .5 per cent per year on average. (Put differently, post-1955 mortality looks .5 per cent less unfavorable in Table 2 than Table 1). However, crucially, panel B shows that the negative shock-response correlations are essentially unaffected by the changed counterfactual. Therefore something more than the potential bias from the pre-antibiotic progress against infectious diseases is driving those correlations. And we are absolved from having to choose between the alternative counterfactuals.⁶

C. What Kind of Behavior was Affected?

It is hazardous to attempt to delineate the response to a breakthrough like the discovery of antibiotics. While the model is couched in the familiar language of individual choice under full information, this is only a convenience. The affected decision margins could include such things as the politics of public funds for medical research, policies toward health education and public safety, etc. Even the individual choice margins are too numerous to catalog.

Moreover, nothing in the theory depends crucially on the chooser's knowledge of the underlying risks.⁷ What is crucial is that different utility-producing activities have different mortality risks.

These caveats understood, I attempt to be more specific in Table 3, which is based on a bifurcation of total mortality into external and non-external causes. External causes are accidents, suicides and homicides. Here, I am guessing that external causes are more immediately malleable than other types of mortality. For example, they do not require cumulative effects of changes in lifestyle.

The data in Table 3 seem consistent with that guess. The bifurcation adds some noise, and the small sample assures that none of the differences are significant statistically. Nevertheless, the immediate post 1955 response of external cause mortality seems more negatively correlated with the antibiotic shock than the non-external cause response. The response-shock elasticities implied by these correlations differ by factors of two or three. As shown in Figure 4, the post-1955 behavior of external cause mortality looks very much like that of total mortality in Figure 3.

Interestingly, the direction and magnitude of these differences reverse in the post-1968 period. So the data seem to be saying that external causes responded more reliably to the antibiotic shock first, then the non-external cause response ‘caught up.’ All of the relevant correlations, however, remain negative.

III. Conclusions

Many years ago (Peltzman, 1975) I argued that automobile accident mortality was affected by offsetting driver behavior following the mandatory installation of safety devices in cars. The data in this paper suggest that the perspective of that paper may have been excessively narrow. In addition to the ‘within-mortality’ kind of response emphasized in the 1975 paper, there appears to be a cross-mortality response that ought to be taken seriously. This is the tendency for a favorable shock in one type of mortality to induce offsetting increases in other kinds of mortality risk.

Economic theory cannot unambiguously imply such a cross-mortality response. It can only leave it as a logically coherent possibility. The reason for giving weight to the possibility is empirical. The data I have used are crude, unfashionably aggregate and

sparse. Nevertheless the important patterns in these data are all consistent with a sizeable offsetting response to the specific mortality shock studied here.

That shock remains as the greatest achievement of modern medicine. It laid the foundation for a research-intensive pharmaceutical industry and the ongoing pursuit of similar medical breakthroughs. A young child today has essentially no risk of dying from something like scarlet fever or of growing up only to die of tuberculosis before reaching the prime of life. But, the data suggest, the absence of these long-forgotten risks has also loosened constraints on behavior that carries other kinds of mortality risk

The existence of such offsetting behavior does not diminish the importance of this breakthrough. It may however shed new light on the nature of medical progress. The more durable reductions of mortality risk may come from the steady accumulation of small advances than from the occasional great triumph.

References

Dow, William; Philipson, Tomas and Sala-i-Martin, Xavier. “Longevity Complementarities Under Competing Risks.” *American Economic Review*, December 1999, 89(5), pp.1358-71.

Peltzman, Sam. “The Effects of Automobile Safety Regulation.” *Journal of Political Economy*, August 1975, 83(4), pp.677-725.

¹¹ This is the total death rate with age-weights fixed at 1940 values.

² I will use ‘antibiotics’ as shorthand for the acceleration of technological progress that was characteristic of the period in which antibiotics were introduced.

³ I exclude the 75 and older groups where behavioral effects on mortality are arguably small.

⁴ The exceptions are the 1-4 group, which is averaged only with the under 1 group, and the latter, which is left as is. Averaging over three groups is arbitrary, but reflects the thirty-year span of the data – someone who is, say, at the top of the 35-44 group in 1970 would have been at the bottom of the 15-24 group at the start of the shock period.

