Control of destiny and health: Towards a more effective preventive medicine

Prof. S. Leonard Syme
School of Public Health, University of California, Berkeley/USA

There are three major failures of epidemiology: our difficulty in identifying disease risk factors, our difficulty in helping people change their risk once they know about it, and the fact that, even when people do change, new people keep coming to take their place. If epidemiology is to be useful in developing a preventive approach, it will need to study the community and the population. The most impressive patterned regularity in morbidity and mortality rates is social class. As one moves down the social class hierarchy, one has less control of one's destiny. Suggestions are made how control of destiny can be improved.

For over 30 years, I have done research and taught students in the field of social epidemiology. I had always thought that epidemiology was the basic science of public health and that it provided the fundamental and necessary scientific basis for preventive medicine. The reasoning behind this belief is straightforward: if we epidemiologists could clearly identify a risk factor for disease (such as smoking or obesity or a deficient diet), we could then share this information with physicians and with people in the population. This sharing, it is argued, would result in people changing their behavior to lower their risk of disease and all would then be fine.

Things have not worked out this way. In fact, it might easily be said that we epidemiologists have failed in our mission and that we still need to develop a more appropriate epidemiology that can lead to a more effective preventive medicine.

There are three major reasons for this failure of epidemiology. The first reason is that we have done such a poor job in identifying risk factors for disease. As an example of this failure, consider our achievements in the area of coronary heart disease - the disease that has been studied most aggressively for over 40 years with the highest level of financial support that the world has ever seen. During these 40 years of incredible world wide effort, a large number of seemingly important risk factors have been identified. The three that everyone agrees on are cigarette smoking, high blood pressure and high serum cholesterol. Dozens of other risk factors also have been proposed but not everyone agrees about them. Included here are such risk factors as obesity, physical inactivity, diabetes, blood lipid and clotting factors, stress, and various hormone factors. When

all of these risk factors, combined, are considered together, they explain about 40% of the coronary heart disease that occurs.

How is it possible that after 50 years of massive effort, all of the risk factors we know about, combined, account for less than half of the disease that occurs? Is it possible that we have somehow missed one or two crucial factors? While this is of course possible, it should be noted that the relative risk of the new risk factors would have to be enormous to account for the other 60% of the coronary heart disease that occurs. It seems not very likely that we would have missed one or two risk factors of such enormous power and importance. And, it must be said, our record of success in the area of coronary heart disease is one of the very best; the results for other diseases are far less impressive.

I do not mean to suggest by these remarks that the risk factors we have identified are unimportant. They **are** important and they have been useful in the prevention of coronary heart disease, but, clearly, there are other very important issues involved that we do not yet understand.

The second failure of epidemiology is that even when we do identify disease risk factors, it has proven very difficult for people to make changes in their risk behavior. An example of this problem is provided by the *Multiple Risk Factor Intervention Trial (MRFIT)*. This project was an experiment designed to reduce the death rate from coronary heart disease in the United States. It was to be the largest, most ambitious and most expensive experiment ever designed to see if by getting people to change high risk behavior, we could reduce the coronary heart disease death rate.

This experiment, we thought, was soundly based on public health principles. It was, we thought, the logical extension of twenty years of previous epidemiologic research showing that coronary heart disease risk was increased among people with high serum cholesterol, high blood pressure, and who smoked cigarettes. The idea was to find people in the top 10% risk category in the nation and get half of them to lower their risk, while the other half would simply be sent back to their own doctor. A straightforward, perhaps obvious, approach to deal with the *number one* cause of death in the United States.

Unfortunately, the experiment did not work and it is important to review this failure because we continue to follow the *MRFIT* logic in much of our work todays and, in my opinion, our current efforts will not work either.

As I indicated, *MRFIT* was a massive effort. The statisticians told us that we would need to recruit 12,000 high risk men into the trial in order to achieve statistically significant results. To find these 12,000 men, we had to screen almost 1/2 million men in 20 cities across the United States. We had to eliminate

women from the study because their rate of coronary heart disease is still too low, and, if they were included, the sample size would have had to be increased two to three times. The study took 10 years from beginning to end and cost over $ 140 million but we thought it was worth it if we could, by this work, reduce the death rate from coronary heart disease.

