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C E N D E S

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DOES UNEMPLOYMENT CAUSE THE DEATH RATE PEAK IN EACH BUSINESS CYCLE?
A MULTIFACTOR MODEL OF DEATH RATE CHANGE

Joseph Eyer

Natural time series and prospective studies are combined to determine the contribution of many causal factors to the business cycle variation of the death rate. The variation of housing and nutrition together accounts for roughly a tenth of the death rate fluctuation. Drug consumption accounts for about one-sixth, with 11 percent of the total variation due to alcohol and 6 percent due to cigarette smoking. Social relationship changes, both as sources of stress and as means of relief, account for the greatest part (72 percent) of the business cycle variation of the death rate.

INTRODUCTION

As I demonstrated in a previous paper (1), the death rate rises during business booms and declines during business depressions (Figure 1) (2-7). The largest, most sustained declines in the death rate occur as unemployment is *rising*: 1872-1878, 1881-1886, 1892-1897, 1908-1915, 1919-1921, 1929-1933, 1936-1938, 1943-1949, and 1968-1975. The cyclical peak of the death rate comes at the low point of unemployment, and the high point of industrial production in the cycle.

In the previous paper I sketched out the broad argument that sources of social stress which rise with the boom are responsible for the business cycle peak of the death rate. The net impact of these sources of stress during booms is greater than that of health risks which increase during depressions, resulting in the death rate peak with the boom.

In this paper I would like to examine the complex of causative factors in the business cycle variation of the death rate in greater depth. Starting with an evaluation of the role of unemployment, I will consider each of the possible factors in turn, using evidence from experimental, prospective, and other epidemiological studies, as well as the time series correlation of each factor with the death rate, to estimate the quantitative contribution of each to the total variation.

Though statistical correlations between natural time series are suggestive of causal relationships, they never suffice to establish the existence or actual pattern of causality (8). The actual relation of causal forces can only be deduced from experiments controlling all the relevant variables. In work on human health and disease, controlled

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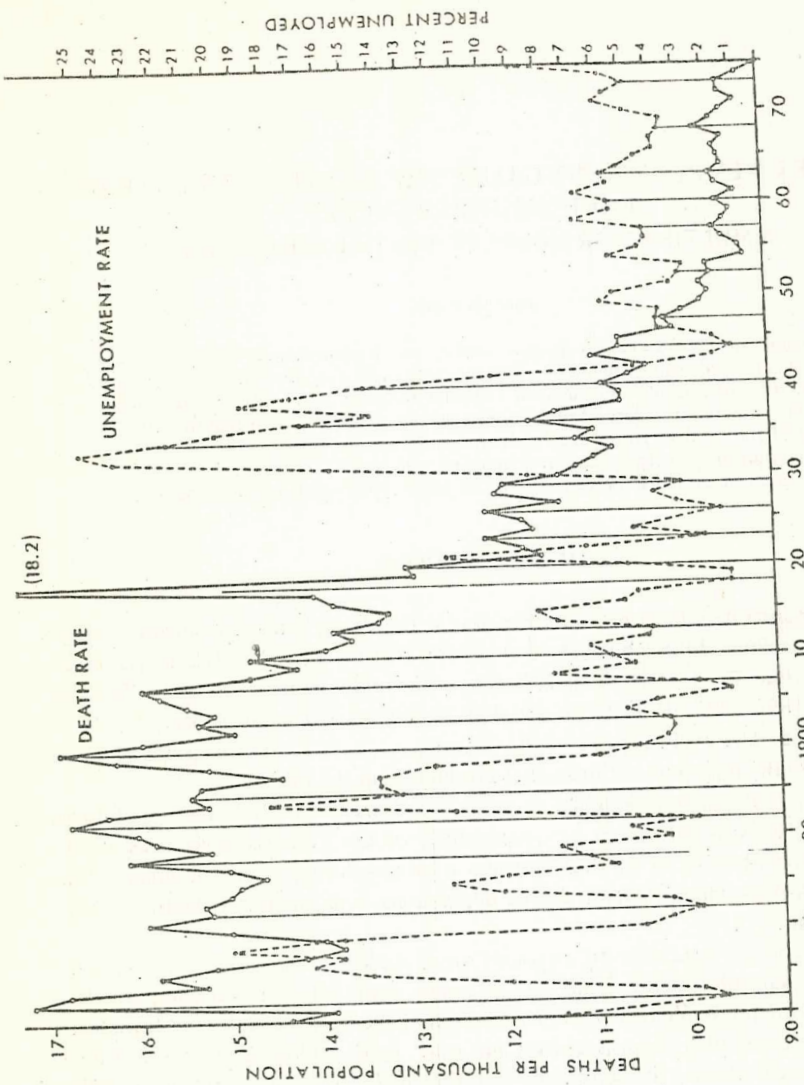

Figure 1. Total death rate and unemployment rate in the United States, 1870-1975. Sources for unemployment rate by year: 1965-1975 (2); 1890-1965 (3); 1870-1890, estimated from a regression of the unemployment rate for 1890-1965 on industrial production, deviations from trend, for the same period (3, series A-15 and A-16). Sources for death rate by year: 1970-1975 (4); 1953-1969 (5); 1910-1953 (6); 1870-1910, death rate data for all states with reliable registration systems: Conn., Mass., Mich., N.H., R.I., Vt. (7). *Vertical lines* mark the peaks of the death rate.

experiments are difficult to do, and prospective epidemiological studies to some exte take their place. If the data from time series correlations are consistent with the resu of prospective or experimental studies, the two sources are mutually reenforcing their estimates of causality. But if the time series correlations disagree with t prospective and experimental studies, it is the latter which establish the necessa interpretation of the former. This is the method I will use in this paper.

ESTIMATING THE IMPACT OF UNEMPLOYMENT

Brenner (9-12) has pursued the hypothesis that the rise of unemployment duri depressions, both by depriving a part of the population of income, and by subjecti the population to various sources of stress, causes the greatest part of the busine cycle variation in the death rate. His method is to describe the death rate peak at t business boom as a lagged effect of the preceding unemployment peak, by generati the appropriate correlation coefficients for many different categories of the basic dat Since it is confined to the correlation analysis of natural time series only, Brenne work entails two important questions. Does unemployment have a large enough effe in controlled studies, to account for the observed death rate variation? Second, do this effect occur predominantly with a 2- to 3-year lag, that is, the average tim interval between the unemployment peak and the business boom in a typical cycl I will address each of these questions in turn.

The Impact of Unemployment in Prospective Studies

There are many cross-sectional comparisons which suggest that unemployment low income have very large effects on the death rate. Figure 2 shows the death rate f males aged 35-64 in the United States in each occupation, along with the unemplo ment rate for each occupation, in 1960 (13, 14). If the entire differential in the dea rate is supposed to be due to unemployment effects, then a 2.5 percent increase the unemployment rate (the average for recessions, 1949-1975) should produce 24 percent increase in the death rate.[1] This increase is nearly five times as large as t actual average business cycle variation of the death rate (5.3 percent, 1949-1975

This estimate of impact would be smaller if we assumed that the pathologic effects of unemployment are smaller for children, old people, and women not in t labor force, than for adult men. But even if we assume that unemployment chang have *no* impact on the part of the population not in the labor force, the typic business cycle increase in the unemployment rate should generate about a 10 perce increase in the total population death rate.

Clearly, the death rate differential by occupation cannot be due to the effect unemployment alone. This is suggested by direct examination of Figure 2, since t majority of the workers are in occupations with closely similar unemployment rate but widely varying death rates (e.g. *A, P, M, C,* and *CS*). This chain of reasoning is on another way of emphasizing that simple one-factor cross sections are inadequate

[1] See Appendix 1.

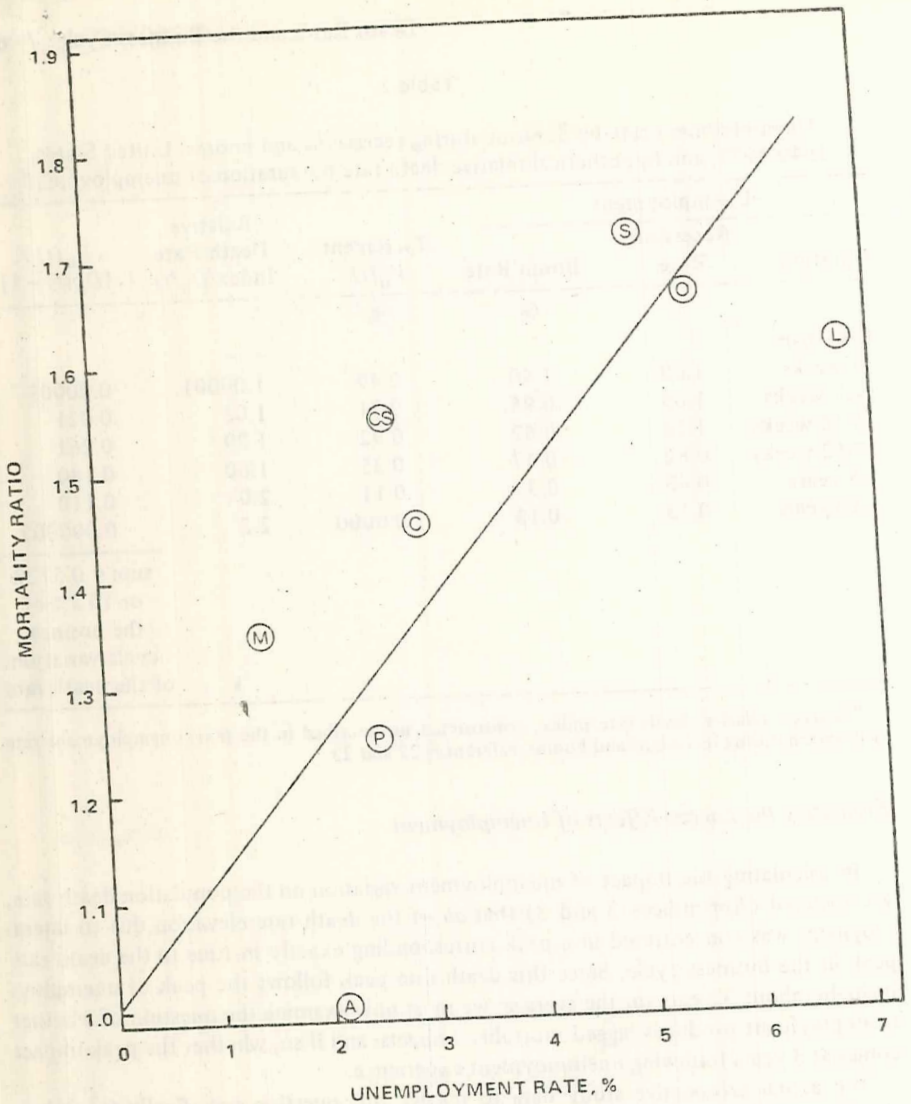

Figure 2. Relative death rate for males aged 35-64 by occupation and unemployment rate by occupation, United States, 1960. *A* = agricultural workers [farmers]; *P* = professional, technical, etc.; *M* = managers, officials, and proprietors (ex. farm); *C* = craftsmen, foremen, etc.; *CS* = clerical and sales workers; *L* = laborers (ex. farm and mine); *O* = operatives and kindred workers [factory workers]; *S* = service workers (inc. private household). Source for death rate by occupation, reference 13; for unemployment rate by occupation, reference 14.

indicate the impact of any factor on the death rate, since they are not controlled for other possible causes of death rate variation.

The independent effect of unemployment alone on the death rate can only be estimated from prospective controlled studies. There are only two such studies;

together, they barely suffice to make a first-order estimate of the effect of unemployment alone.