⁵ The possibility can be illustrated by a highly stylized example that captures the relevant history. Suppose we have two groups whose mortality we track over three equal periods: period 1 precedes a breakthrough, and is used to establish a counterfactual trend for all subsequent periods. In period 2 the breakthrough eliminates infectious disease mortality; this produces a shock that we estimate as the group’s period II total mortality trend less the period I counterfactual. Period 3 begins when infectious diseases are conquered, and we estimate the response as the period 3 trend minus the counterfactual.

The two groups differ only in the relative importance of infectious disease mortality (I) and non-infectious disease mortality (N) in total mortality (M=I+N) at t=0. For simplicity, assume that ½ of M is I for group A, while I=0 for group B. Further assume there is no medical progress in N in any period; the trend decline in N mortality is 0 in all periods. Thus for group B, the shock and response are both zero. Accordingly only group A is affected by medical progress.

The progress is entirely confined to I and takes the following form: in period I there is gradual progress that eliminates a fraction $k < 1/2$ of the initial I. The progress accelerates in II when the remaining $(1-k) > 1/2$ of I is eliminated. Thus group A has the following mortality history:

$$\text{Start of period 1: } M_0 = I_0 + N_0$$

$$\text{Start of period 2: } M_1 = I_0 - kI_0 + N_0 \quad (\rightarrow \text{ counterfactual trend} = -kI_0)$$

End of period 2: $M_2 = N_0$ (\rightarrow 'shock' = $-(I_0 - kI_0) - (-kI_0) = (2k - 1)I_0 < 0$)

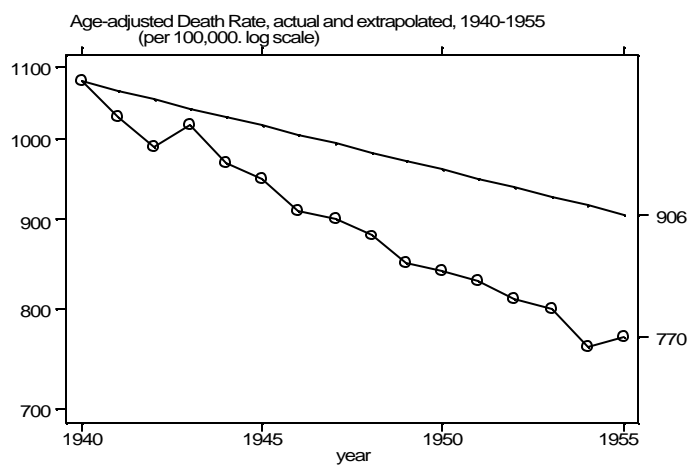
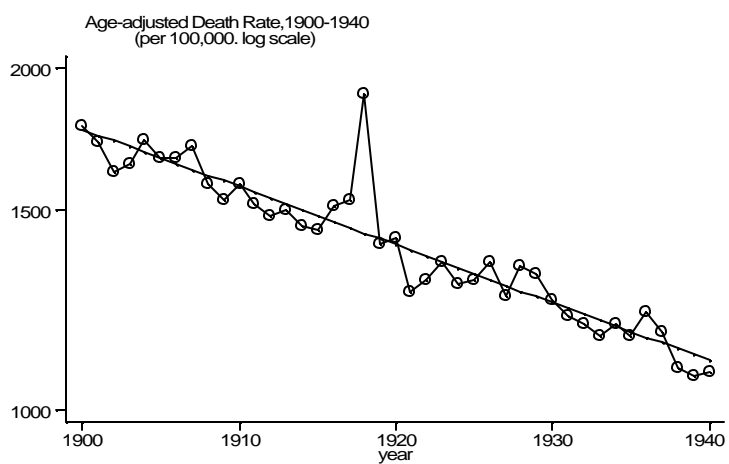
End of period 3: $M_3 = N_0$ (\rightarrow 'response' = $0 - (-kI_0) = kI_0 > 0$)

In this example, group A's (shock, response) pair is a point in the second quadrant; group B's pair is the origin and the correlation across groups is negative. There is, however, no behavioral significance to this correlation.