The reason that this trial is so important for us is that the 12,000 men we chose were a highly select group of motivated and health conscious men. We put them through two grueling medical exams taking over 3 hours, we had them complete hundreds of pages of questionnaires, and we repeatedly warned them of the terms involved: they had to be willing to change their diets, take pills for blood pressure if necessary, and stop smoking cigarettes. They were warned that if they were selected they would have to come to the clinic as often as two to three times a week at the beginning, that their families would be expected to come in from time to time, and that this would go on for six years. They were cautioned that if they were chosen, they would be randomly assigned to work either with us at the clinic or with their own doctor and that, if that was not agreeable, they should not volunteer. One more thing: from the group that agreed to join us, we on the staff rejected some because we did not think they would be good participants. So the men finally chosen for the trial were highly informed, highly health conscious and highly motivated, the kind of people all of us wish we could work with all of the time but which, of course, rarely happens.

Well, as I said, the trial failed. After six years, the group that worked with us did not achieve a lower death rate of coronary heart disease than the control group. This is not to question the importance of serum cholesterol, physical activity, cigarette smoking and high blood pressure as risk factors for coronary heart disease. These risk factors clearly have been shown to be important in increasing the chances of developing coronary heart disease. What I **am** criticizing is that so few people changed their behavior. In spite of the fact that they were highly motivated and that we worked very hard with a wonderful intervention plan.

We used all of the best health education techniques, we had a large and well-trained staff, and we had six years to work with each man. Some would argue with my statement that so few changed behavior. In fact, 42% of the smokers stopped smoking after six years. This is probably the best record ever achieved in a smoking cessation programme. But 58% continued to smoke. Of the men with hypertension, about half had it under control at the end of the trial, but half did not. Our record in reducing serum cholesterol by dietary intervention is probably not worth even mentioning. We achieved a 6.7% reduction in serum cholesterol levels. So, some changes took place but in this group it should have approached 100%. This group of men was selected in the best way we know to

be perfect compliers. The limited success we did achieve is, I argue, probably the best we can ever hope to achieve in one-to-one intervention programmes. It was the high water mark. The message from this extreme example is clear and well-known: it is very difficult for people to change high risk behaviors even when they really want to and even when we do all the good things we can to help them.

The third failure of epidemiology is that even when people do successfully change their high risk behaviors, new people continue to enter the at-risk population to take their place. For example, every time we finally helped a man in the *MRFIT* project to stop smoking, I used to think that, on that day, one to two children in a school yard somewhere probably were for the first time taking their first tentative puffs on a cigarette. So, even when we do help high risk people to lower their risk, we do nothing to change the distribution of disease in the population because, in one-to-one programmes like *MRFIT*, we have done nothing to influence those forces in the society that caused the problem in the first place.

We epidemiologists tend to look at individuals in order to find causes of disease even though it is clear that this will not be helpful in understanding the distribution of disease in the population. This point was forcefully made at the turn of the century by EMILE DURKHEIM, the French sociologist. DURKHEIM studied suicide - a behavior that clearly is the result of problems experienced by individuals. Indeed, suicide might be considered as one of the most personal and intimate behaviors possible. In spite of this, DURKHEIM noted that suicide rates exhibited a patterned regularity over time and place. Thus, he said, suicide rates are consistently higher in certain countries and in certain groups, over time, even though individuals come and go from these groups. If the causes of suicide are rooted in the individual, how can it be that there exists a patterned regularity in groups? DURKHEIM reasoned that there must be something about the group that somehow promotes a higher or lower rate in the group. This factor would not, of course, account for **which** individuals in the group committed suicide but it would account for the fact that the **rate** in the group was consistently high or low.

The value of this approach is that it can lead to a more effective preventive medicine. This is not to deny the importance of working with individuals one at a time: this clearly is worthwhile and important. But it is as least as important to recognize that this approach is very limited because it does nothing about those forces in the society that cause our problems in the first place and that will continue to provide a fresh supply of at-risk people, forever. If epidemiology is to be useful in developing a preventive approach, it will need to study the

community and the population. As matters now stand, almost all epidemiologists study large numbers of individuals in communities. This is not epidemiology. It is clinical medicine in large groups.