In Kasl and Cobb's studies of a plant closing (15-20), workers unemployed in one plant as it closed were compared to other workers, matched on many variables, who were continuously employed. The time period of observation and the sample sizes were not large enough to obtain meaningful mortality data, but significant changes in mortality risk factors, such as blood pressure, serum cholesterol and uric acid, and other measures, were demonstrated in the unemployed men. For instance, blood pressure appears to have risen about 5 mm Hg in the unemployed group during the initial period of unemployment.

These data may be translated into a probable mortality impact by use of the equations summarizing the independent impact of each such factor on cardiovascular mortality in the Framingham study (21). For instance, a 5 mm Hg elevation in blood pressure above 140 mm systolic (the average blood pressure for people aged 30-60), sustained for 11 weeks (the average duration of unemployment, 1949-1975), translates into a 0.0027 percent increase in the subsequent total death rate.[2] If all of the other demonstrated risk factor increments in Kasl and Cobb's studies are similarly treated as independent factors, and the total summed, the overall probable impact of the average duration of unemployment on total subsequent death risk may be a 0.013 percent increase.

The second study is Kitagawa and Hauser's examination (13) of a representative sample of death certificates for white males aged 35-64 in the United States in 1960, versus the corresponding Census information for these individuals in 1950 and 1960. While in fact a retrospective study, this examination approaches a prospective design because of the very nearly complete cross-referencing of information for both periods. In addition, the representative sample randomly chosen, and the large sample size, make this study particularly interesting for a determination of the impact of unemployment on the death rate. The relevant data are summarized in Table 1 (13).

Men unemployed for the whole 10 years between 1950 and 1960 had about a threefold elevation of adult age-specific death rates compared to farmers, the lowest mortality group, and a twofold elevation of death rates when compared to the majority of other urban workers (Table 1).

Neither of these two studies alone allows us to calculate the typical effect of unemployment on the ordinary business cycle variation of the death rate, since Kitagawa and Hauser's study provides no information on the specific effect of unemployment for periods shorter than 10 years, while Kasl and Cobb's studies provide no information on periods longer than several months. If we *assume* that the death risk associated with unemployment increases rapidly from a few weeks to a year, but increases more slowly after that, as shown in Table 2 (22, 23), we can calculate that the typical change in unemployment by duration during business recessions might account for 10.7 percent of the business cycle variation of the death rate.[3]

This estimate is smaller than the one from the simple cross section principally because the proportion of the population unemployed for very long periods of time

[2] See Appendix 2.

[3] See Appendix 3.

Table 1

Mortality ratios by occupation for white males aged 35-64, United States, 1960[a]

Occupation	Mortality Ratio (Agricultural Workers Equal 1.00)	Percent Contribution of This Group to All Excess over Agricultural Workers
All males	1.48	(100)
Agricultural workers [farmers]	1.00	–
Professional, technical, etc.	1.24	5.6
Managers, officials, and proprietors (ex. farm)	1.37	9.5
Craftsmen, foremen, etc.	1.47	21.8
Clerical and sales workers	1.57	15.7
Laborers (ex. farm and mine)	1.60	6.8
Operatives and kindred workers [factory workers]	1.64	26.2
Service workers (incl. private household)	1.71	7.2
Occupation not reported	1.67	5.6
Unemployed since 1950	2.91	5.2

[a]Source, reference 13, Tables 3.1 and 3.2.

is small, and the business cycle *variation* in this proportion is tiny, as shown in Table 2. The overwhelming majority of the variation in unemployment occurs at short periods of exposure, for which the death risk increment is likely to be small.

Yet this last estimate may also be an overstatement of the effect of unemployment on the variation of the death rate, because the excess mortality experienced by men unemployed for the decade between 1950 and 1960 may not be due to unemployment alone. As Kitagawa and Hauser put it, "Mortality ratios for those with no work experience since 1950 are substantially higher than those with work experience. This is not unexpected, because the former group includes persons not employable for health reasons" (13).

The high excess mortality among the very long term unemployed may reflect primarily the disabling effects of previous work and community experience, disabling effects which also produce unemployment. This interpretation is consistent with the fact that any cross section comparing employed workers of any kind with men or women at home shows the employed people healthier, despite identifiable occupational hazards (the "healthy worker effect"). It is only by doing a prospective study that it can be shown that this effect is due to the early retirement or rejection of workers made sick or disabled by production experience (24). This interpretation is also supported by the fact that the proportion of men in Kitagawa and Hauser's representative sample unemployed for 10 years increased sharply toward older ages, and that the majority of these people were old. In contrast, the highest unemployment rates, and the great cyclical variation of unemployment, occur at ages 15-24 (22).

Table 2

Unemployment rate by duration during recessions and booms, United States, 1949-1975, and hypothetical relative death rate by duration of unemployment[a]

Duration	Unemployment: Recession Rate	Unemployment: Boom Rate	Increment $V_u(t)$	Relative Death Rate Index $D_u(t)$	$V_u(t) \times [D_u(t) - 1]$
	%	%	%		
Less than 5 weeks	1.89	1.40	0.49	1.00001	0.00005
5-14 weeks	1.65	0.95	0.71	1.03	0.021
15-26 weeks	1.54	0.62	0.92	1.29	0.261
27-52 weeks	0.82	0.47	0.35	1.60	0.180
1-5 years	0.48	0.37	0.11	2.0	0.110
5-10 years	0.13	0.13	0.0000	2.5	0.000003
					sum = 0.572% or 10.7% of the business cycle variation of the death rate

[a]Sources, relative death rate index, constructed as described in the text; unemployment rate by duration during recessions and booms, references 22 and 23.

Evaluating the Lagged Effects of Unemployment

In calculating the impact of unemployment variation on the population death rate, we assumed (Appendices 2 and 3) that *all* of the death rate elevation due to unemployment was concentrated in a peak corresponding exactly in time to the death rate peak in the business cycle. Since this death rate peak follows the peak of unemployment by about 3 years on the average, we must now examine the question of whether unemployment produces lagged mortality impacts, and if so, whether the peak impact comes at 3 years following unemployment experience.

The actual prospective study data to resolve this question specifically for unemployment are even more scanty than in the previous section. It is interesting to note, however, that in Kasl and Cobb's studies, the elevation of mortality risk factors is often *anticipatory* of unemployment, rather than lagged. Blood pressure, for instance, seems to have risen at the announcement of plant closing, some weeks before the actual experience of unemployment, and blood pressure fell off for unemployed workers during unemployment, though it did not reach preclosing values until the workers were integrated in new jobs.

Though this is the only study which evaluates the possible lagged impact of unemployment per se, unemployment via loss of income and changes in self-image and social status (25) is linked also to increases in divorce and separation (26), child beating (27), and other phenomena (28), which are themselves sources of stress. Though all of these ramifications are comprehended by the design of Kasl and Cobb's

studies, their existence allows us to widen the investigation of lags between life changes and health changes somewhat. This broadening confirms the suggestive results of Kasl and Cobb, that the lags between life events and illness are on the order of weeks and months, not several years.

Table 3 summarizes the relevant studies (16, 18, 29-41). It is plain that for a wide range of diseases, from cardiovascular through infectious, the lags between life events and illness are short. The longest is between 7 and 12 months, and most are on the order of a week or so. These results are specially important because the studies simply describe the overall lag between the given life experience and the peak probability of pathology; the result is not dependent on a detailed understanding of either the social ramifications of the event, the individual psychological and physiological reaction, or the pathological process itself within the body. All of these must eventually be understood in all of their complex integration, but these studies provide us a summary overview that will remain unchanged by the development of the detail.

It is clear that the lagged pathological impact of life events is an order of magnitude shorter than the lag required to explain the major peak of the death rate with business booms. Therefore, unemployment cannot account for as much as 10.7 percent of the variation of the death rate, our previous maximal estimate, and probably accounts for much less.

Though the lag is not explicitly known for unemployment, it is probably short. If we assume that the lag between peak unemployment and peak pathology is less

Table 3

Lags between stressful experience and pathology in prospective studies

Pathology	Situation	Lag	Sources
Colds, flu	Privates at Fort Dix: military stresses	3 days	29
Upper respiratory, gastrointestinal, and other infectious diseases	Migration	0-3 months	30
Ulcers, stomach spasms	Military stresses	7-14 days	29
Blood pressure elevation	Unemployment	within days of announcement of plant closing	16, 18
	Normal work pressures	minutes	31
Blood cholesterol elevation	Tax deadlines for tax accountants	less than one week	32
Ventricular premature beats	In daily routine	minutes	33
	Experimental stresses	minutes	34-36
Heart attack	Multiple ordinary stresses	0-6 months	37-39
		7-12 months	40
	Bereavement	0-6 months	41

than 6 months, then unemployment variation can only account for 1.07 percent of the business cycle variation of the death rate.[4]

Another method of analyzing lags points to a similar small contribution of unemployment change to death rate variations. As I demonstrated in my previous paper, the overwhelming majority of the variation of the death rate with the business cycle is produced by causes of death which peak at the minimum of unemployment and the peak of industrial production. Figure 3 shows the nine leading causes of death in the United States, 1950-1975, which together comprise 76 percent of the total death rate and 81 percent of the total short-cycle variation (4, 5). Many other smaller causes, such as ulcers, show the same peak with the boom, and altogether these causes sum to 95 percent of the variation of the death rate. The remaining 5 percent of the variation is due to causes, such as suicide (Figure 4) and to some extent homicide, which typically peak within weeks of the peak of the unemployment rate in the business cycle (4, 5, 42-47).

Since it is probable that the lagged impact of unemployment does not come more than half a year after the unemployment rate peak, this 5 percent is also a possible estimate of the actual impact of unemployment variation on the business cycle fluctuation of the death rate. However, this estimate attributes the *entire* rise and fall of suicide and homicide to unemployment impacts, though both of these causes rise significantly during the boom as unemployment is still falling, prior to the recession. Therefore, this may also be an overestimate of the impact of unemployment.

In summary, the most likely quantitative estimate of the contribution of unemployment variation to the business cycle variation of the death rate lies somewhere between 1 and 5 percent. Clearly, unemployment is a minor factor in producing the death rate variation.

A reexamination of Table 1 suggests where to look for possible factors that can account for the death rate variation. The death rate elevation of the very long term unemployed is high, greater than that of any group of employed workers. However, the very long term unemployed constitute only a small fraction of the population. The greatest part of the elevation of the total death rate above that of farmers, the lowest adult mortality group, is due to the contributions of factory workers (operatives), craftsmen, and clerical and sales workers—employed groups with moderate death risk elevations but large proportions in the population. Since these workers were *employed* between 1950 and 1960, these data suggest that we must examine the risks associated with work, rather than the lack of work, to account for the business cycle variation of the death rate. Since the death rate peaks with the boom of the cycle, when work is most prevalent, this suggestion is not unreasonable.

Before following up this suggestion, however, I will digress to describe the social and economic mechanisms responsible for the variation in production and distribution of commodities through the business cycle. Then I will show that the variation of income, the ability to buy commodities, cannot account for the variation of the death rate. Nor can the consumption of particular health-promoting and health-damaging commodities account for more than a part of the variation. At this point, I will return

[4] See Appendix 4.