⁶ The ex-infectious disease counterfactual implicitly assumes that the discovery of antibiotics would have had no effect on non-infectious disease mortality. However, the model in Dow et al (1999) suggests that there would have been a favorable effect on such mortality. Recall that, in their story, once the infectious diseases have been wiped out, resources would be diverted to reducing other mortality risks. That kind of response implies that the ex-infectious disease counterfactual is too conservative and that the more comprehensive counterfactual used in Table 1 may be the more appropriate.

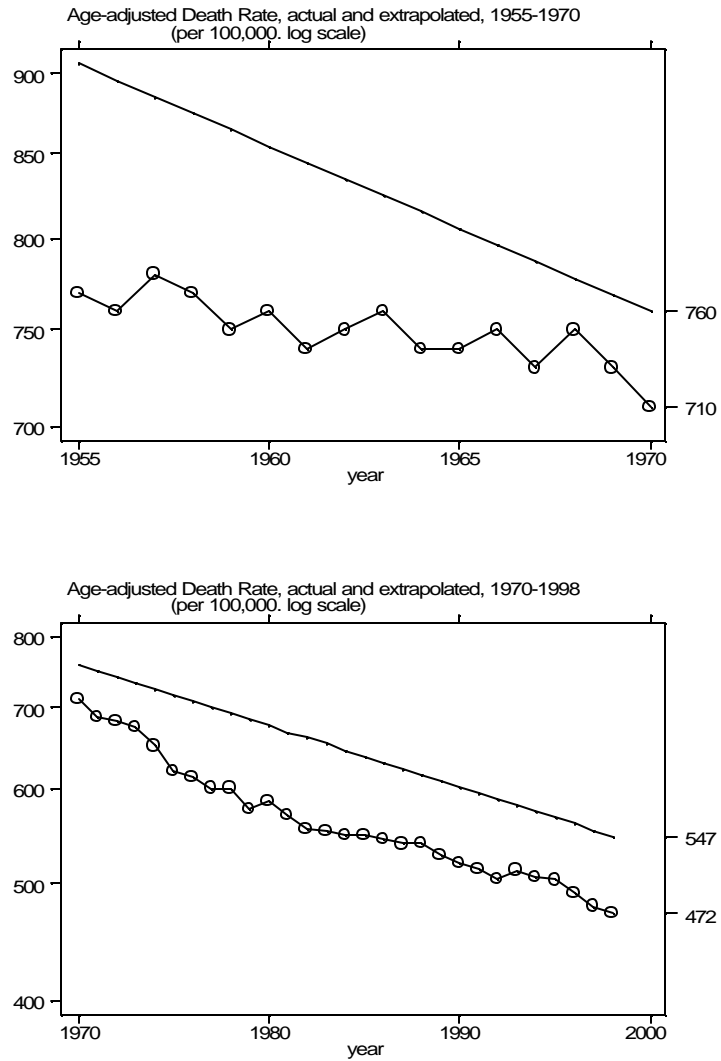
⁷ Specifically, the comparative static exercises in equations (4)–(6) can be re-worked with the $G(\cdot)$ function assumed, incorrectly, by the chooser to be a constant. The crucial result – a shift to activity Y (the one with non-infectious disease risk) – still goes through.