How did this unfortunate circumstance come to pass? One reason is that the clinical tradition runs so strong that it has come to dominate not only clinical medicine, but epidemiology and public health as well. In epidemiology, we typically use a **clinical** classification of diseases in our research. Thus we study diseases such as *heart disease*, *cancer*, and *AIDS*. These are clinical conditions that are of concern to clinicians - to those who must work with individuals on a one-to-one basis to treat or control their disease.

Heart disease, *cancer*, and *AIDS* are not public health problems, they are clinical problems. And yet much of the funding of epidemiologists and much of the way epidemiologists organize themselves nevertheless is centered around these clinical disease categories.

Let us think about the approach that epidemiologists used in the control of infectious diseases. That approach to infectious disease was organised around such diagnostic categories as *water-borne*, *food-borne*, *air-borne*, and *vector-borne* conditions. This diagnostic classification scheme is of little use in clinical practice, since it does not give any insight into treatment possibilities. But the job of the epidemiologist is not to treat individuals anyway. What insight this classification scheme **does** provide is to target those aspects of our environment that are generating disease and it suggests at what points in the environment our interventions might be deployed. This cannot be done with words such as *coronary heart disease*, *cancer*, and *AIDS*.

In short, I think epidemiologists have subtly adopted a disease classification scheme from the clinical model that inevitably focuses our attention on clinical states of health and that inevitably draws our attention to the affected individual. When we **do** consider social, community and environmental forces - and, of course, we do so to shed light on the individual and his health. The focus is not where it ought to be - on the social and physical aspects of our environment as objects of concern in their own right.

This appeal is made, interestingly, about 450 years after PARACELSUS brilliantly made the same point in his study of miners and metal workers. At the time he was studying their working conditions, the main causes of diseases were said to be internal to the individual and a result of an imbalance of bodily humors. PARACELSUS explained that miner's sickness was a disease of the lungs that also produced stomach ulcers and that both were a result of the air that miners breathed and of exposure to minerals taken in through the lungs and the skin.

Medical treatment of affected individuals was, of course, important but prevention required a totally different approach. PARACELSUS was not attended to at the time he wrote and, unfortunately, not much progress has been made today in understanding his message. I have two thoughts about why it is so difficult to think in these terms.

As I indicated earlier, the first possibility for explaining why we find it difficult to look at the social environment as a determinant of health, is that the clinical tradition is so pervasive, it overwhelms all other approaches. In the United States, it certainly has overwhelmed the *National Institutes of Health (NIH)*. One can apply for money from the *NIH* to study arthritis and metabolic disease, heart disease, cancer, and eye disease, but one cannot request funds to study health from the *National Institutes of Health*. Only diseases of clinical relevance are funded. I have nothing against categorical support but I do question why almost all the money goes to clinical categories and not to public health categories. Of course, it does not help that we do not have a well-reasoned and useful set of categories to offer.

The second possibility is more subtle and perhaps even more important. It may be that a community approach to infectious disease is easier to develop because most of us are exposed to infectious agents whether or not we want to be. A contaminated water or food supply puts all of us at risk, as does toxic air or infected mosquitoes. It is clear that public health agencies should look after these things on behalf of us all. This clearly is a public health issue. In contrast, the way we eat, drink, smoke, drive, sit, run and work can be seen as being our own affair and not anyone else's business. These behaviors, it could be said, are private matters and are not the province of public health concern. In this view, we are responsible, each of us as individuals, for the heart disease, cancer, and AIDS that we get. It is my opinion that this line of reasoning is one of the prime explanations for our current neglect of a social environmental approach to health promotion and for our adoption of a clinical, one-to-one perspective, including our adoption of a clinical disease classification scheme.