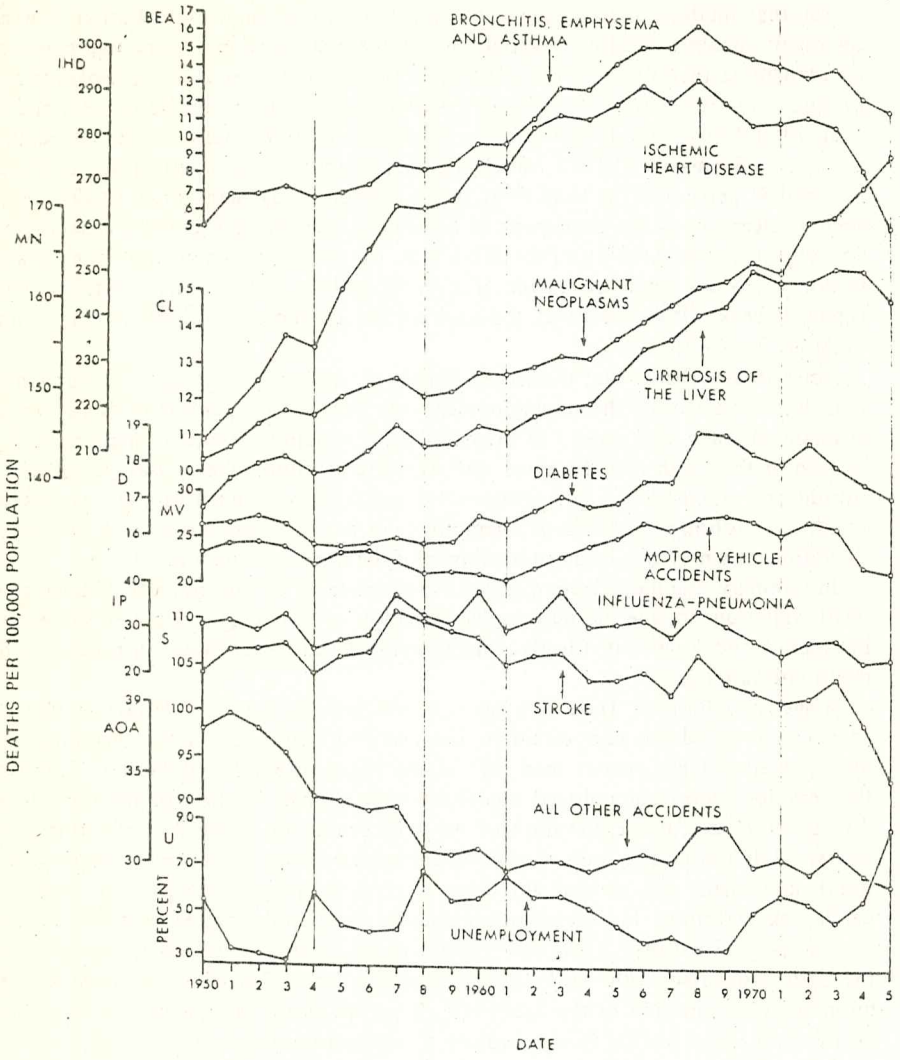

Figure 3. Unemployment rate and death rates by cause of death in the United States, 1950-1975. Sources for death rates by cause by year: 1970-1975 (4); 1950-1969, adjusted for comparability across the 8th International Revision in 1968 (5). Sources for unemployment rate by year, references 2 and 3. *Vertical lines* mark the peaks of the unemployment rate.

to analyze the social process of the business cycle, including overwork and community disruption, to show that this process itself is probably the fundamental cause of the business cycle variation of the death rate.

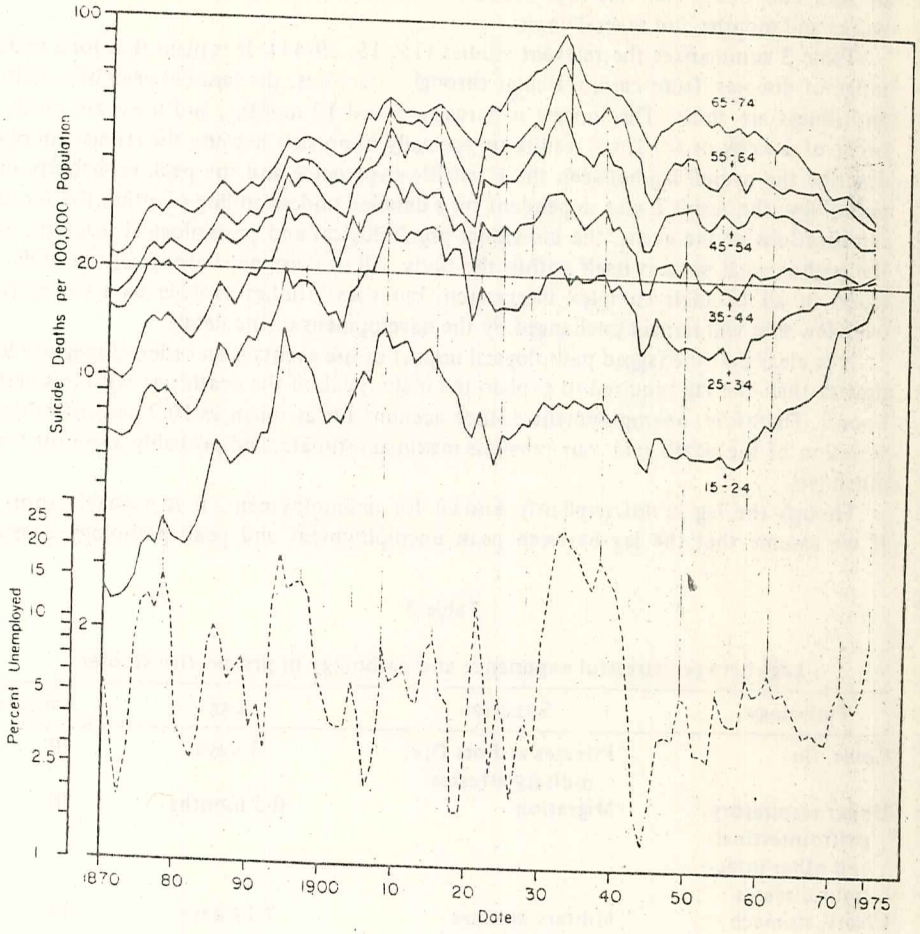

Figure 4. Male suicide rate by age, and unemployment rate, United States, 1870-1975. Sources for suicide rate by year: 1969-1975 (4, 42-44); 1950-1969 (45); 1900-1950 (5); 1870-1900 (46, 47). Sources for unemployment rate by year, references 2 and 3. *Vertical lines* mark the peaks in the unemployment rate.

WIDENING THE FRAMEWORK OF POSSIBLE CAUSES: THE BUSINESS CYCLE AS A CLASS STRUGGLE OVER CONTROL OF THE ECONOMIC PRODUCT

If unemployment cannot account for the business cycle variation of the death rate, perhaps the variation in production and distribution of economic product can.

Figure 5 shows several business cycle series for the years 1949-1975 that are relevant to this path of investigation[5] (48, 49).

At the top of Figure 5 are the monthly data for industrial production (I). The second series on this graph shows the data for profits per dollar of income in private nonagricultural corporations (PS), quarterly. This is a rough indicator of the proportion of the economic product going to the ruling class for consumption and/or investment.[6] The third series is the corresponding data for wages and workers' benefits per dollar of income (LS), a summary indicator of the fluctuating power of the working class over the economic product. The fourth series is the monthly data for percent of the labor force unemployed (U). The fifth series shows percent of employed workers involved in strikes per year (S), an index of the intensity and time extent of labor struggles. The last series is the quit rate (Q), per 100 employed workers, in manufacturing industry. This can be taken as an index of individual worker self-confidence in the labor market.

Several relationships are evident in the data. The share of economic product controlled by the ruling class moves inversely to the share controlled by labor, not a surprising finding. Industrial production and unemployment also move inversely. But there is a characteristic lag between these two groups of series. The high point in the profit share in each cycle comes, not at the peak of industrial production, but over a year earlier, at a point roughly two-thirds the way down the downswing of the unemployment rate from its preceding peak. The low in the profit rate does not occur at the peak of unemployment or the low of industrial production, but rather half a year earlier, roughly halfway up the rise of unemployment from its preceding minimum during the boom. These lags are consistent with the following model mechanism for the change of shares of economic product going to labor and capital through the business cycle.

Rising unemployment weakens the labor market position of workers. As strike funds are exhausted, unions are no longer able to press wage and benefit demands. In addition, capital is freer to fire unproductive workers and hire only the hardest working from among the unemployed, abrogating seniority and other job security structures. These changes not only mean that a higher proportion of social product goes to capital, but that output per worker increases. Both of these trends contribute to the turnaround in the profit rate, as unemployment continues to rise and production falls.

[5] All of the series in Figures 5, 6, 7, and 10 are either monthly or quarterly data that have been seasonally adjusted by the source mentioned, or 12-month moving averages centered on the month for which the data point is plotted, calculated by me from the raw data cited. The use of monthly series allows us to examine the relationship between series with much greater precision than does the use of yearly series, but the large seasonal variation needs to be factored out; otherwise, only the seasonal relationships would be obvious to the eye in many cases. The relationships between these series in the seasonal cycle are themselves a fascinating object of inquiry, which I will develop in another paper.

[6] A detailed investigation of this proportion would also include an examination of the data for interest, rent, and the capital consumption allowance in taxation, as well as comparable income distribution data. While these data are unquestionably of importance for understanding the economic mechanism of the business cycle, profits per dollar of income suffices as a summary measure of the fluctuating power exerted by the ruling class over the economy as a whole.

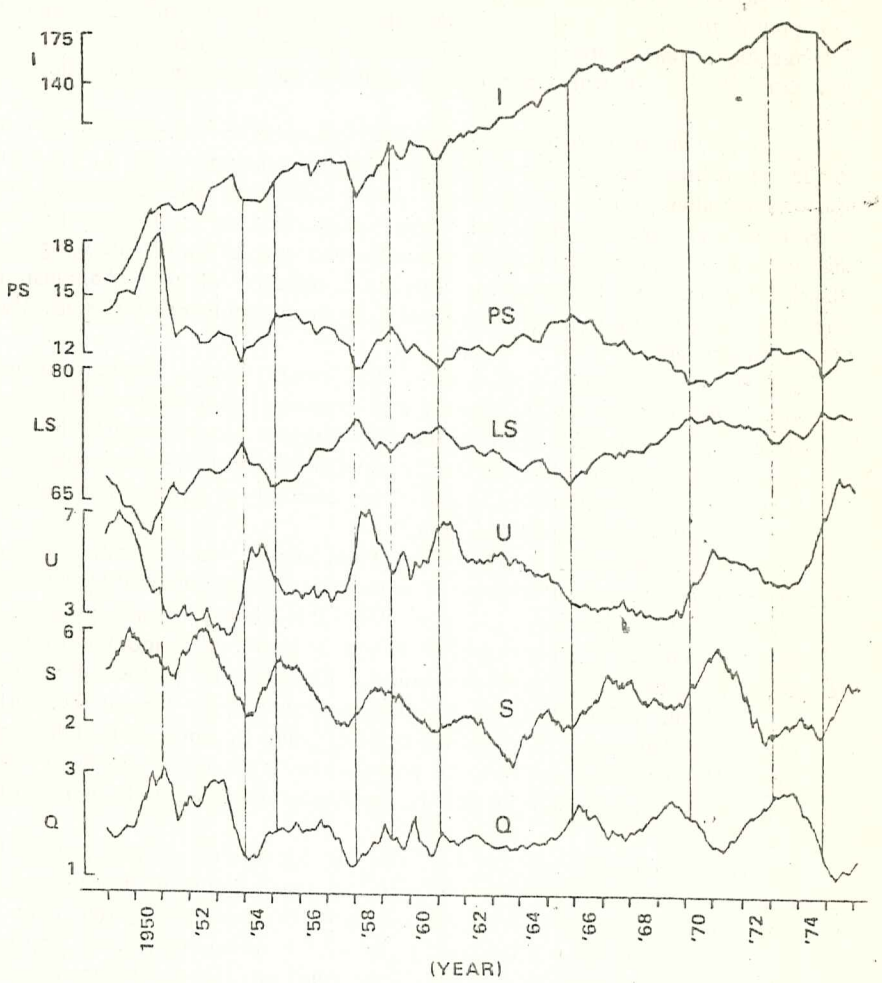

Figure 5. The business cycle as a class struggle over share of the product, 1949-1975. I = industrial production index, log scale (48); PS = profits (after taxes) per dollar of income in private nonagricultural corporations, percent, linear scale (48); LS = workers' wages and benefits per dollar of income in private nonagricultural corporations, percent, linear scale (48); U = unemployment rate, percent of labor force unemployed, log scale (48); S = percent of employed workers involved in labor stoppages, yearly basis, linear scale (49); Q = quit rate, per 100 employees, manufacturing, linear scale (48). *Vertical lines* mark the turning points of the profit rate.