Fig.1 Mortality Before and After Antibiotics



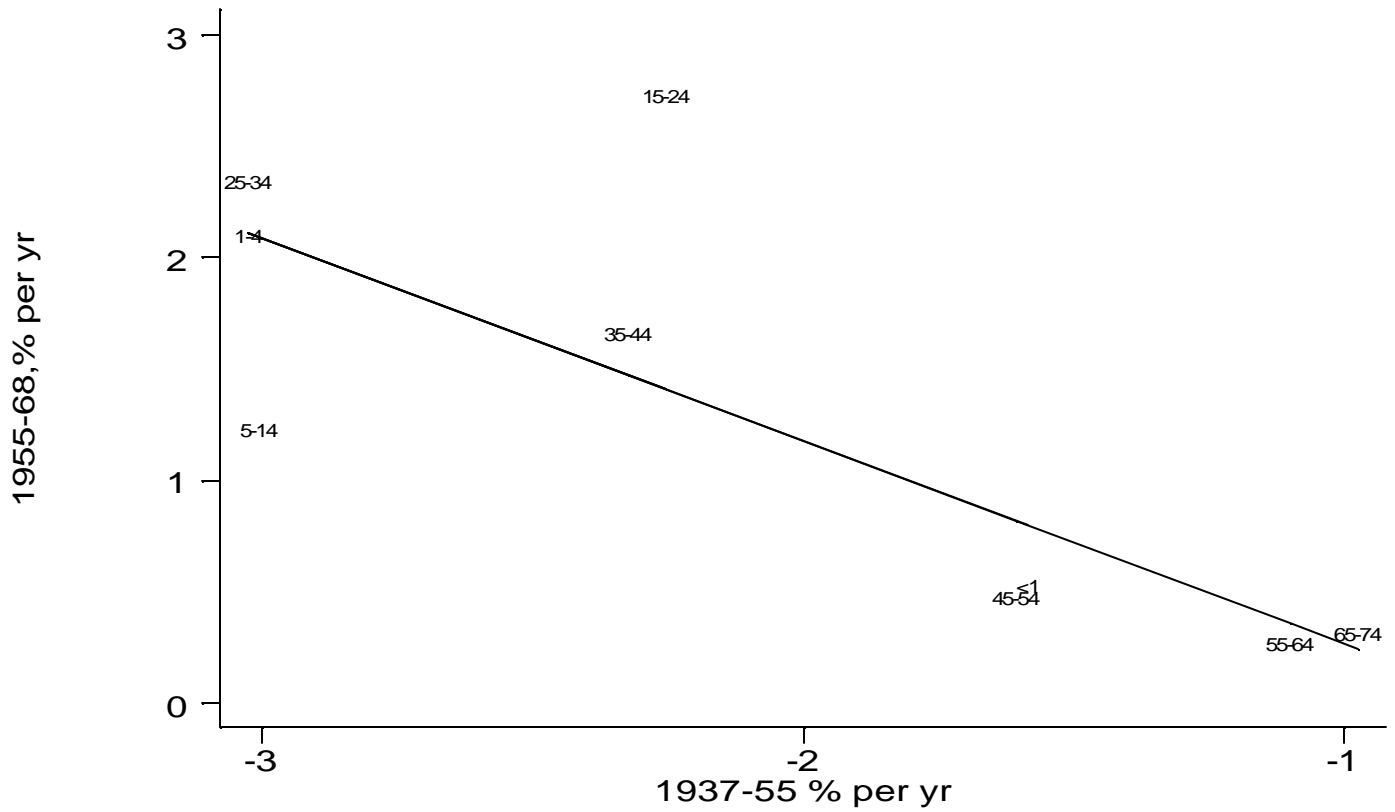
Note: Straight line in upper panel is fitted trend. Straight line in lower panel is projection of that fitted trend from 1940.