What classification categories do we have today that are equivalent to those of *water-borne*, *food-borne*, *air-borne*, and *vector-borne*? We cannot come up with a very long list. What we need are categories that organize our knowledge in appropriate and relevant ways. We have in epidemiology not devoted much attention to this issue. One approach might be to think about a system based on risk factors, since that is at the heart of the problem. How about *cigarette smoking diseases*? The trouble with this concept is that, as SYLVIA TESH has noted, we then continue to focus attention on smoking as an individual problem when what we probably really intend is to focus on tobacco as the issue.

Our determined focus on the individual has made it difficult to understand several important issues. One such issue is to explain the large, dramatic decline in death rates from coronary heart disease in a number of countries where the rate used to be very high. In the United States, coronary heart disease mortality rates have plummeted over 40% since 1968. Many people have tried to explain this decline in terms of lower smoking rates, better control of hypertension, lower fat diets, increased levels of physical fitness, better medical care of patients with coronary heart disease, and so on, but none of these explanations have been satisfactory. In fact, all of the explanations considered together account for less than half of the decline. But far more puzzling is that **all** causes of death have declined at about the same level (with the exception of smoking related cancers). We can perhaps try to explain the decline of mortality from one or another specific disease, but it is difficult to explain the decline of **all** diseases. Our vocabulary and training do not permit an easy explanation for this problem.

Another example, as noted earlier, is that disease-specific risk factors such as serum cholesterol, smoking and blood pressure account for only some of the diseases they are supposed to explain. On the other hand, certain psycho-social risk factors such as social class and social support are related not just to one or two diseases but to virtually every disease we know about. In a paradigm where specific diseases have specific causes, this generalized relationship does not make sense. One way to think about this is to say that certain psycho-social factors compromise the body's defenses against disease and that this makes people susceptible to disease in general, while disease-specific risk factors determine **which** disease a person gets. This hypothesis may be wrong, but if it is, we need some other credible explanation for this phenomenon.

Some have argued that environmental interventions to clean water and food are appropriate things for public health interventions but that it is inappropriate for public health professionals to interfere with the free choice of individuals in deciding how they want to live. As individuals, all of us ultimately **are** responsible for our own health and our own behavior. But it is naive to think that we are free agents in this. All of us are influenced by forces in the community that shape our choices and preferences. When we go to the large food market, it is almost always true that the bad foods are at eye level, badly labelled, and reasonably priced, while healthy foods with helpful labels are tucked away on remote shelves and are more expensive. Do we really have a free choice in that circumstance? The way in which food market people arrange foods slants that choice in certain directions. We would have more of a free choice if both good and bad foods were placed side-by-side at competitive prices. How about the teenager being bombarded in subtle and not so subtle ways about the attractiveness of smoking?

That teenager really does not have a free choice until we in public health provide an equally compelling argument against smoking. The environment at present is still pro-smoking and we need some good environmental interventions to redress that imbalance. When you enter a 2, 3, or even 4 story building, at least in the United States, it is easy to notice that the elevator is always placed in the center of the building and that it often has music, carpeting, and soft lighting. The stairs, if you can find them, are at the end of the building with bare floors and harsh lighting.

Just as bad water and food affect all of us, unhealthful forces in our society influence our choices and opinions and thereby affect us all. It makes no sense to ignore a contaminated water-supply and instead, tell people to boil their household water supply. It makes no sense either to urge people to change their unhealthful behaviors when we leave untouched those forces that prompted much of that behavior in the first place.

Some have suggested that a community approach to health is paternalistic or dictatorial. In my view, the type of approach I am suggesting gives people an opportunity to make real choices among alternatives.

To this point, I have discussed the three major failures of epidemiology: our difficulty in identifying disease risk factors, our difficulty in helping people change their risk once they know about it, and the fact that, even when people do change, new people keep coming to take their place. I have emphasised the importance, for an effective preventive approach, of a community approach to health and disease. It is, of course, easy to talk about the need for a community approach but it is not as easy to see how we might in fact go about doing this type of work. I think the first thing we need to do, following DURKHEIM, is to take advantage of patterned regularities in morbidity and mortality rates. The most impressive patterned regularity of all is social class. Everyone knows, and has known for hundreds of years, that people lower down in social class have higher rates of virtually every disease and condition. In spite of this universal recognition, we know almost nothing about the reasons for this phenomenon. The list of possible explanations is long and well-known. It includes poverty, bad housing, unemployment, poor nutrition, inadequate medical care, and low education. We do not know the relative importance of these various factors because we do not study social class. Social class is of such overwhelming power that we epidemiologists, in our research, typically *«hold it constant»* so that we can study other things. If we did not do this, social class would swamp all other factors and we would not be able to see the role of any other issues. In consequence, we know virtually nothing about the various subcomponents associated with social class.

