Mortality and the Business Cycle: Some Questions about Research Strategies When Utilizing Macro-Social and Ecological Data

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Introduction

If one classifies submitted manuscripts according to two dichotomous judgments—substantively important vs. trivial, and methodologically appropriate vs. flawed—one can easily see that two of the four cells are much more problematic for editorial decision-making. The technically correct but trivial paper may invite editorial agonies regarding the possibly unduly subjective nature of the judgment of "trivial." The important but flawed manuscript, on the other hand, raises the hope that its publication may precipitate a useful debate which will advance the field, rather than only setting a precedent for the publication of numerous similarly flawed articles.

The purpose of this commentary on the Bunn article, "Ischaemic Heart Disease Mortality and the Business Cycle in Australia,"[1] is not to provide a detailed critique of its data analysis methodology and its interpretative reasoning; in part, that is done in a separate commentary, also included in this issue. Instead, I wish to use the article as a starting point for initiating a discussion about some issues of research strategy and research design, and the consequent limitations of these on permissible inferences from obtained results.

Discussion

A Complex Problem

The Bunn article follows a number of other recent publications, using a highly similar orientation to such macro-social and ecological data: these have dealt with heart disease mortality,[2,3] infant and maternal mortality,[4] general mortality,[5,6] alcohol consumption,[7] and psychiatric hospitalizations[8] (used optimistically as an indicator of the incidence of mental illness). Disturbing questions have been raised about the approach[9-11] and fundamental controversies have emerged, such as whether the associations are actually with a downturn or an upturn in the economy.[5,6] However, the prospects for a systematic debate of these issues appear small and, instead, we are likely to see more publications like the Bunn article which use this methodology uncritically and unquestioningly. Considerably more disturbing are the prospects of congressional committees[12] accepting wholeheartedly the interpretations and conclusions from these analyses, without realizing that the scientific community has not yet engaged in a thoroughgoing examination of the underlying methodology.

The use of the business cycle and related macro-social data in trying to account for the behavior of individuals is, of course, nothing new to the social sciences. However, it would seem that this approach has had its ups and downs in popularity[13-19] (outside of economics) and any chances of its thoroughgoing evaluation at any time were probably reduced by its temporary disappearance, once more. The classical article on the "ecological fallacy"[20] is about 30 years old and it only scratched the surface of the issues needing discussion. Within public health and epidemiology, critical scrutiny of the methods of ecological analyses are also rare and seldom deal with the full range of issues (e.g., Stavraky[21]).

In this commentary I would like to initiate and partly develop several interrelated arguments. The *first* one is that when facing the results of a macro-social or ecological analysis, the safest attitude for the reader to adopt is one of profound skepticism. In justification for this advice, I wish to cite some results from a recent study entitled "Commodity consumption and ischemic heart disease mortality, with special reference to dietary practices."[2] In this study, temporal changes in ischemic heart disease (IHD) mortality (age standardized, 35-64 age group) for England and Wales, 1950-1967, were related to consumption of various commodities. The highest correlation with male IHD mortality was −.93 with flour consumption; in contrast, the correlation with cigarette consumption was .28. Exploratory analyses with temporal lags of different lengths revealed that changes in the correlations stabilized at a lag of 9 years; at this lag, the correlation with flour consumption was −.92 and with cigarette consumption, −.33. First order partial correlations (at

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zero lag), with the effect of flour consumption removed, produced a partial r = −.30 with cigarette smoking. Among women, the results with flour consumption were quite comparable; however, the data on cigarettes revealed a correlation of .95 (or .81 with a 9 year lag) and a partial correlation of .66.