The exact unemployment rate at which the turn occurs varies in different business cycles, depending largely on the prior power of labor, and to a lesser extent on the structural encumbrances of capital. The debate about the Phillips curve since the early 1960s embodies a consideration of these factors.

The rising profit rate results in the expansion of investment and employment by the ruling class, producing the turnaround in production about half a year later. As the unemployment rate is reduced, the strength of labor's position in the market

mproves. This improvement is evident in the series for strikes (*S*, Fig. 5), which hows a decided tendency to rise early in the business cycle, just after the peak of nemployment, and to reach a cyclical high early in the boom[7] (22, Table 40). Growing labor market strength is also evident in several other series, such as the uit rate (*Q*, Fig. 5), which rise rapidly early in the cycle and peak during the oom.

As a result, the share of economic product controlled by capital does not go on teadily increasing, but reaches a high point before unemployment or production each values characteristic of the boom. If the capitalists were capable of precise coordinated economic planning and timely execution, they could keep the profit share at this peak by maintaining the unemployment rate at a moderately high level. Clearly, this has been the explicitly articulated objective of every Administration since 1960 in defining that elusive quantity, the unemployment rate representative of "full employment."

However, considerations of political stability, need to rapidly expand production for war or to maintain foreign markets and resources, inability to rapidly expand foreign immigration or the transmutation of farmers and housewives into wage workers, or other non-labor-market factors impinging on profits, have in every United States cycle so far prevented the success of any attempts to freeze the class struggle on terms cyclically most favorable to capital.

In particular, confronted by a tightening labor market, most capitalists have consistently pursued the overall strategy of attempting to displace labor by investment in more productive machines or processes. While this strategy ultimately does create permanent technological unemployment in one industry after another, over short periods of time the rising overall demand for capital goods further increases the demand for labor and exacerbates the shift in market power toward the workers. The result is a rising share of economic product going to labor, and a falling share to capital. This situation can only be reversed by undermining the labor market power of the workers, usually by the unemployment increase of the next business cycle recession.[8]

There is also a parallel series of events in the organization of capital itself, both technically and financially, which contribute to the variation of investable profits in productive corporations. The social conditions that influence productivity also contribute to this variation. These two pathways are summarized in an Appendix[9] since they contribute less than a third of the total variation of shares. Two-thirds of this variation is due to the class struggle process described above.

[7]Out of a total of 25 peaks in the series "percent of employed workers involved in work stoppages, 1881-1975," 15 come early on the cyclical downswing of the unemployment rate, 5 come either during a war boom or the immediate postwar period when unemployment rates are still low, 2 come during peacetime booms, and 3 occur nearly at the same time as the peak in unemployment, rather than later in the downswing. Of these three, two (1970-1971 and 1974-1975) are shown in Figure 5; the other occurs during 1932-1934. This last change in timing may be a social feature of the onset of major depressions. For further discussion of the war boom peaks in strikes, see footnote 12.

[8]Military suppression and other means of nonmarket social control, including patriotic war against foreign "enemies," are also used to reestablish work discipline favorable to capital, but these are not regularly recurring features of the short business cycle.

[9]See Appendix 5.

VARIATION OF MATERIAL CONSUMPTION AS A POSSIBLE CAUSE OF THE DEATH RATE PEAK WITH THE BOOM

Along one dimension, the business cycle is a back-and-forth class struggle between capital and labor for control over disposition of the economic product. Since the proportion of the population who are capitalists is quite small and, in addition, except for farmers, has the lowest adult death rate (Table 1), it is appropriate to examine the variation in real material product per worker as a possible determinant of the business cycle variation of the death rate. Figure 6 displays the relevant series, with unemployment, industrial production, and the death rate also included for comparison purposes (45, 48, 50, 51).

The *D* series in Figure 6 (and in Figs. 7 and 10 below) is a 12-month moving average of the total death rate; the vertical lines indicate all the peaks in the death rate large enough to significantly affect the yearly figures. As noted above, we must be looking for possibly causal series that show at most a several-month lag from this death rate series, and have at least a roughly similar shape. Data series which do not satisfy these requirements probably do not represent major factors in the business cycle variation of the death rate. A definitive conclusion about each factor, of course, depends on the review of prospective and experimental studies, and the calculation according to equation (1) in Appendix 4.

Series *W'* (Fig. 6) is the take-home (after tax) wages, in constant dollars, of a worker with three dependents in the private nonagricultural economy, 1949-1975. There are four periods in the change of real wages in each business cycle. The low in real wages of employed workers occurs near the peak of the unemployment rate, sometimes lagging it by a month or two (as in the 1950s cycles) or leading it by a similar amount (as in the cycles since 1965). From this low point, real wages increase at the maximum cyclical rate as unemployment drops and output per man-hour rises most rapidly. Like output per man-hour, real wages tend to stagnate during the boom at low unemployment. Real wages typically show a last slight peak late in the boom as unemployment has already started to rise. Real wages of employed workers then decline to reach the cyclical low at high unemployment.

The real income picture looks a little different when unemployed workers and others out of the labor force who also receive income are included. For the mass of workers since the 1930s in the United States, unemployment means a reduction of income to two-thirds to one-half of former pay, on unemployment compensation, roughly correlated with the unemployment rate.

For the lumpenproletariat at the bottom of the urban hierarchy of labor (15 percent of the population), however, the cyclical pattern of income is very different. Welfare payments typically expand from the *middle* of the boom through the peak of unemployment, in response to political necessities. It is only when the capitalists have decisively regained control over both production and the state that welfare payments are cut back to compel the lumpenproletariat into the most undesirable forms of labor. This typically does not happen until the unemployment rate has already started to decline. In the 1930s, for instance, the cutbacks in welfare did not come until 1938-1940, 5 years after the large peak of unemployment in 1933 (52). As a result, the net real legal income of the lumpenproletariat tends to expand from

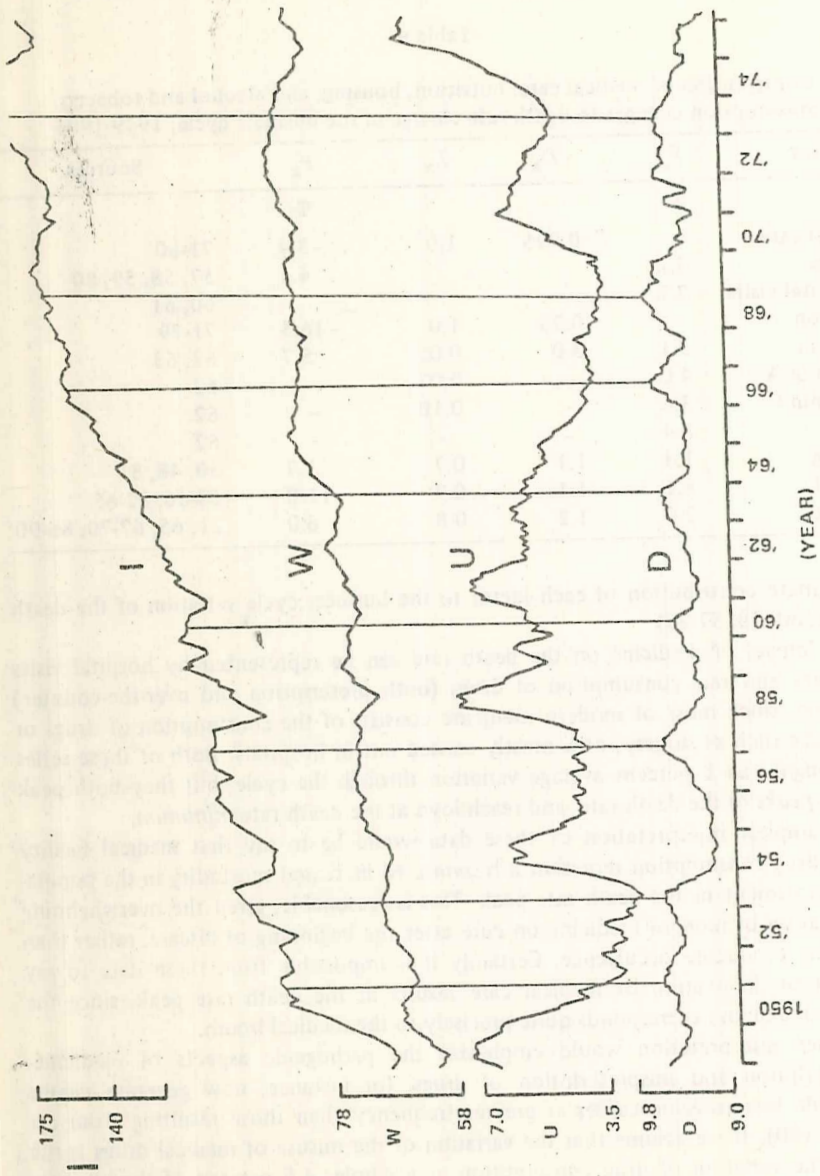

Figure 6. Real consumption and the death rate. I = industrial production index, log scale (48); W = take-home wages in 1967 dollars of a worker with three dependents, total private economy, linear scale (50); U = unemployment rate, log scale (48); D = death rate, per thousand population, linear scale (45, 51). *Vertical lines* mark the peaks of the death rate.

the midst of the boom through the height of the depression. Brenner (53) has made the most thorough survey of these data, in connection with his work on the cyclical peaking of mental illness rates with the unemployment rate.

For certain parts of the lumpenproletariat, illegal income also expands dramatically, as burglaries and other crimes against property also rise from mid-boom to peak with unemployment. A somewhat smaller consequent increase in legal income results for the rising proportion of the population housed in jails with rising unemployment (12, 54, 55).

It is clear that the total level of real consumption by the mass of the population is probably not a major cause of the recurrent peak of the death rate with the boom in the business cycle. The *low point* in total real consumption for over 90 percent of the population comes on the average 1.3 years *after* the death rate peak, and the *beginning* of the fall of real consumption comes on the average 1.8 years *after* the beginning of the rise of the death rate to its business cycle peak. Clearly, the rise of the death rate *precedes* by a considerable time period any fall in real consumption levels by the mass of the population. This is particularly true for the part of the population with the highest death risk by income—the lumpenproletariat.

As I pointed out in the previous paper, however, income and consumption are not homogeneously health-promoting substances. Of the total modern economic product, less than 10 percent is positively health-promoting, and as much as two-thirds may be pathogenic (1). Perhaps the cyclical low in the death rate, which occurs at the peak of unemployment or a little after, corresponds to a change in the *composition* of consumption toward health-promoting items or away from health-deteriorating ones.

This hypothesis is to some extent substantiated by the data, but it is important to make an accounting of the probable contribution of each factor to death rate change, utilizing the information from experimental and prospective studies along with the average variation of each item through the business cycle to attribute proportions. The next section pursues this analysis.

THE CONTRIBUTION OF NUTRITION, MEDICAL CARE, HOUSING, AND ALCOHOL AND TOBACCO CONSUMPTION TO DEATH RATE CHANGE IN THE BUSINESS CYCLE

The factors in this title are most of the economic goods demographic experts believe responsible for death rate changes, both historically and cross sectionally (56). Figure 7 shows the per capita consumption series for each item (45, 51, 57-70). Table 4 shows the basic sources and a simplified form of the calculation[10] of the

[10] The proportions of the business cycle variation of the death rate due to each factor are calculated according to equation (1) in Appendix 4, and the corresponding quantities V_X, D_X, L_X, and P_X are displayed in Tables 4 and 5 and discussed in the text. The reader may approximately verify L_X from examination of Figures 7 and 10 and what is said of prospective studies in the text. When the D_X specified is for a continuous variable (e.g. per capita consumption), V_X may also be verified by examination of the figures. However, when D_X is specified for a high-risk group versus a nonrisk group (e.g. smokers versus nonsmokers), V_X represents the variation of the population at high risk (i.e. not per capita cigarette tobacco consumption rates), which I have calculated using additional data not presented here. These calculations are available on request.

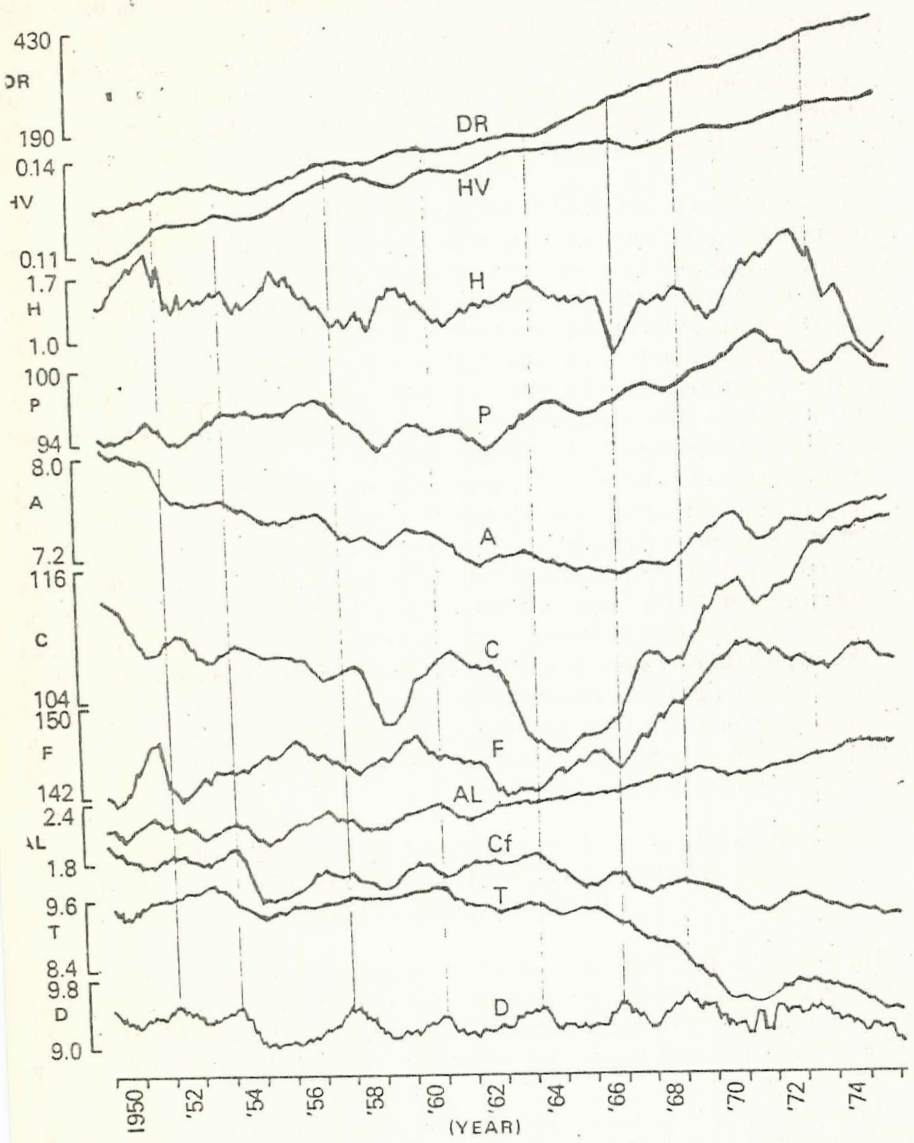

Figure 7. Health-promoting and pathogenic components of consumption, 1949-1975. *DR* = real per capita expenditures on drugs and sundries, index, log scale (57-59); *HV* = hospital visits per capita, number per year, log scale (58, Table 125; 60, 61); *H* = new private housing units started, number per year, log scale (48); *P* = protein consumption per capita, in grams per day, linear scale (62, 63); *A* = vitamin A consumption per capita, thousand I. U. per day, linear scale (62, 63); *C* = vitamin C consumption per capita, milligrams per day, linear scale (62, 63); *F* = fat consumption per capita, pounds per year, linear scale (62, 63); *AL* = alcohol consumption per capita, gallons of absolute alcohol per year, log scale (64-66); *Cf* = coffee consumption per capita, pounds of green coffee beans per year, log scale, scale omitted (62); *T* = cigarette tobacco consumption per capita, pounds per year, log scale (67-70); *D* = death rate, per thousand population, linear scale (45, 51). *Vertical lines* mark death rate peaks.

Table 4

Contribution of medical care, nutrition, housing, and alcohol and tobacco consumption changes to death rate change in the business cycle, 1949-1975

Factor	V_x	D_x	L_x	P_x	Sources
	%			%	
Medical care		0.975	1.0	-3.4	71-80
Drugs	8.2			4.5	57, 58, 59, 80
Hospital visits	7.7				60, 61
Nutrition		0.75	1.0	-16.5	71-79
Protein	3.1	3.0	0.05	5.7	62, 63
Vitamin A	4.0	–	0.07	–	62
Vitamin C	3.6	–	0.10	–	62
Fat	3.4	–	–	–	62
Housing	1.3	1.1	0.7	1.7	30, 48, 81
Alcohol	8.1	1.1	0.7	11.0	64-66, 82-85
Tobacco	2.0	1.2	0.8	6.0	21, 65, 67-70, 86-90

approximate contribution of each factor to the business cycle variation of the death rate (21, 30, 48, 57-90).

The impact of medicine on the death rate can be represented by hospital visits per capita and real consumption of drugs (both prescription and over-the-counter) per capita, since most of modern medicine consists of the consumption of drugs or procedures such as surgery, now mostly carried out in hospitals. Both of these series show roughly an 8 percent average variation through the cycle; but they both peak with the *peaks* of the death rate, and reach lows at the death rate *minimum*.

The simplest interpretation of these data would be to say that medical facility use and drug consumption represent a *response* to increased morbidity in the population, also evident in the death rate peak. This is reasonable, given the overwhelming concentration of modern medicine on cure after the beginning of disease, rather than prevention of disease occurrence. Certainly it is impossible from these data to say that cyclical deprivation of medical care results in the death rate peak, since the mortality maximum corresponds quite precisely to the medical boom.

Another interpretation would emphasize the pathogenic aspects of medicine. Overprescription and misprescription of drugs, for instance, now generate deaths attributable to iatrogenic causes at greater frequency than those resulting from car accidents (80). If we assume that the variation of the misuse of medical drugs is the same as the variation of drug consumption as a whole, 4.5 percent of the business cycle variation of the death rate could be accounted for by this misuse. If excessive and misused surgery produces a similar iatrogenic effect, then as much as 10 percent of the business cycle variation of the death rate might be due to readily analyzable pathogenic effects of medicine. Insofar as the technical misuse and overconsumption of medicine have the character of an addiction (91), it is not surprising that the consumption of medicine peaks along with that of alcohol, tobacco, and coffee in the business cycle (Figure 7, series *AL*, *T*, *Cf*).

However, this analysis of medical nemesis must be balanced by the indubitable beneficial impacts of modern medicine on survival of accident trauma, the prevention of infectious disease through immunization, the reduction of infectious mortality through proper use of antibiotics, and the impact of birth control and abortion on preventing unwanted, high-risk pregnancies and births, for example. Only by comprehensive, prospective controlled study of modern medicine, or the systematic piecemeal review of all double-blind placebo controlled medical evaluation studies of specific techniques, would it be possible to describe this balance. The latter is beyond the scope of this paper, but the former has been assayed by Scrimshaw (76-79), whose results parallel those of McKeown (71-75), based on historical analysis.

Briefly, Scrimshaw found much greater improvement due to nutrition than that attributable to medical care (antibiotics and immunization) in a controlled study of the reduction of infectious morbidity and mortality in several Central American villages. The proportional impact of these two factors in Scrimshaw's studies is congruent with McKeown's independently arrived at estimates for the contributions of nutrition, environmental sanitation and housing, and medicine to the death rate decline in modern countries since the late 19th century. Improved nutrition contributes over half of this decline, environmental sanitation and housing about a quarter, and specific medical measures less than one-tenth.

If these estimates are used as a basis for assigning a net impact to medical consumption changes, medicine may act as a "buffering" factor, reducing the business cycle variation of the death rate 3.4 percent (Table 4).

Both McKeown's and Scrimshaw's studies demonstrate that nutritional changes have a large impact on overall mortality rates. By and large, nutrient consumption peaks with business booms and therefore with the peak in the death rate, though with several discrepancies. Using these same studies to estimate the impact of nutrition, the increase in nutrients consumed during the boom may "buffer" the business cycle variation of the death rate by as much as 16.5 percent.

Alternatively, we could start from the discrepancies rather than the covariation of nutrition and the death rate, to develop the possible causal pathway between nutritional deprivation and death rate increase. Though this pathway is unreasonable given the actual pattern of nutrient consumption variation, it leads to the second estimate shown in Table 4 under protein. Because protein consumption mostly peaks with the death rate and otherwise has erratic relations to it, and because the lag in prospective studies is less than 6 months, L is very small on this hypothesis. The situation is similar for vitamins A and C, both of which also affect immunity to infectious disease. The contribution of the variation of these vitamins to the variation of the death rate is not computed, because prospective studies disagree whether D_x is any greater than 1.

For fat consumption, it is worthwhile to note that this series peaks with the death rate minima and bottoms out at the death rate peak. This is counterintuitive if we believe that fat consumption contributes to cardiovascular disease mortality. However, this view is questionable, since the Framingham study, the largest American prospective study, shows no relationship between dietary fat and cholesterol, and cardiovascular disease risk (92).

Series H (Fig. 7) shows new housing starts, 1949-1975. Housing construction is very sensitive to interest rates and credit flows, and to the prices of raw materials and labor. Since credit is most easily available, prices are stable or falling, and labor's share of the product is decreasing early in the business cycle, housing construction booms and peaks out at this time. During the overall boom of the economy, housing production declines, often reaching its low at or slightly after the peak in general production. This pattern of variation obviously makes it a candidate to account for the business cycle variation of the death rate. In Figure 7, the peaks of the death rate do indeed correspond to the troughs in housing construction in about half the cases.

In simple cross sections within cities, poor housing conditions are strongly associated with high death risks (93-100). However, rural housing has historically been and continues to be poorer in quality than urban housing, yet adult death rates are lower in rural areas, and rural infant and child mortalities were lower than urban before the 20th century in many modernizing countries (101). Clearly, simple cross sections do not suffice to define the health impact of housing.

There are very few prospective controlled studies of the health impacts of housing improvement. The best one was carried out in Baltimore, 1955-1958 (30), and involved a lower-class group which moved into new public housing, compared to a matched group which remained in the slums. The mortality experience was too limited to be meaningful in this study, but if we assume that mortality changes would be of the same magnitude as morbidity changes, the probable impact of new housing construction on the business cycle variation of the death rate comes out as shown in Table 4.

The contribution of housing is small mostly because the probable mortality impact of housing change per se, when other factors are controlled in a prospective study, is small. It is also smaller than we would anticipate because this study demonstrated that sickness *increased* in the first few months after moving into new housing, a migration effect I will review in greater depth later in this article. The maximum reduction of morbidity occurred a year or more after the move. As a result, L is lower than would be anticipated if the health improvement resulting from housing change occurred without a lag.

Series AL, Cf, and T (Fig. 7) show the business cycle variation of alcohol, coffee, and cigarette tobacco consumption per capita. Mass drug consumption clearly correlates well with death rate increases and decreases since 1949, though, as I pointed out in my previous paper, tobacco consumption does not show a business cycle variation during the 1920s and 1940s. Thus the factor L is nearly 1, though somewhat less because of discrepancies (e.g. the lack of a 1963 peak in alcohol consumption to correspond to the death rate peak).

Many analyses have implicated smoking as a fundamental cause of excess mortality—of males over females (102-104), of urban dwellers over rural (86), of blue-collar workers over higher-class people (89). Though smoking is thought to account for over half the excess in some of these comparisons, the contribution of the variation of smoking to the business cycle variation of the death rate is smaller, for two reasons. The variation of tobacco consumption with the business cycle, though significant, is small (2 percent). Second, not all of the excess mortality among smokers compared to

nonsmokers can be thought of as a short-term effect developing over times as short as 3 or 4 years.

Indeed, the smoking effect can be partitioned into two components, only one of which is of interest to our analysis. Over long periods of time, the carcinogens in tobacco smoke contribute to the development of lung cancer. Over equally long periods, the carbon monoxide in tobacco smoke accelerates the process of atherosclerosis, potentiating heart attacks. Over somewhat shorter periods, but still longer than those of the business cycle, smoking produces a chronic irritation of the upper respiratory mucous membranes, yielding increased frequencies of colds and eventually chronic bronchitis, which can progress toward emphysema.

Smoking also has immediate effects. The primary such effect is the reduction of oxygen-carrying capacity of hemoglobin in the blood by the binding of carbon monoxide, blocking the oxygen-binding site, thus potentiating heart attack and increasing death risks for newborn infants of mothers who smoke. This effect does not result in significant mortality until atherosclerosis is already well advanced. The figure given for D_x, 1.2 (Table 4), is an estimate of this immediate impact. The total excess mortality ratio of smokers over nonsmokers is about 1.6X in several large prospective studies (21, 86-88).

In the postwar United States, alcohol variation actually makes a larger contribution to the business cycle variation of the death rate than smoking (11 percent compared to 6 percent, Table 4). This is entirely due to the fact that the variation of alcohol consumption with the business cycle is much larger. As with smoking, the figure for D_x is based on the partition of alcohol impacts into long-term developments, resulting, for instance, in liver cirrhosis and certain cancers, and short-term effects, such as the potentiation of accidents, suicide, homicide, or the susceptibility to tuberculosis or pneumonia (82-85).

Together, the factors examined in this section can account for 24.4 percent of the business cycle variation of the death rate, with an additional 3.4 percent in the reduction of variation due to medicine, and 16.5 percent in the reduction of variation due to nutrition.

THE AGE AND SEX PATTERN OF THE BUSINESS CYCLE VARIATION OF THE DEATH RATE SUGGESTS A SOCIAL NETWORK STRESS EFFECT AS FUNDAMENTAL

Figures 8 and 9 show that the peak of the death rate with each boom of the business cycle not only occurs in all age and both sex groups, but is roughly the same size and shape, in proportion to the level of the death rate, in all these groups (2-7). This pattern is not what would be expected for several of the factors reviewed in the previous section.

For example, it is known that nutritional deficiencies affecting immunity are decidedly most common among infants, toddlers, and the very old in the United States (63). We should expect a much greater proportional variation of death rates at these ages as nutrition varies through the business cycle, if nutrition is a primary factor. The effect of housing on health is most pronounced for these same groups (30); this also differs from the actual pattern of variation.

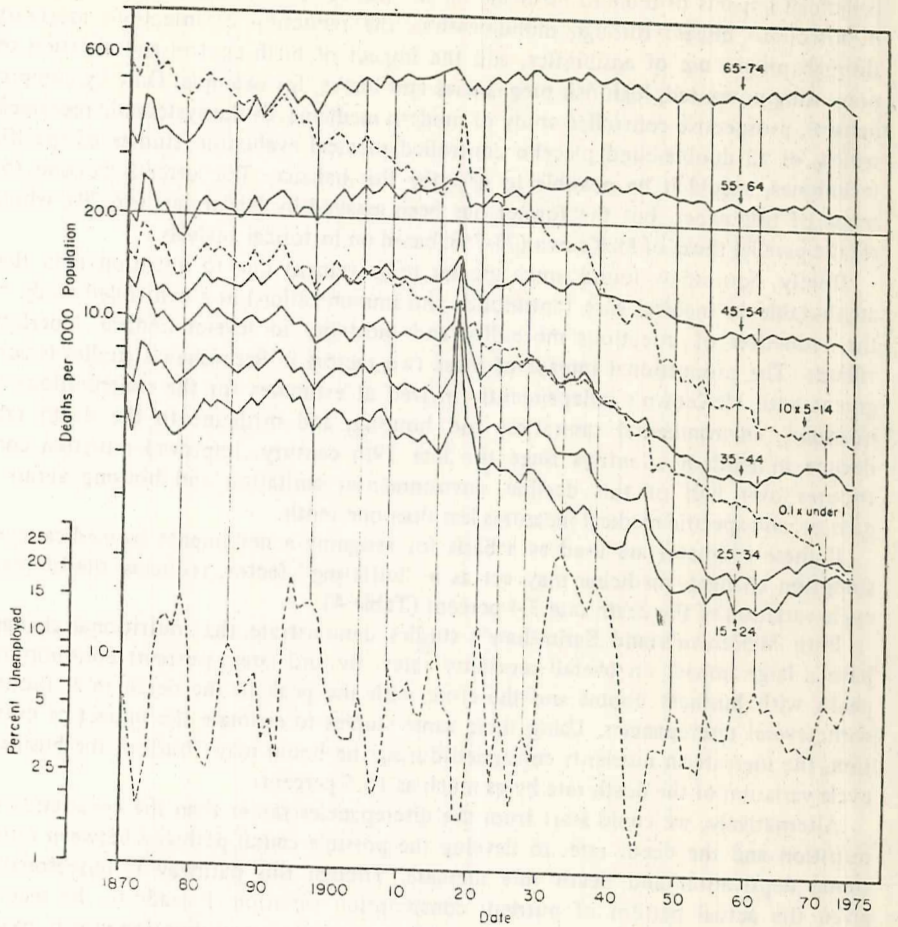

Figure 8. Male death rate by age, and unemployment rate, United States, 1870-1975. Sources for death rates by sex and age, and unemployment rate, as in Figure 1 (2-7). *Vertical lines* mark unemployment rate peaks.

Heavy alcohol and tobacco consumption are engaged in much more by males than by females. Drug use typically begins during adolescence and declines from a young adult peak with age. The mortality consequences of drug use start with accidents, suicide, and homicide in young adulthood and extend through middle age with cirrhosis and into the fifth and sixth decades with bronchitis, emphysema and asthma, lung cancer, and ischemic heart disease. If mass drug consumption were the primary factor in the business cycle variation of the death rate, we would expect the largest proportional variations of the death rate among males aged 20-65. A similar pattern would be expected for specific workplace occupational hazards, such as toxic chemicals, radiation, or local pollution.

Different causes of death account for the overall death rate variation at each age,

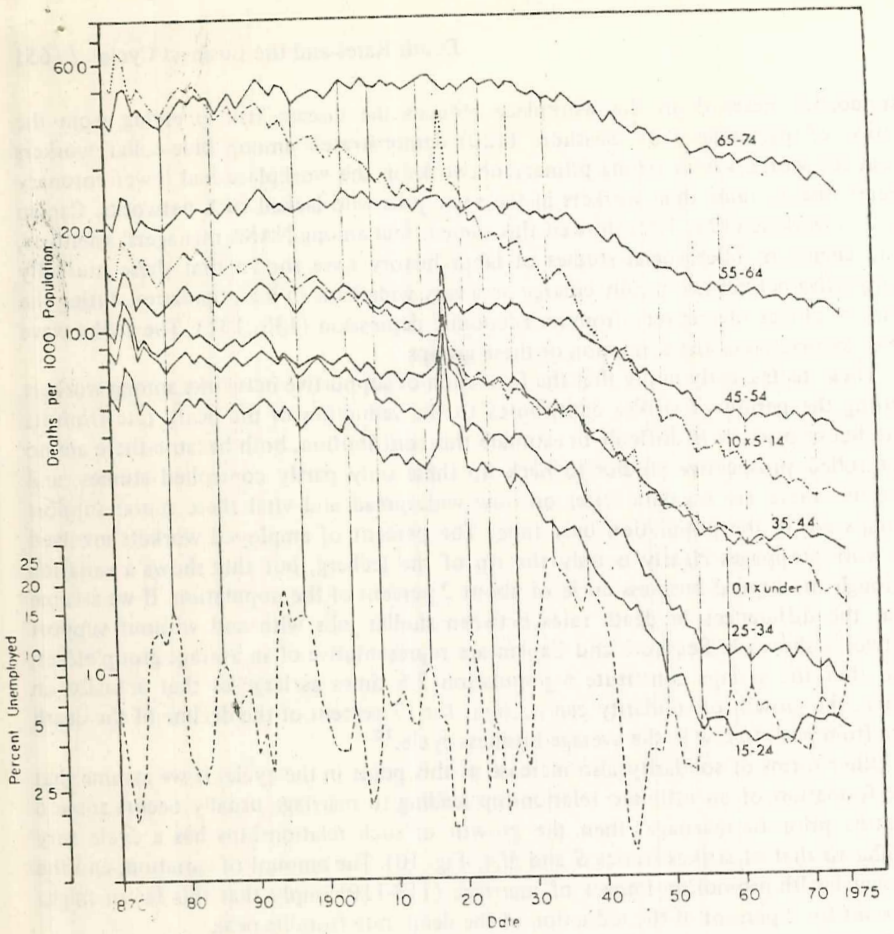

Figure 9. Female death rate by age, and unemployment rate, United States, 1870-1975. Sources for death rates by sex and age, and unemployment rate, as in Figure 1 (2-7). *Vertical lines* mark unemployment rate peaks.

and to some extent by sex. Among infants and toddlers, the greater part of the variation is due to infectious diseases. Among children and adolescents, accidents are responsible for most of the variation. Among young adults, accidents are joined by suicide, homicide, and drug-related deaths. Beyond age 30, influenza-pneumonia plays a larger and larger role, finally dominating the variation as a whole at the oldest ages; but in the decade of the 30s, accidents, homicide, suicide, and cirrhosis outweigh it, in the 40s and 50s cirrhosis and cardiovascular disease predominate, and in the 60s and 70s, cardiovascular disease and cancer account for most of the variation. For women, this variational pattern differs only by the absence of cirrhosis as a major cause of variation at ages 30-50.

Single factors linked to single diseases clearly cannot account for this pattern, unless they themselves vary similarly. As we have suggested, such similar variation is probably due to underlying social stress variation.

The spreading impact of social stress in a network, however, can easily generate an overall population death rate variation pattern in which all ages and both sexes are similarly affected.

Minuchin and coworkers (105-107) have shown that the level of free fatty acids in the blood, an index of physiological arousal, is elevated in every member of a stressed family network. However, during family therapy and in ordinary situations, stress in the network is focused now on one individual, then on another, as patterns of alliance, stigmatization, and exclusion shift. Corresponding to these shifts, the person on whom the network stress is focused shows the greatest elevation of free fatty acids. At one time this individual may be the child or infant, at another the wife, grandfather, husband, close friend of the family, or coworker.