Fig.2 Mortality Before and After 1970



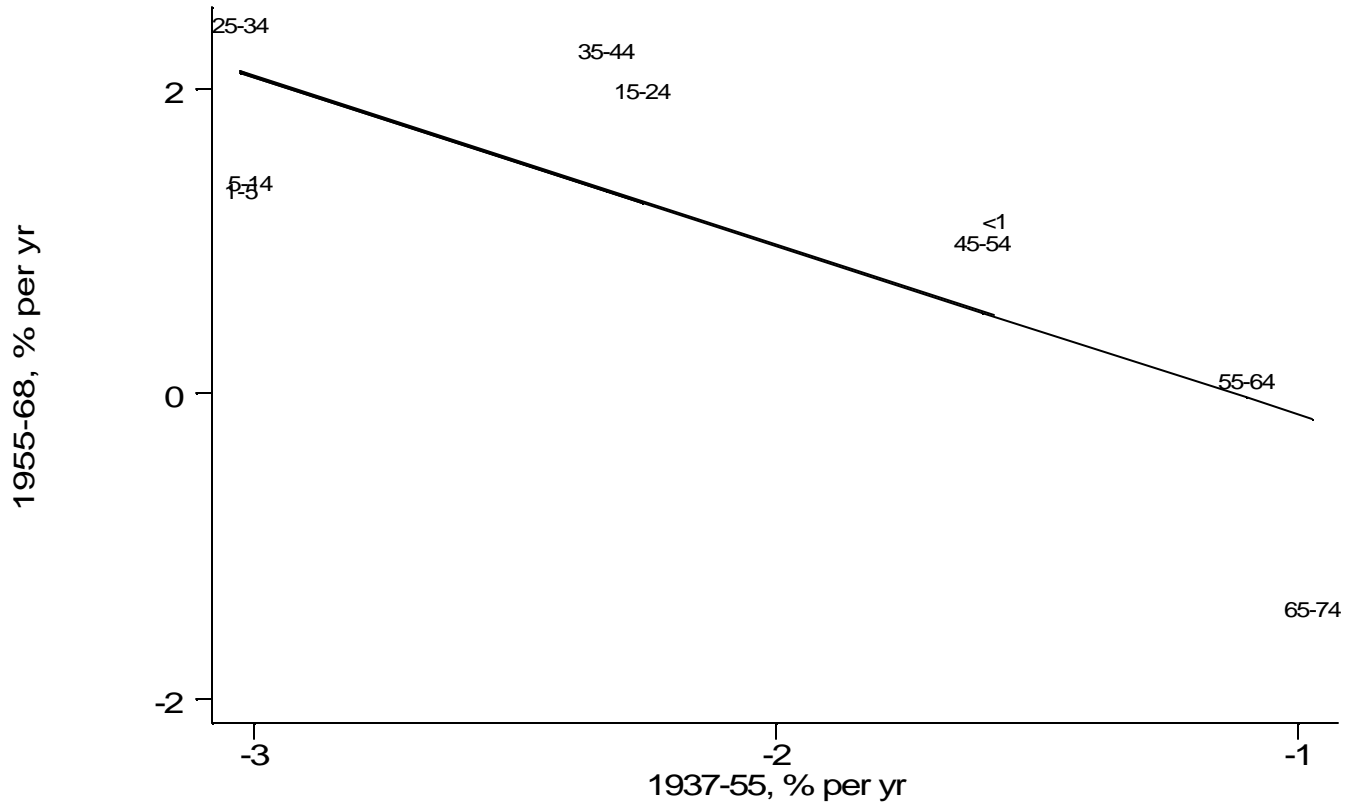
Note: Straight line in both panels is a continuation of the post-1940 projection of the fitted pre-1940 trend from the top panel of Figure 1.

Fig. 3. Mortality Trends By Age Group (Net of Counterfactual).
After v. Before Antibiotics



Note: y = annual % change mortality, 1955-68 minus annual % change 1902-37
 x = annual % change mortality, 1937-55 minus annual % change 1902-37.
 Solid line is fitted values from regressing y on x

Fig. 4. Response of Mortality from External Causes, 1955-68 to Antibiotic Shock, by Age Group



Note: y = annual % change mortality, 1955-68 minus annual % change, 1902-37, external causes only
 x = annual % change mortality, 1937-55 minus annual % change 1902-37., all causes
 Solid line shows fitted values from regressing y on x

Table 1. Response of Mortality to Antibiotic Shock Across Age Groups

A. Descriptive Statistics

Sex/Race Group	Antibiotic Shock*		Response**(1955-68)		Response 2 (1968-98)	
	Mean	Std Dev	Mean	Std Dev	Mean	Std Dev
Total	-2.10%	0.82%	1.26%	0.95%	-0.05%	1.09%
Male	-1.68	0.94	1.33	0.89	-0.20	1.26
Female	-2.82	0.99	1.00	0.90	0.13	0.97
White	-1.82	0.78	1.53	1.11	0.11	1.21
Non-white	-2.35	1.76	1.27	1.23	-0.51	1.53
White Male	-1.35	0.99	1.44	0.93	-0.02	1.49
White Female	-2.46	0.81	1.43	1.11	0.76	1.08
Non-white Male	-2.04	1.58	1.73	1.03	0.33	1.34
Non-white Female	-2.7	2.15	0.51	1.49	0.10	1.69

B. Correlation Coefficients of Response to Shock

Sex/Race Group	Correlation between group's response (1955-68) and			Correlation between Response 2 and
	Total Shock (1)	Own group Shock (2)	Avg. total shock (3)	Total Shock (4)
Total	-0.78	..	-0.81	-0.89
Male	-0.73	-0.37	-0.76	-0.88
Female	-0.82	-0.70	-0.80	-0.86
White	-0.83	-0.55	-0.88	-0.84
Non-white	-0.53	0.04	-0.16	-0.61
White Male	-0.75	-0.11	-0.81	-0.84
White Female	-0.81	-0.87	-0.92	-0.84
Non-white Male	-0.58	-0.22	-0.38	-0.66
Non-white Female	-0.30	0.39	0.19	-0.67

Note The means and correlations are based on 9 observations within each sex/race group. The observations are the cohorts aged: <1, 1-4, 5-14, 15-24, ..., 65-74.

For n= 9, correlation coefficients > .6 are significant at 5 per cent

*The 'antibiotic shock' is defined as the average annual change in log mortality from 1937 to 1955 minus the average annual change from 1902 to 1937 (x 100).

Total shock' is the shock estimate for the whole population in an age cohort. 'Own group shock' is estimated for the indicated sex-race group in that age cohort.

Average total shock' is the average of the total shocks for an age cohort and the two preceding cohorts (except:one preceding cohort for age1-4; none for age<1).

**'Response' is the average annual change of log mortality in the indicated period minus the 1902-37 change

Mortality rates for terminal years are averages of 3 or 4 years surrounding the indicated terminal year.

Table 2. Response to Antibiotic Shock. Alternative Counterfactual

A. Descriptive Statistics

Group	Antibiotic Shock		Response(1955-68)		Response 2 (1968-98)	
	Mean	Std Dev	Mean	Std Dev	Mean	Std Dev
Total	-2.10%	0.82%	0.74%	0.78%	-0.58%	0.97%
Male	-1.68	0.94	0.75	0.73	-0.79	1.09
Female	-2.82	0.99	0.56	0.89	-0.30	1.13

B. Correlation Coefficients of Response to Shock

Group	Correlation between group's response (1955-68) and			Correlation between Response 2 and
	Total Shock (1)	Own group Shock (2)	Avg. total shock (3)	Total Shock (4)
Total	-0.85	..	-0.71	-0.91
Male	-0.68	-0.39	-0.58	-0.88
Female	-0.86	-0.50	-0.65	-0.76

See *note* to table 1 for definitions of shock, response and response 2 and for description of samples.

The values for the Antibiotic Shock here are the same as for Table 1 - i.e. they are the 1937 to 55 annual change in log mortality less the annual change from 1902 to 1937. The values of the Response variables are post 1955 trends less a counterfactual that removes infectious disease mortality from the 1902 to 1937 mortality trend

Data for race and race/gender are unavailable, because cause-specific mortality is unavailable by race for the early years of the 20th century

Table 3. Antibiotic Shock and Mortality Response for External and Non-external Causes. Correlations and Elasticities

<i>Population group and Mortality Response by Cause</i>	Correlation [elasticity] of Response and Shock in	
	1955-68	1968-96
<i>a. Total Population</i>		
All Causes	-.78	-.89
External causes	-.76 [1.11]	-.52 [0.50]
Non-external causes	-.50 [0.32]	-.90 [1.23]
<i>b. Males</i>		
All Causes	-.73	-.88
External causes	-.83 [0.99]	-.19 [0.18]
Non-external causes	-.32 [0.23]	-.87 [1.70]
<i>c. Females</i>		
All Causes	-.72	-.86
External causes	-.56 [1.05]	-.48 [0.39]
Non-external causes	-.46 [0.36]	-.89 [1.04]

See *notes* to tables 1 and 2 for general definitions of shock, response and response 2 and for description of samples.

The values for the Antibiotic Shock here are the same as for Tables 1 and 2 - i.e. the 1937 to 55 annual change in log mortality less the annual change from 1902 to 1937. The shock for the total population is used throughout.

The response variables are based on a bifurcation of mortality into external and non-external causes. External causes are accidents, suicides and homicides. As with tables 1 and 2, each response variable equals a trend over some period after 1955 less a counterfactual trend. The counterfactual is the 1902-37 trend in the group's mortality from the indicated cause.

Values in [] are absolute values of slope coefficients from the simple regression of the indicated response on the antibiotic shock.

The correlations for 'All Causes' just repeat, for convenience, results from Table 1.