But there is another, even more important, reason for our failure to study social class. This reason is that we do not feel that there is anything that *can be done about it*. Social class, we say, is a product of vast historical, economic and cultural forces and short of revolution, it is not something one targets for intervention. So we give up and instead urge people to lower the fat content of their diets. The problem with the view that there is nothing to be done about social class is that it is based, not on facts, but on speculation. If research were to show that people in the lower social classes had higher rates of disease because they were poor. I might agree that interventions would be difficult. But we have no evidence that lack of money is, in fact, the major culprit and, without knowledge, it seems premature to conclude that social class is too difficult to consider or deal with.

Allow me to offer an example of how this problem might better be approached. A former student of mine, Professor MICHAEL MARMOT of the University of London, has shown in his study of British civil servants, that those at the very bottom of the civil service hierarchy have heart disease rates four times higher

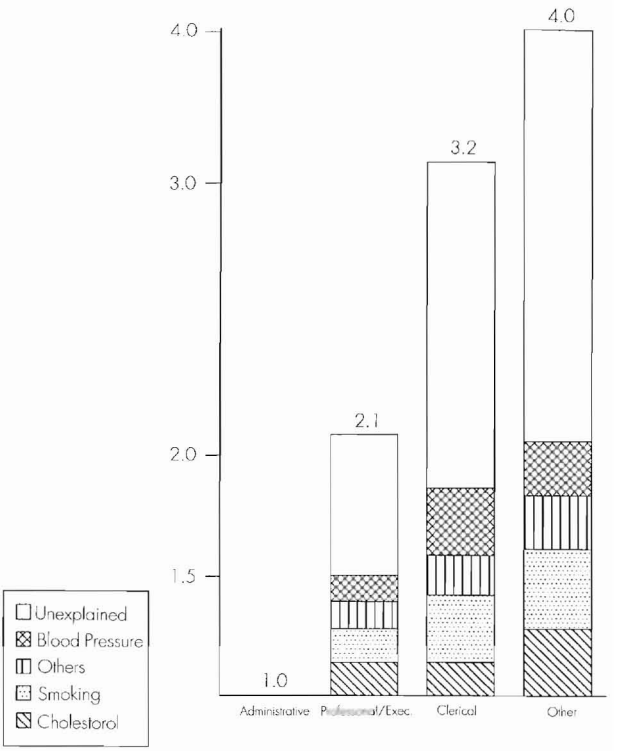

Fig. 1: Socioeconomic status and relative risk of coronary heart disease in male civil servants

than those at the top *(Fig. 1)*. After adjusting for such heart disease risk factors as blood pressure, serum cholesterol, smoking, physical activity, and so on, the difference between these groups is still threefold. But in this study, he shows that those one step down from the top of the hierarchy, civil servants who are professionals and executives, such as doctors and lawyers, have heart disease rates that are twice as high as those at the very top. Those at the very top are upper class directors of agencies, all of whom have been educated at *Oxford* and *Cambridge*, and whose career usually ends with knighthood.

It is not surprising that those at the bottom have higher rates of disease than those at the top, but it is surprising that doctors and lawyers, one step down from the top, also have higher rates. Doctors and lawyers are not poor, they do not have bad houses or bad medical care, and they do not have poor education or poor nutrition. It is not just that those at the bottom have the highest rates of heart disease: there is a gradient of disease from the top of the British civil service hierarchy to the bottom.

At first I thought this phenomenon might be a finding that was somehow unique to the British civil service. It is not. We recently completed a review of this issue and we found a similar gradient almost everywhere in the world and for virtually every disease that has been studied *(Fig. 2)*.

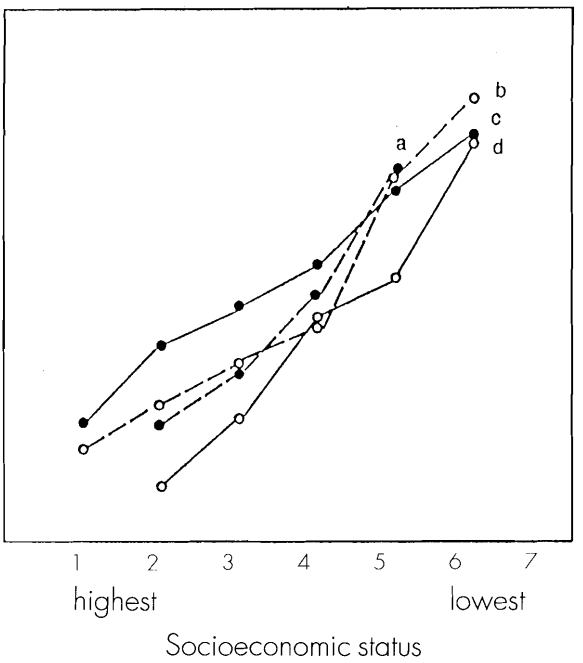

Fig. 2: Socioeconomic status and prevalence of disease in various communities

●- - -● a. Percent diagnosed osteoarthritis (USA)

○- - -○ b. Relative prevalence of chronic disease (UK)

●——● c. Prevalence of hypertension (USA)

○——○ d. Rate of cervical cancer per 100,000 (USA)

The problem posed by this finding is the following: we can imagine why those at the bottom have higher rates of disease but how can we explain a gradient? How can we explain the findings that those one or two steps from the top have higher rates of disease even though they do not suffer from the problems experienced by those at the bottom? People one or two steps from the top do not experience poverty, or poor nutrition, or problems of access to medical care, or bad housing, or poor education.

I do not know the answer to this. The only hypothesis I have been able to come up with is that as one moves down the social class hierarchy, one has less control of one's destiny. By this I mean less opportunity to influence the events that affect one's life. At first, I thought this idea about control of destiny was a brilliant, original idea. Unfortunately, it is not. In fact, it was a little embarrassing to realize that this great truth that I had discovered had been a topic of research by psychologists and sociologists for dozens of years. Many scholars have studied the concept of mastery, self-efficacy, locus of control, learned helplessness, controllability, predictability, desire for control, sense of control, powerlessness, hardiness, competence, and so on.

It is important not to over-interpret the fact that so many investigators have suggested the importance of control for health and well-being. In fact, few of these people are using the same term in exactly the same way; each use tends to have a special focus and each has been found of value in explaining the different disease outcomes. For this reason, it is an exaggeration to claim that they are variations on one theme. On the other hand, it is intriguing that so many different scholars, from different backgrounds and with different research objectives, should come up with ideas that are so similar to one another.

More recently, a group of epidemiologists led by ROBERT KARASEK from the Unites States and TORES THEORELL from Sweden have shown that rates of coronary heart disease are higher among workers who experience not only high job demand but low discretion and latitude for dealing with these demands. The work of these researchers is especially impressive because previous studies of job stress had for decades failed to establish a link between job pressures and health even though this issue had been examined intensively. When the concepts of control and discretion were included in the research, important findings have at last emerged and in fact are now being replicated by many others around the world.

I do not know if the idea of control is correct or not, but I do know that some idea like it is necessary to deal with the difficult problems we face in trying to prevent disease. We need an idea that will help us understand why disease rates

are higher in certain groups than in others - over time - even though people come and go from these groups.

Since I do not at present have a better idea than that of control, we are using it in helping us understand the health of bus drivers in *San Francisco*. Several previous studies have noted that bus drivers have a higher prevalence of hypertension, as well as diseases of the gastrointestinal tract, respiratory system and the musculoskeletal system as compared to workers in other occupations. These results have been obtained from studies from different transit systems, under different conditions, in several countries. Based on these findings, it has been suggested that certain aspects of the occupation of bus driving may create an increased risk of disease for workers in that occupation.

From a clinical view point it is of value to identify drivers with disease in order to treat them. It would also be of value to teach drivers about better posture, more healthful eating habits and alternative ways of dealing with job stress. However, from an environmental perspective, it would perhaps be more useful to identify those aspects of the job itself that might be changed.

In our study of drivers, their exposure to noise, vibration and carbon monoxide fumes is being monitored but particular attention is being paid to the social environment of the driver. For example, in preliminary studies of drivers, the «tyranny of the schedule» has been forcefully brought to our attention. Drivers must keep to a specific schedule, but in almost every instance, this schedule is arranged without realistic reference to actual road conditions, and in fact cannot be met. If this and other characteristics of the job that are associated with disease can be identified, it may be possible to introduce interventions, not merely among bus drivers, but directly on those environmental factors associated with the job. For example, it may be that by changing the way in which schedules are arranged, the bus company would be able to earn more money than it loses because of lower rates of absenteeism, sickness, accidents, and in particular, turnover of employees.

In the case of bus drivers, a clinical focus either on hypertension, gastrointestinal diseases, respiratory disease or musculoskeletal disorders clearly is useful. However, from an environmental and preventive perspective, it might be useful to group together these different diseases and conditions associated with common work exposure so they can be studied as related phenomena. If this is not done, the circumstances they share will not likely be appreciated.

We inform mothers that if they want to have a baby they can have it in a hospital or at home. They have a choice. And we provide information on where they can find more information about this. If they want to have a baby in the hospital, we

inform them that they can have the baby with them in their hospital room or not. They have a choice. And we tell them where they can get more information about this. We give mothers other useful information such as when they call the state or local government office for help, the first thing to do is to get a chair and sit down and to bring with them a book or knitting. These calls will probably take a long time to complete but it can be done! We give them advice on how to do difficult jobs. We suggest to them that there are ways to solve problems which many people, especially poor people, have long since given up on. We are now testing this guide by distributing it to 100,000 women in a program we call *WIC - Women, Infants and Children Nutrition Supplement for pregnant women.*

In my view, these ideas expressed in this talk are simple, straightforward and even obvious. Certainly, nothing I have said is new. The puzzle is that we nevertheless cling to a way of looking at the world that we know does not deal with the problems we face. It is interesting that *500 years after* PARACELSUS, we still tend to struggle with the same type of problem he dealt with all of his life. To attack the traditional and established interest of his day demanded a willingness on his part to defy the canons of respectability and to uproot himself from the university and professional community. As DANIEL BOORSTIN has noted, to do this also required as much passion as knowledge and more daring than prudence.

To do this, a person needed the **knowledge** of a professional but the ability to avoid being **committed** to the profession. One of our problems today, I think, is that we continue to organize our training and certification programs in ways that would effectively silence a modern-day PARACELSUS. We have done this with the very best of intentions, but it is a pity nonetheless. It is good therefore to celebrate PARACELSUS. Perhaps we may even learn something.

Further reading

ADLER NE, BOYCE T et al. Socioeconomic status and health: The challenge of the gradient. *American Psychologist* 1994; 49: 15 - 24

KARASEK R, THEORELL T. Healthy work: Stress, productivity, and the reconstruction of working life. Basic Books, New York 1990

SYME SL. Control and health: A personal perspective. In: Steptoe A, Appels A (eds.). *Stress, Personal Control and Health.* John Wiley, New York 1989

COHEN S, SYME SL. Social support and health. Academic Press, Orlando/Florida 1985

BOORSTIN D. The discoverers: A history of man's search to know his world and himself. Vintage Books, New York 1985

MARMOT MG, ROSE G et al. Employment grade and coronary heart disease in British civil servants. *Journal of Epidemiology and Community Health* 1978; 3: 224 - 249

SYME SL, BERKMAN LF. Social class, susceptibility and sickness. *American Journal of Epidemiology* 1976; 104: 1 - 8