At first blush, the above results appear to be sheer nonsense. At second blush, the impression remains. There is no way we can take the data at face value and make some inferences about new powerful risk factor for IHD mortality, low flour consumption, or about the relative importance of low flour consumption compared to cigarette smoking. Even the modest claim which some make for the usefulness of such ecological data, i.e., that they are suggestive of new hypotheses or new avenues of research, leaves one in great puzzlement in this instance: How does this add to our present knowledge? What new avenues should we be pursuing on the basis of this? It is also disconcerting to contemplate how one might use these data if they were obtained at a time when we still had no information about IHD risk factors from prospective epidemiological studies. Would we start intervention trials designed to increase the consumption of flour?

One sometimes encounters the argument that even if a particular study has various methodological problems or shortcomings, internal comparisons of subgroups are valid because these subgroups share these shortcomings and differential results can therefore be trusted. On the basis of this argument, we might have a good deal of confidence in noting the differential results with cigarette smoking for men and women and in concluding that cigarettes are a problem only for women but not for men. This, too, is a conclusion which is quite unsupportable from current knowledge.

In the above study,[2] many other high correlations were obtained, some of which might be considered consistent with present knowledge (e.g., consumption of butter). However, the point is that the plausible as well as the implausible variables produced high correlations, but no guidelines about detecting the "spurious" correlations. It might also be noted that in a correlational analysis, many such high correlations produce serious problems when one starts certain data analyses, such as stepwise multiple regression or attempts to partial out effects of variables.

Potentially Misleading Analyses

At this point I would like to conclude that ecological analyses lead to results which, *by themselves,* are opaque, unhelpful, potentially misleading. However, I am embarrassed to have cited only one study in support of this assertion and so I will rephrase it: Ecological analyses can produce correlations as high as the mid .90s with variables, which to the best of our current knowledge, are likely to reflect spurious associations only. I would also like to note the recent curious article in which the validity of cigarette smoking as a risk factor for lung cancer is being denied.[22] It is instructive that in this somewhat perverse effort, the major line of argument—aside from highly specific methodological criticism of individual studies—is to cite selected ecological data which are not in full agreement with the cigarette smoking hypothesis, e.g., changes in lung cancer mortality in a particular country-period-demographic subgroup and corresponding changes in cigarette consumption. Finally, I would like to refer the reader to a different type of a literature, that dealing with urban ecology and schizophrenia.[23] A great many ecological studies have been published over the years and a great many hypotheses have been offered to explain the ecological distribution of the rates: social isolation, mobility, cultural change or cultural conflict, diverse social stresses, psychological frustration, self-selective in- or out-migration, and various class-related biases inherent in detection, diagnosis, and treatment of persons with mental health problems. Unfortunately, these ecological studies generate new hypotheses but do not help us to reject even a single interpretation in this ever-growing list of possible explanations.

The *second* line of argument which I would like to propose is that studies of the business cycle and mortality are properly seen as part of a larger public health effort to understand the contribution of psycho-social factors to disease. Such ecological and macro-social studies should be carried out, as much as possible, in the context of examining other known biological and psychosocial risk factors, rather than in splendid statistical isolation away from all of them. In public health, we are interested in accounting for the level of morbidity or mortality at any one time, and in changes in this level over time: all differences in level of morbidity or mortality across countries (or other aggregation units), all secular trends (similar or variable), all cyclical variations (large and small) are of interest and need to be explained. In contrast, the Brenner-type analysis performs various statistical adjustments on the raw data—such as removal of trends and some cycles, or standardization of amplitude of variation in other cycles—so that one can no longer tell what the residual phenomenon is which is being studied in relation to the business cycle or how large it is. In these analyses, for example, business cycle fluctuations may account for, say, 60 per cent of the variance in the residual IHD mortality: but what does this residual phenomenon mean and does it represent 10, or 5, or 1, or 0.1 per cent of the total disease phenomenon in which we are interested? Similarly, it is quite possible in these analyses to obtain a result where the business cycle accounts for 99.9 per cent of the variation in IHD mortality fluctuations in both of two countries, and yet provide no explanation whatever of why the rates in one country are double those of the rates in the other.

The above is not an argument for working with raw data only and against statistical adjustments. However, it is an argument for the need to consider explicitly and justify the rationale for various statistical adjustments, to fit this rationale into the overall context of what is currently known about the other biological and social risk factors for that disease, and to trace the consequences of such adjustments for the explanatary power of the various analyses.

Detrending Adjustments

The issue of detrending in the Brenner-type analyses is a particularly bothersome one, since no conceptual rationale is offered and since the potential (and the actuality) of the de-

trending adjustments distorting the basic data is a serious one. There would seem to be four types of occasions in which detrending may be under consideration.

- The first one is where a technical adjustment is necessary, such as representing the state of the economy in constant dollars rather than inflated dollars, or in correcting for changes in a diagnostic classification, such as going from the 7th to the 8th revision of The International Classification of Diseases (ICDA). Such detrending is easy to justify.
- The second type of occasion is when certain abrupt changes in the society may alter the meaning of the phenomenon, such as divorce rates after a major change in the law or economic data on auto accidents after no fault insurance. The need for detrending here still appears compelling but does need some thinking through of the rationale.
- The third type of occasion is when the onset of some environmental variable directly influences the rates, such as a new effective treatment reducing case fatality. Here detrending, at best, loses information since ideally we wish to represent the impact of the treatment, of the business cycle, and of the interaction, i.e., how the influence of the business cycle is affected by the introduction of the new variable.
- The fourth type of situation is where certain secular trends in the morbidity or mortality rates exist but we do not really know why. Here, the need for an explicit rationale for detrending is the greatest as well as the most difficult to come by. In the absence of a rationale and without a careful tracing through of the statistical consequences of detrending, the potential for a serious distortion of the results is great indeed.

In the report on infant and maternal mortality,[4] the raw infant mortality data look quite straightforward: relatively high and stable rates from the early part of this century until about 1937, then a steady decline until the early 1950s, and then a leveling off with some minor fluctuations, including a slight rise to 1963 and a slight decline after. The unemployment data for the same period show a pattern which bears no resemblance whatever to the infant mortality data. However, through the magic of detrending (apparently only a simple linear trend is removed, in spite of obvious better fit from a nonlinear trend), the high rates in the 1920s now become "low" rates and the slight elevation around 1963 now becomes a very "high" rate. Then with a little lag thrown in and some other manipulations, the fit with the unemployment cycle is beginning to "emerge". It is, of course, hard to know what it all means.

Lagged Analyses

The issues regarding lagged analyses are equally troublesome and equally in need of explicit rationale and justification. For example, in the study of commodity consumption, the correlation between IHD mortality and milk consumption was −.85 for men and −.81 for women, at zero lag (another curious finding, incidentally); by 9 year lag, the correlations became +.58 and +.50, respectively. Such instability of correlations depending on the lag is most disturbing. In the absence of any independent rationale for choosing a lag time, it strongly suggests the possibility that an investigator can influence the direction of his results. The scanning of the data in order to determine the "optimal" lag appears curious enough, but the inclusion of a minus one year lag[8,24] borders on the incredible: surely that must undermine and ridicule the investigator's own efforts to suggest unidirectional causal interpretations.

The issue of a proper lag analysis also appears to be the major component in the disagreement over whether it is the downturn or the upturn in the economy which is associated with an increase in pathology and mortality.[5,6] One can imagine that our congressional leaders would like to have that one settled before they contemplate policy and legislation! Eyer is correct in pointing out that the selection of a proper lag should be neither an à priori arbitrary decision, nor an à posteriori optimizing-of-results decision, but should be carried out with reference to relevant data from independent studies. However, his own efforts[6] to cite evidence justifying an extremely short lag for most stressful life experiences suggest that the accumulation of such independent evidence, guiding us firmly toward a particular lag period, will be long in coming. Meanwhile, the prudent investigator without an ax to grind would do well to simply display the full set of results obtained for the various lag periods examined.

Guidelines and Ground Rules Needed

In the *third* of interrelated arguments, I would like to suggest that we need to begin to systematically develop a methodological metatheory—a set of guidelines and ground rules—for comparing ecological and macro-social analyses with epidemiological studies of individuals. Some questions that need to be addressed: What are the comparative limitations of evidence coming from each type of studies? What unique contributions, if any, may be claimed for each type of study? How does one understand discrepancies in findings generated by the types of studies? What are the dangers of generalizing findings across levels of analysis? These are important issues which epidemiology and public health have addressed in the past only on an ad hoc basis as a particular study or finding generated some specific issue of interpretation. The need here is to approach the methodological aspects of these issues, not the philosophical ones; the latter would only precipitate once more a debate of the merits of the doctrine of reductionism in science.

It is instructive to consider briefly the current hot issue in cardiovascular (CV) epidemiology, the decline in CV mortality in the U.S. since about 1964-1968.[25-28] This decline has coincided pretty well with a reduced consumption of tobacco products, fluid milk and cream, butter, eggs, and animal fats and oil; over the same period, the consumption of vegetable fats and oil has increased. Decline in serum cholesterol has also been noted,[25,26] although it appears to be of more modest proportions. (The trends in control of hypertension also suggest a nationwide improvement in the picture,[25,29,30] although this is probably not influencing specific IHD mortality.[26]) Overall, the picture is very suggestive and clearly contradicts Bunn's introductory statement that "the post-1968 decline is particularly puzzling."

Nevertheless, the National Heart, Lung, and Blood Institute and most epidemiologists around the United States appear to be taking the stand that the decline cannot be definitively interpreted. Why? One reason is the felt need for

additional information on secular trends in: all the risk factors, incidence of first vs. repeat cases of IHD, and case fatalities. In addition, there is a vague need to find out about "all the other things that have been changing as well." Another reason is that while many things are going down (or up), not all of them are necessarily involved in contributing to the mortality trend. For example, Stamler[27] emphasizes the role of nutrition over smoking; but given the simple data on trends, it is difficult to agree with him, or disagree with him, with great conviction. Walker[28] has noted that there has been little change in the U.S. diet between 1909 and 1965; however, he misses the opportunity to point out that there is a disturbing lack of symmetry in the argument that the recent decline in IHD mortality is being related to dietary changes, but that the earlier rise obviously cannot be so related.

Careful Interpretation

We must also be cautious in interpreting the effects of broad social interventions which generally take place after an "alarming rise" or "alarmingly high levels" are observed. If many disease phenomena show cyclical or epidemic curves, or unexplained fluctuations, then the rate of the disease may well start to decline independently of, but coincidentally with, the onset of such interventions. Thus the impact of the publication of the Surgeon's General Tobacco Report or the American Heart Association recommendations regarding dietary changes in the mid 1960s may well be overestimated.[28] This point of societal interventions capitalizing on chance fluctuations is nicely described in Campbell's article[31] on traffic fatalities in Connecticut and the state's "successful" response to it.

The general point, then, is that we have a good deal of information regarding the circumstances of the recent decline in CV mortality. Nevertheless, the current stance in the scientific community is one of reserving judgment regarding the cause(s) of this decline. Since there is almost no independent information regarding the impact of unemployment on CVD, it would be most awkward if the ecological data on the business cycle were viewed with less, rather than more, skepticism.

There is no denying the fact that our current knowledge of IHD risk factors is still limited. Only about one-half of heart attacks in the population are said to be explained by the well-established risk factors.[32] Similarly, Blackburn[33] notes that "coronary risk factors already demonstrated within populations explain . . . half the variability in disease rates among populations." As a result psycho-social variables and macro-social phenomena remain a fertile and promising source of additional risk factors which might increase our explanatory powers.[34]

The primary strategy for detecting the promise of macro-social phenomena has been to show that the cumulative impact of the established risk factors is inadequate to explain group differences. Thus, CHD rates in Honolulu or Puerto Rico are much lower than in Framingham, even after adjusting for levels of risk factors.[35] Thus, Japanese-Americans retaining traditional cultural practices have a lower CHD prevalence than Japanese-Americans adopting practices of their new homeland, even after adjusting for levels of risk factors.[36] Such results suggestively implicate "membership in the culture of Puerto Rico" or "membership in traditional groups" as variables worth further investigation. What the underlying dynamic processes actually are may still take a long time to uncover.

In the absence of the above strategy, ecological differences by themselves offer much less promise of detecting new and promising risk factors or macro-social processes. For example, the atlas of U.S. cancer mortality rates often tends to reveal fluctuations in rates which either remain inscrutable or are quickly resolved by noting local variations in known risk factors, such as cigarette smokers exposed to industrial carcinogens. The promise of this lesser strategy can be enhanced, however, if we pay attention to the type of the ecological variable with which we are working. Aggregating individual data over spatial or temporal units does not automatically transform the aggregate into a macro-social variable which has emergent and unique properties. For example, aggregating data on divorce for city census tracts and relating them to census rates of psychiatric treatment is very likely only an indirect way of looking at the individual association between divorced status and treatment; per cent divorced may in no way represent an emergent, macro-social property of the census tract. On the other hand, aggregating juvenile offense rates over census tracts and relating them to psychiatric treatment among elderly may well reflect something in the residential environment of the elderly, a genuine social-context variable. We need to be much more sensitive to the conceptual properties of our aggregate variables and to the significance of the units over which the data are aggregated.

There is a conspiracy of silence regarding one obvious fact of ecological analysis: the investigator works with the data which are made available to him, and these are seldom collected and aggregated for the research purpose to which they are being put. For example, administrative units of spatial aggregation may average out a good deal of environmental variation which assorts itself in a quite different way. On the other hand, many investigators in this field have worked with higher units of aggregation than they needed to. For example, there is a good deal of local and regional variation in unemployment rates which gets wiped out in using annual U.S. rates.[37, 38] Taking advantage of local and regional variations in the business cycle as well as in disease rates represents a promise of a more refined ecological analysis which has gone largely unfulfilled. This promise is an important one, since if the institution of more precise and refined analyses leads to a shrinkage, rather than enhancement, of the size of the effect, then one has reason to worry that the earlier cruder analyses picked up only a spurious relationship.

At this point I should like to come back to the Bunn article and note two promising innovations over the earlier Brenner report on heart disease mortality.[3] One is the author's desire to make certain deductions from his "model" and thus make some predictions about the future or about some other data. This is crucial since so much of the earlier approach represents a highly elaborate and encapsulated system of after-the-fact reasoning with no attempt at independent verification.[7] Unfortunately for Bunn's model, his

prediction does not appear to be supported, at least in the U.S. data: the unemployment data for the last decade indicate a considerable upward slope and the IHD mortality, an uninterrupted downward slope.[6]

The second innovation comes in the use of general practice prescribing data. Clearly, there is a need to include in the business cycle data of this kind so that we may have as complete a picture as possible about many other potentially relevant coincidental fluctuations and secular trends. However, the results again are more troublesome than helpful for Bunn's overall interpretation of his results. Essentially, if rates of prescribing are to be interpreted as an indicator of "stress" and thus as a variable which mediates between the unemployment rates and IHD mortality, then one should see the magnitude of the path coefficient between unemployment and mortality to be roughly equal to the product of the other two coefficients.[39] Table 3 of the Bunn paper,[1] however, reveals that the path coefficients between prescribing and mortality are either zero or negative. This is a striking lack of support for his interpretation.

I must emphasize that the promise of the two innovations introduced by Bunn is that he makes his interpretations vulnerable to disconfirmation—which is, of course, crucial to scientific progress. In actuality, the additional results are a small comfort to his interpretation.

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