Using theory to guide policy relevant health promotion research

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SUMMARY
The concept of health promotion has evolved into a strategy for improving health that goes beyond individual behaviour to include the physical, social and economic environments in which both health and behaviour are shaped. The core of the strategy is to stimulate action against the root causes of ill health in communities. A prerequisite for effective health promotion action is valid knowledge about how forces protect or damage health in daily life. Developing and using theory to guide the collection, analysis and evaluation of empirical evidence is a neglected aspect of obtaining the knowledge needed for promoting health. