Green College Lectures

Social factors and disease: the sociological perspective

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Much of the work of medical sociology is concerned with how disease and illness are defined and managed,[1] but with all this activity little, particularly in Britain, has been focused on the facts of disease and suffering (M Jeffreys, unpublished paper). I will restrict myself to the neglected issues of the aetiology and course of disease as I believe that they present sociology with the toughest intellectual challenge. In dealing with these issues it is not unreasonable to expect sociology to take a grand perspective, and Richard Warner's recent impressive inquiry *Recovery from Schizophrenia* does just this. It argues for important social influences both on the medical management of this potentially chronic and highly handicapping disorder and on its course. At its core is a claim that the recovery rate from schizophrenia was lower during the great depression in the 1930s than either earlier or later, and particularly that the dramatic changes in the care of schizophrenic patients in the 1950s were a consequence of the extremely low rates of unemployment and a postwar demand for labour.[2]

Macrolevel effects and causal mechanisms

Such points about macrolevel effects are not likely to be convincing, and it will usually be necessary also to have evidence of mechanisms at an individual level. This is illustrated in our lack of understanding of the failure of social class differences in mortality to narrow despite an appreciable general decline in mortality this century. Recent research begins to make an impressive case that real differences are entailed,[3-5] but we are far from understanding the mechanisms.

Let me illustrate such mechanisms. Follow up inquiries of schizophrenic patients have established that whether or not a patient relapses with florid symptoms is highly related to the emotional climate in the home to which he returns. The original insights emerged from an inquiry focused on the impact on the course of the disorder of characteristics of the living group, but subsequent studies developed methods of measuring the actual emotional climate in the home.[6] It is just this kind of detailed research concerning the individual and his milieu that enables us to begin to interpret the broad picture presented by Warner's review. As he argues, it might well help to explain the better outcome of schizophrenia in developing countries. Recent research in India has shown a much lower presence of critical comments and emotional overinvolvement by relatives about such patients than in Europe or the United States, but where such conditions do occur in India the relapse rate is also high.[7]

But, given the wish to establish such mechanisms, research dealing with the characteristics of the person's milieu will not always be enough. At no point in the schizophrenic research was any weight placed on exploring what such experiences meant for the patient; progress was made because it might be argued that schizophrenic patients tend to have a common and unique characteristic—their sensitivity to emotional arousal of any kind. Possibly this approach to vulnerability will be found for other conditions, but it is likely to be scientifically stultifying and chilling on humanitarian grounds for a social scientist to start aetiological research only with assumptions of this kind. An approach is required than can deal more directly with the meaning of experience, particularly potential environmental stressors and possible protective factors.

Measurement of meaning

To simplify and assume that meaning emerges from our plans and commitments, it is when we see how some incident relates to our plans and purposes that we are most likely to be able to assess its meaning for us. Emotions are crucial since they are concerned not only with the way the world is but with how it ought to be—that is, with hopes, desires, intentions, and commitments.[8] They are particularly caught up in the ways we have invested a sense of our various selves in the world, and the feelings of self worth and self esteem that arise from such investments.[9] The methodological question is how to collect such material in aetiological research.

Difficulty arises when we try to extend the long concern within sociology with meaning in the context of everyday life to studying causal processes taking place over long periods of time, or when there is a discontinuity in the level of the systems, as with the example of the British economy and the course of schizophrenia. Simply to ask about the meaning of experience may bring immense problems. One is circularity: it would hardly be surprising, for instance, if respondents with clinical depression reported that they had more often felt depressed by their recent experiences than respondents without clinical depression. A possible solution is to recognise that the meaning of an event and the emotion it arouses are more correlated with the objective features of a person's biography and current circumstances than many seem to allow. Given that much is "built in" in this way, a more "behaviouristic" approach concerning measurement of potentially stressful experiences is possible, in the sense that we can rely on the knowledge of the context rather than on subjective reports for understanding meaning and the likely emotional response.[10]

Given this, one way forward is still to talk to people about their lives, but to use ourselves as a measuring instrument to take account of context to assess a person's plans and commitments relevant to a particular situation. In this way we can create a picture of the likely meaning of an occurrence. It is this vital step that many in "harder" disciplines such as epidemiology find difficult to accept—that is, that the investigator can be his own effective and sensitive measuring instrument so long as he takes care over the calibration and possible bias.

In the late 1960s such an approach to meaning was developed in

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London and embodied the life event and difficulties schedule, or LEDS. Detailed questioning—obtaining a narrative account of relevant behaviour and circumstances, particularly concerning relevant commitment and plans—is used as a basis for assessing the likely meaning of an event.[11 12] Trained raters arrive at these consensus judgments and are kept blind to the knowledge of potentially biasing information by the original interviewer, who describes only the event and its context.

Specific and general aetiological effects

It has been possible with this approach to establish links between the onset of various disorders and specific types of life events. For clinical depression only events with considerable long term threat appear to be concerned—severe events. But there are various kinds of severe event and on the whole only a certain type is involved—events concerning loss and disappointment.[13] The size of the aetiological effect of such events is considerable. But with anxiety or phobic states, although severe events also play an important part, it is danger—that is, the threat of future loss—rather than actual loss or disappointment as in the case of depression that is critical[14]; and in schizophrenic patients any event capable of producing emotional arousal appears to be sufficient, including joy or excitement.[15 16]

These are specific aetiological effects. Nevertheless, there is also some evidence for more general effects—that several different diseases may relate to a broadly comparable set of stressful experiences. Severe events capable of provoking depression are also important for the development of some physical disorders. They have been found, for example, to occur commonly before the onset of multiple sclerosis.[17] In a study of patients having had an appendicectomy those found to have an inflamed appendix had had only a modest increase in severe life events before the onset; but those who had had a non-inflamed appendix removed had, like patients with depression, had a much increased rate of severe events.[18]

But despite this evidence for a general aetiological effect in physical disorders there is also a good deal of evidence for specific effects. A study of patients with gastrointestinal disease has repeated the finding of the appendicectomy research about the importance of severe events for those with a "functional" condition but has also shown that those with a condition with a clear organic basis, such as a duodenal or peptic ulcer, tend to have a different type of life event—one characterised by goal frustration.[19]

There is a second and equally important type of aetiological factor. Crucial and complementary to the notion of the quality of events in terms of meaning is that a person may be more or less vulnerable in terms of a propensity to respond after a stressor. This mediating vulnerability may be psychosocial, as, for example, the tendency of women who experienced deficient maternal care in childhood to react to losses in adulthood with clinical depression.[20 21] Vulnerability helps to explain why only a minority succumbs to a particular disorder after a specific major life crisis. The role of social factors in vulnerability is particularly clear in relation to clinical depression, when internal and external resources that affect coping with the crisis are the strongest candidates. Continuing emotional and practical support from core relationships and self esteem seem to be crucial.[22]

A factor such as social support may have widespread relevance—for example, it may play a part in low birth weight, and the provision of emotional support during pregnancy may be one way of reducing the social class differences in prenatal mortality.[23] But it is necessary to emphasise the issue of specificity for vulnerability factors as well. There is no evidence that support protects against anxiety or phobic attacks after a danger event,[24] and the special role of support in schizophrenia has already been noted.

Other approaches to aetiology

A recent review is critical of the potential narrowness of this approach in terms of the study of meaning and maintains that lack of sleep, physically taxing work, a hazardous environment, and the like may have an impact on health independently.[25] But there is no reason why such environmental factors cannot be included in the aetiological model I have outlined. At times, for example, they are likely to interact with effects mediated through meaning. For example, accidents are a major cause of death among children and are a significant contribution to the class differences in mortality, and undoubtedly straightforward environmental factors such as hazardous living conditions play a part. And yet there is also evidence that serious accidents to children are related to depression in mothers and that the risk is increased after the onset of even mild depression. Moreover, the risk increases outside the home and whether or not the mother is present.[26]

I have so far ignored the most obvious candidate for sociological interest in disease: the consequences of long term behaviour, often referred to as lifestyle—cigarette smoking, alcohol consumption, and nutrition are examples. Most risk factors of this kind have important social correlates. Wider societal processes may be concerned—such as the effect on consumption of the price of alcohol or licensing hours and thus the risk of cirrhosis of the liver—and the behavioural patterns may be in part a response to adverse social experiences. There has been a good deal of research into such patterns of behaviour—for example, the documentation that the use of heroin among American servicemen in Vietnam did not usually persist on their return to civilian life.[28] But, despite much promising work, we are only beginning to integrate such research with that into stress and disease.[28 29] It is important that this is done.

One of my main themes has been the need to deal with specific diagnostic groupings, but there have been arguments for a quite different, general, approach.[25] Indeed, in the results reviewed earlier there is evidence for such an effect in that the stressors producing depression appear to raise the risk of several other physical disorders as well. My worry is that to study all or most disorders in a population would require large numbers and considerable resources, and this would tend to push research back into relying on demographic type measures and the somewhat vacuous speculation of so much work. I suspect that some compromise will inevitably emerge, with studies beginning to examine a range of disorders with apparently similar aetiological factors, and in this way a more general perspective will gradually be approximated while the strengths of the alternative approach will be retained.

Conclusions

I have therefore argued for a particular sociological approach to aetiology, what might be called intensive epidemiological, by emphasising its adaptability. But the very flexibility of the approach raises the question of disciplinary allegiance. Already the necessity for compromise is clear. While, reasonably, sociologists see the individual as psychologically uncomplicated and look for complications at a system level, this will not do for the study of disease. This does not mean that differences should not be encouraged. For psychologists research on depression has led to an emphasis on how a person who becomes depressed is in some way distorting experience by maladaptive thoughts—that is, by overreacting in terms of how most people would respond. The alternative, more sociological perspective, quite compatible with a cognitive view of the origins of depression, has underlined its naturalness, emphasising that it is understandable, given evolutionary based propensities to react with specific emotions to particular types of experience and given the degree of adversity inherent in a person's life.[30] It is not that one must choose between such perspectives: it is a matter of getting the right emphasis. But, though psychologists tend to underestimate the sheer unpleasantness of many people's lives, sociologists need to take into account what may be termed personality differences. There is now evidence, for example, that childhood experience of indifference or rejection by key caretakers relates to a higher rate of clinical depression among women in adult life and there is also some understanding why this should be so.[20 21 31]

One critical chain of influence is environmental: early experience of indifference on the part of caretakers is often followed by, say,

premarital pregnancy and this in turn by the kind of current social difficulties found to raise risk of depression. In addition to such "external" influences, however, there begins to emerge evidence for the importance of continuing styles of attachment that have their roots in early experience, such as behaviour that tends to make it more difficult for a person to develop supportive relationships. But ever here it is possible with profit to contrast personality with the more sociological idea of character, using character to describe the feelings of self worth that are the tangible results of the success we have made of the roles and tasks we have accepted or had thrust on us.[32] It is highly likely, for example, that current successes, say, in the roles of wife and mother (entailing character) can often neutralise the predisposition to depression arising from early adverse experience (this predisposition involving personality).[33-35]

I end therefore on a note of optimism. Nevertheless, the contribution of sociology to aetiological research is bound to overlap with that of other disciplines, and research will probably prove most productive when carried out in an interdisciplinary setting. The potential rewards for sociology in such collaboration are many—not least that the study of disease and disorder can tell us a good deal about the working of social configurations. In this sense medicine promises to bring as much to sociology as sociology to it—and it takes no special knowledge of sociological theory to realise that such reciprocity provides a sound basis for future collaboration.

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I have had an anecdotal report of two patients who underwent a prostate operation and soon after developed Parkinson's disease. Is there any association between general anaesthesia and Parkinson's disease?

There is no causal relation between general anaesthesia and Parkinson's disease. By the time the disease shows its earliest symptoms, 80% of dopaminergic neurones of the nigrostriatal pathway have been depleted; this subclinical process takes many years. Patients often seek a cause for idiopathic illnesses and it is therefore not uncommon for patients to claim a causal role for some non-specific trauma, anaesthetic, or infection that seems to precede the appearance of symptoms. Although severe anoxia and hypoperfusion states that may complicate anaesthesia may cause diffuse brain damage, parkinsonism is not the outcome of such occasional incidents. I assume that chronic neuroleptic drugs have been excluded.—J M S PEARCE, consultant neurologist, Hull.

Is there any contraindication to patients with manic depressive psychosis flying in passenger aircraft?

Obviously a patient in the manic or depressive phase of such an illness would be subject to the airlines' regulations governing the carriage of mentally ill patients.[1] Of more scientific interest is the possibility that air travel may precipitate a relapse in manic depressive patients in remission. Jauhar and Weller reported that in a consecutive series of patients admitted from Heathrow Airport depression was more common in patients flying east to west and hypomania in those flying west to east.[2] Although it was not reported whether the patients' illness preceded or followed the flight, it suggests that crossing time zones may specifically predispose manic depressive patients to relapse over and above the general effects of the stresses of air travel. Jauhar and Weller suggested that this may be due to a disruption of the circadian pattern of melatonin secretion. So far as I know no controlled prospective study has been undertaken of the effects of air travel on manic depressive patients in remission. Given the lack of reports and the volume of air passenger traffic, the risk of air travel causing relapse in an individual patient is probably slight.—PAUL T LELLIOTT, lecturer and honorary senior registrar in psychiatry, London.

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A 60 year old patient is developing a considerable dorsal kyphosis. She had an early menopause but has enjoyed excellent health. Both her parents and two aunts also suffered considerably from this condition in their late 70s. What investigation, if any, should be performed and what advice should be given?

It is essential that the patient should be investigated to make certain that she, like her parents and aunts, has postmenopausal osteoporosis and that there is no other underlying disease despite her apparent excellent health. She requires radiographs and full biochemical screening. If the diagnosis is clearly postmenopausal osteoporosis a combination of an oestrogen and a progestogen with a calcium supplement and regular exercise is the best method known for preventing the progress of her disease. It is important also to control intake of drugs and nutrients that interfere with effective utilisation of calcium, such as high protein intake, smoking, and too much alcohol.—LESLIE KLENERMAN, consultant orthopaedic surgeon, London.

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