These alliance and exclusion patterns are not only shifting over periods as long as a year or a business cycle, but have different configurations in different families and work groups. Whatever its initial point of application, if the net stress on a large population rises and falls over periods of several years, the death rate impact will therefore be distributed roughly proportionally in all age and both sex groups.[11]

As I demonstrated in the previous paper, stress causation can also account for the presence of the same variation pattern in many different disease processes, all of which are affected by physiological arousal (1).

SOCIAL RELATIONSHIP CHANGES MAY ACCOUNT FOR THE GREATEST PART OF THE DEATH RATE VARIATION THROUGH THE BUSINESS CYCLE

The same class struggle which determines the growth and decline of production and the varying distribution of commodities to different classes is itself a *social* process which can independently account for the business cycle variation of the death rate. This social causality flows through two opposite sets of phenomena: the growth of solidarity, and the spread of atomistic, alienated disintegration in the majority of the population. Figure 10 shows the natural time series for these social processes (48, 49, 54, 58, 108-112), and Table 5 shows the relevant basic data and sources (1, 30, 32-40, 48, 49, 54, 58, 87, 88, 101, 108, 109, 110-129).

Careful comparison of series *S* in Figure 10 (percent of employed workers involved in work stoppages) with the death rate (series *D*) shows that the low in strike activity corresponds to the peak in the death rate, and the peak of strike activity, coming early in the boom, corresponds to the cyclical low in the death rate (see footnote 7 for comment) (22, Table 140; 130). Several studies demonstrate that a cohesive,

[11] While this is true for short, small fluctuations of social sources of stress, it is not so for large-magnitude chronic differences in stress impact on population subgroups. Thus, adult males currently suffer more work stress than adult females; though this impacts women also through the family and nonfamilial intimate relations, female death rates for stress-related causes of death are lower than male's at corresponding ages. For a detailed analysis of these chronic stress impacts, see references 101 and 104.

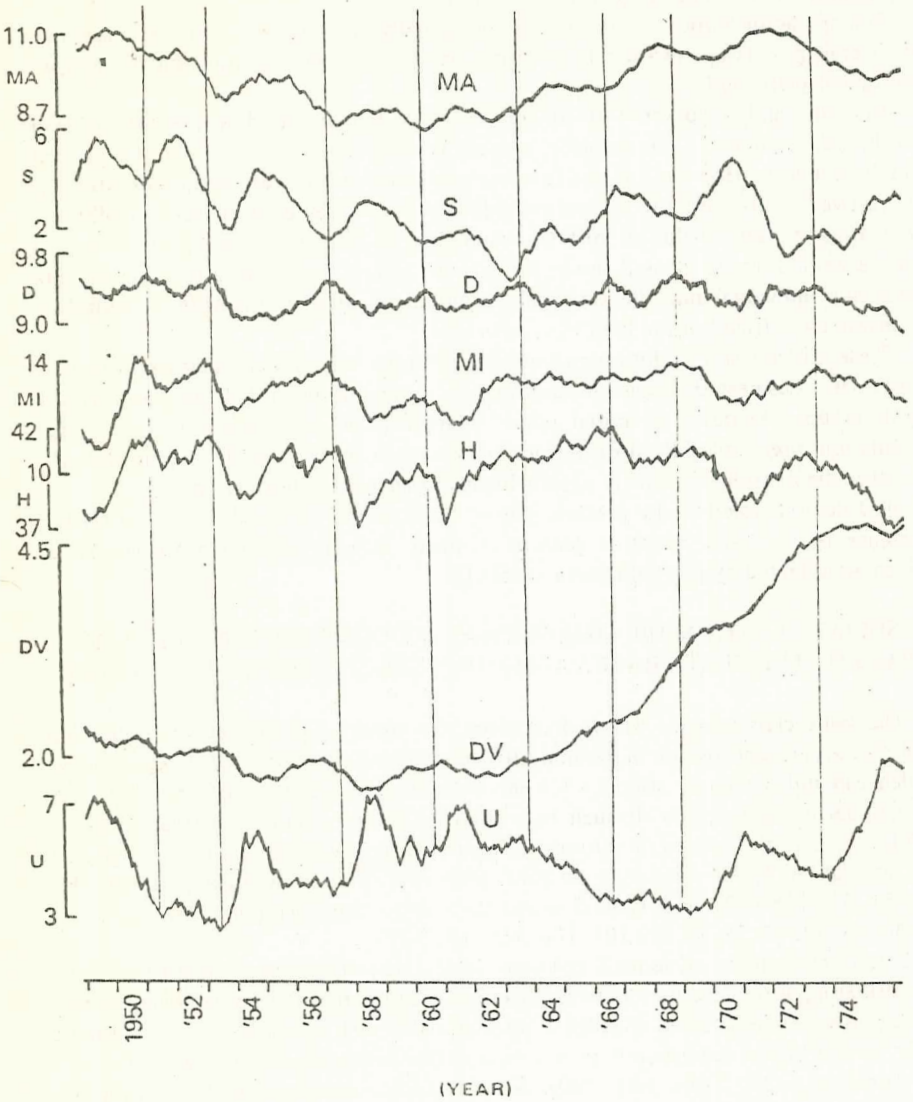

Figure 10. Social solidarity and social disintegration-overwork as determinants of the death rate. *MA* = marriage rate, per thousand population, linear scale (108, 109); *S* = percent of employed workers involved in labor stoppages, yearly basis, linear scale (49); *D* = death rate, per thousand population, linear scale (45, 51); *MI* = migration rate, people living in different county after one year, per thousand population, linear scale (54, Series C81 and C85; 58, Table 157; 110); *H* = average workweek, hours, production workers in manufacturing, linear scale (48, 111); *DV* = divorce rate, per thousand population, linear scale (108, 112); *U* = unemployment rate, percent of labor force unemployed, log scale (48). *Vertical lines* mark death rate peaks.

supportive network in the workplace reduces the disease risk emerging from the stress of particular jobs. Seashore (120) demonstrated among blue-collar workers that the workers with strong primary networks in the workplace had lower coronary heart disease rates than workers in the same jobs who lacked such networks. Caplan and coworkers (121, 122) showed this same effect among NASA managers, scientists, and engineers. Microsocial studies of labor history have shown that these mutually supportive networks typically emerge on a very wide basis in the population during the earliest phases of recovery from an economic depression (130, 131). The strike wave is an expression of the formation of these groups.

These facts clearly imply that the formation of supportive networks among workers during the period of strikes contributes to the reduction of the death rate from its late boom peak. It is difficult to estimate this contribution, both because there are no controlled prospective studies to back up these only partly controlled studies, and because there are no data series on how widespread and vital these mutal support groups are in the population over time. The percent of employed workers involved in work stoppages clearly is only the tip of the iceberg, but this shows a variation through the typical business cycle of about 2 percent of the population. If we assume that the differences in death rates between similar jobs with and without support networks shown in Seashore and Caplan are representative of an average group effect, and that the groups constitute a population 2.5 times as large as that actually on strike, the growth of solidarity can account for 17 percent of the decline of the death rate from peak to low in the average business cycle.[12]

Other forms of solidarity also increase at this point in the cycle. If we assume that the formation of an intimate relationship leading to marriage usually occurs some 6 months prior to marriage, then the growth of such relationships has a cycle very similar to that of strikes (series *S* and *MA*, Fig. 10). The amount of variation, and the known health-promoting impact of marriage (113-119) imply that this factor might account for 2 percent of the reduction of the death rate from its peak.

Together, readily identifiable forms of solidarity might account for 19 percent of the business cycle variation of the death rate.

As the boom develops, this solidarity disintegrates, usually long before the rise of unemployment. *Migration* destroys solidarity by enhancing labor market competition

[12] The two peaks in strike activity after World War I and World War II are the largest in the whole series and exceed ordinary peacetime strike waves by a factor of 6 or 7. Therefore, it is interesting to note that these two postwar peaks of strikes at low unemployment correspond to dramatic death rate *declines* (1919, 1946, in Figure 1), rather than the death rate peak that would be expected from the stresses of the economic boom alone. The three-fourths of the strike peaks which come early on the downswing of the unemployment rate in the cycle correspond to death rate minima. These data imply that strikes operate as an *independent* factor in reducing the death rate, countering the influence of social sources of stress which rise with the boom.

If this effect is strictly proportional to the percent of workers on strike, we should expect that a strike wave 7 times the average should be able to reduce the death rate 7 × 17 percent or 119 percent of the average business cycle variation of the death rate. If this occurred during the average boom, the result would be to turn the normal boom death rate increase of 5.3 percent into a decline of 1 or 2 percent. Thus, our estimate of the impact of worker solidarity on the death rate, derived from small-scale studies, is broadly consistent with the historical data, but may be an understatement.

among strangers. *Divorce* in part reflects the divergent pulls of high demand on male and female labor. *Overwork* deprives people of the relaxed time necessary to form cooperative social bonds. All of these phenomena peak with the boom and account together for the greatest part of the death rate increase.

Series *MI* (Fig. 10) shows the variation of long-distance net permanent migration with the business cycle. Migration starts to rise when the labor market is getting tight and the rise of real income makes migration possible and attractive in terms of wage differentials (132). At the end of the boom, there is usually a lag of information of several months between the actual ending of economic opportunity elsewhere and the departure of migrants. Therefore, the migration rate peaks with the boom, mostly late in the boom. As the figure shows, the migration rate peaks with every peak of the death rate, bottoms out early in the recovery, and otherwise has a very similar business cycle "shape."

The relative death risk for long-distance migrants moving into a hostile economic and cultural context is quite large, on the order of two- to threefold over the average adult death rate at the point of arrival. The relative death risk elevation for shorter moves into familiar contexts where there is a known social support network is small, perhaps 1.0-1.2X (30, 101, 127). The figure shown in Table 5, 1.4X, is a probable figure for long-distance permanent migration, the phenomenon for which we have data. As I pointed out in the review of the Baltimore housing study, this impact occurs in the first few months following migration (30).

Together, these data imply that the variation of migration can account for 9.1 percent of the variation of the death rate through the business cycle.

The divorce rate is shown in series *DV* (Fig. 10). The divorce rate does not peak during the depression, when one might think the financial stresses on the family most severe, but rather late in the boom. Economic demographers have a number of explanations for this (133). First, they usually point out that divorce costs money

Table 5

Contribution of solidarity and social disintegration-overwork to the business cycle variation of the death rate

Factor	V_x	D_x	L_x	P_x	Sources
	%			%	
Marriage	0.2	1.6	0.9	2.0	108, 109, 113-119
Strikes	5.0	1.2	0.9	17.0	49, 120-122
Migration	1.5	1.4	0.8	9.1	1, 30, 54 (Series C81 and C85), 58 (Table 157), 101, 110, 127
Overwork	2.0	2.0	0.9	34.0	1, 32-40, 48, 87, 88, 111, 123-129
Divorce and separation	0.27	3.0	0.8	8.1	108, 112, 113-119
Unemployment	2.84	1.2	0.1	1.07	48, Table 2

and the rise of real wages to the boom provides the resources for divorce. Second, working women have a higher divorce rate than housewives, at least in part because of their economic independence. Women's labor force participation typically rises cyclically during the boom and peaks out at the beginning of the depression. This rise in labor force participation by women usually contrasts with a level or falling labor force participation rate of adult men during the boom (134), and can be understood in the context of capital's attempt to replace expensive male workers, in order to stave off the fall of the profit rate during the boom (135). While these explanations do not exhaust the phenomenon of divorce as a whole, they may well account for the *variation* of divorce (which is smaller than the total) with the business cycle.

The variation of divorce affects about 0.09 percent of the population, but there is reason to believe that informal separations proceed from the same causes and have a similar cyclical timing. Especially among the urban poor, the growth of welfare income, predominantly oriented to the support of single-parent families, reenforces the effect of preferential female hiring late in the boom in producing separations or unmarried families. Census figures show that informal separations are two times more prevalent than divorces (58, Table 59). Therefore, 0.27 percent of the population might be affected by the variation of divorce and separation together.

The relative death risk of divorced or separated people is highest immediately after divorce, and can be quantitatively estimated from the death rate by marital status and prospective studies (113-119). These data yield a three- to fourfold risk increase through most adult ages, and imply an even higher risk increment for the children of divorce, particularly young adolescents (119). Therefore, as much as 8 percent of the variation of the death rate through the business cycle might be due to marital disintegration.

Series *H* in Figure 10 shows average hours of work per week per worker in manufacturing establishments in the United States, 1949-1975. This series clearly also peaks with the peaks of the death rate. Together with the distribution of hours worked (123), these data imply that the business cycle variation in the proportion of the population working over 60 hours per week is about 2 percent.

The relative risk associated with overwork has not been studied prospectively for total mortality, but has been investigated intensively for coronary heart disease, which is the largest single cause of death. Table 5 summarizes the studies; these yield a reasonable estimate of an increase of 2.0 to 2.5X with work over 60 hours per week, in the weeks and months immediately following overwork. A similar impact on total mortality is not unreasonable to presume, since it is known that overwork increases the risk of tuberculosis, potentiates accidents, and increases incidence of ulcers and many other causes of death, though these effects have not been subjected to controlled study (1, 136-138).

Together these data imply that the variation in overwork can account for 34 percent of the business cycle variation of the death rate.

If solidarity and disintegration-overwork are treated as independent factors, then all of the social processes described in this section can account for the majority, 72 percent, of the business cycle variation of the death rate.

SUMMARY AND FUTURE WORK

Table 6 summarizes all of the causal relations we have examined in each section of this paper. The factors shown account for 97 percent of the variation of the death rate through the business cycle. These factors are also organized into what I believe is the most reasonable causal hierarchy. Thus, social relationships account for the greatest part of the variation directly (19 percent plus 53 percent). However, social stress also works indirectly, via the increased consumption of alcohol and tobacco, to produce a further 17 percent of the variation. The reasoning behind this causal hierarchy is explained at length in my previous paper (1).

The figures summarized in Table 6 are clearly first order of magnitude estimates only. They could be greatly improved by carrying out a large-scale prospective study, involving frequent measurements extending through several business cycles, of all of the factors considered here. To a certain extent, this study can be approximated by a reanalysis of the Framingham data, which extend over many business cycles since the early 1950s and cover almost the whole of a working-class town.

It would be particularly interesting to study prospectively the health impacts of solidarity, since this is a rather large factor in the present causal accounting of the business cycle variation of the death rate. Beyond workplace groups and nuclear marriage, other forms of solidarity, such as cooperative networks among adult women (139), also deserve explicit attention, since there is reason to believe that they also promote health by enabling people to deal better with stress, and ultimately, to control the source of stress.

Table 6

Causal relations accounting for the business cycle variation of the death rate, 1949-1975

MATERIAL CONDITIONS (8%)
Housing (2%)
Nutrition (6%)
SOCIAL RELATIONS IN CLASS STRUGGLE (89%)
SOCIAL SOLIDARITY (19%)
Strikes (17%)
Marriage (2%)
SOCIAL DISINTEGRATION AND OVERWORK (53%)
Overwork (34%)
Migration (9%)
Divorce (8%)
Unemployment (2%)
DRUG CONSUMPTION (17%)
Alcohol (11%)
Tobacco (6%)

APPENDICES

APPENDIX 1

Since we assumed that the whole differential by occupation was due to unemployment alone, a linear regression line was constructed through the points in Figure 2, going through the origin. A 2.5 percent increment in unemployment rate along this regression line, starting at 4 percent (the average value of the unemployment rate during business booms in the post-World War II period), corresponds to a death rate index increase from 1.49 to 1.85, a 24 percent increase.

APPENDIX 2

The Framingham death risk equations for blood pressure represent the summation of blood pressure values for many years, versus eventual cardiovascular death risk. If eventual cardiovascular death risk is linearly proportional to the time spent at elevated pressure, the total death rate increment to be expected from a given blood pressure increase in the whole population, sustained for a specified duration, can be approximated as follows:

$$I = \begin{array}{c}\text{expected relative}\\ \text{increment in}\\ \text{death rate}\end{array} = \begin{array}{c}\text{relative cardiovascular}\\ \text{death rate increment in}\\ \text{the F study for blood}\\ \text{pressure increase specified}\end{array} \times \frac{\text{duration of blood pressure increase, years}}{\begin{array}{c}\text{average number of years used to compile}\\ \text{F equations}\end{array}}$$

This yields the relative death rate increment, were the whole population exposed to the blood pressure increase. If only a fraction (V) of the population is newly exposed to the increase for the duration specified, the increase of the death rate (relative to an initial death rate = 1), is given by

$$\begin{array}{c}\text{expected total population}\\ \text{death rate increment}\\ \text{relative to 1}\end{array} = [(V)(1+I)+(1-V)(1)]-1, \text{ or } (V)(I).$$

In the typical postwar business cycle, the unemployment rate increases 2.5 percent from boom to recession, the average duration of unemployment is 11 weeks, and the relative cardiovascular death rate for a 5 mm Hg increase in blood pressure starting from 140 mm Hg systolic is 1.04. Therefore the total relative death rate increment to be expected is:

$$(0.025)\left(0.04 \times \frac{11/52 \text{ year}}{8 \text{ years}}\right) = 0.000027, \text{ or a } 0.0027 \text{ percent increase.}$$

APPENDIX 3

The method of calculation in Appendix 2 can be extended by a summation process over the different durations of unemployment, thus:

$$\begin{array}{c}\text{expected total population}\\ \text{death rate increment}\\ \text{relative to 1}\end{array} = \sum_{t=0}^{10} \left\{[V_u(t)][D_u(t)] + [1 - V_u(t)](1)\right\} - 1$$

$$\text{or} \sum_{t=0}^{10} V_u(t)[D_u(t) - 1],$$

where $V_u(t)$ is the increase, in the average recession, of the proportion of the population unemployed for duration t; $D_u(t)$ is the relative death risk of being unemployed for duration t; and t extends from zero to 10 years, as in Table 2.

APPENDIX 4

The method of calculation in Appendix 3 can be extended to include a lag term, L_u, which compares the pattern of change of the death rate following a peak in unemployment with the time pattern of change of mortality risk in a prospective study, following the unemployment of individuals in the study. If these two patterns coincide when the initial values are adjusted to be the same, then $L_u = 1$: to the extent that the patterns diverge through one whole cycle of the death rate, L is proportionally less than 1, with $L = 0$ for a comparison in which the death rate and the prospective risk pattern move exactly inversely. L therefore gives a rough idea of the proportion of the calculated increment in the death rate previously attributed to unemployment which might reasonably fit with what is known of the time decay of the effect of unemployment.

The full formula for any factor is

$$\begin{array}{c}\text{expected total population}\\ \text{death rate increment}\\ \text{relative to 1}\\ \text{from factor } X\end{array} = V_x (D_x - 1) L_x$$

and this can be expressed relative to the average business cycle variation of the death rate (5.3 percent) as a proportion of the business cycle variation of the death rate possibly due to factor X, P_x:

Equation (1):
$$P_x = \frac{V_x (D_x - 1) L_x}{0.053}$$

which becomes a percentage of the average business cycle variation of the death rate if V_x is expressed as a percentage rather than a decimal. Thus for unemployment,

$$P_u = \frac{(2.5)(1.227 - 1)(0.1)}{0.053} = 1.07\%.$$

Here D_u, 1.227, is the weighted average of $D_u(t)$ from Table 2, and L_u, which would be zero if no lag were permitted, is 0.1 because of the allowed lag up to 6 months.

APPENDIX 5

The *data series* referred to in this Appendix may be examined by the reader in *Business Conditions Digest* (48), which has detailed graphs of monthly or quarterly series such as I have illustrated in this paper.

Changes in the organization of capital itself during the boom also contribute to the decline in investable profits. At first, the boom in capital goods production is fueled more or less completely by the rise of internally originating profits early in the boom, as the parallel between the rising profit share and the series for *orders of new capital equipment* shows. New capital equipment typically requires between 6 months and 2 years to construct prior to delivery in immediately useful form, however; inventory (of both raw materials and finished goods) also has a long adjustment time. Therefore, the actual *expenditures on capital goods* tend to peak late in the boom, when internally originating profits are declining.

As a result, corporations borrow heavily from financial institutions toward the end of the boom, which raises *interest rates*. The increasing cost of new investment, compared to a steadily declining rate of return, contributes to determining the point at which capitalists stop investing. This strike of capital results in the decline of employment and production—the depression phase of the cycle.

As unemployment rises and production falls, there are several changes, in addition to the breaking of labor's power, which lead toward economic recovery. First, the boom in capital goods, raw materials, and inventory buying comes to an end, resulting in a dramatic collapse in *loan demand*, with an attendant decline in interest rates. This makes subsequent new borrowing easier. But recovery does not fundamentally come from the renewed availability of cheap credit; when the upswing comes, it is financed predominantly from internally originating profits.

Therefore, we must examine the factors, beyond the re-creation of labor discipline, which contribute to the rise in the profit rate within corporations.

Output per man-hour falls with the beginning of the recession, but starts to rise at the same point on the rise of unemployment as the turning point in shares to profit and labor. Output per man-hour then rises at its maximum cyclical rate through the peak in unemployment and into the early boom, and then begins to stagnate. In part, this series behaves in this way as a reflection of the general turnaround in labor discipline and organization in favor of capital.

However, capital itself undergoes a process of reorganization from the boom through the depression which contributes to increased efficiency and therefore to the rise in productivity. This reorganization is highlighted by the cyclical behavior of *percent of industrial capacity actually utilized*. Despite a late boom wave of new capacity installation, the rate of capacity utilization starts to fall early in the boom and sometimes falls dramatically before the peak in industrial production; in the recession, it falls still further. Under the social, economic, and political conditions of the market late in the boom, capital is unable to utilize fully the capacity of its machinery and supplies to produce.

As capacity utilization declines while much new capacity is being installed, there is a point at which the old capital goods are put out of use, and the new made the basis of production. This point does not occur immediately with the installation of new capacity, but rather follows the collapse of the *stock market* and the rise of *bankruptcy* and *loan delinquency*. In the financial markets of the boom, the older capital had been represented as the leading part of the paper value of the corporation. As this paper value collapses, it is no longer important to use or maintain the older capital equipment, which is then sold off for what it will bring, rather than the inflated values used as a basis of borrowing previously. This results in the primary utilization of the new equipment as a basis for production.

The old equipment is also characteristically interwoven with the labor agreements about job structure, pay schedules, seniority, and so on, which labor wins during the boom. These agreements can only be abrogated and the new equipment efficiently utilized when unemployment has re-established labor discipline. The selling off of the old equipment obviously finalizes this process.

Much the same sort of process occurs in a reorganization of paper claims upon raw material flows and flows of intermediate goods and final inventories. In all of these cases, the heavy borrowing of the late boom allows inefficient economic organizations to survive temporarily, contributing to overall inefficiency and thus to the stagnation of output per man-hour. All of these inefficiencies tend to be corrected dramatically by a healthy dose of business failures.

Therefore, there are at least three fundamental underlying mechanisms for the business cycle as a socioeconomic process. The most important is the class struggle over the control of the social product. Lesser roles in the variation of investment are played by the technical and social conditions determining raw material and equipment use and labor productivity. These in turn are partly influenced by finance capital and the market for paper claims on real capital, outside the sphere of production itself.

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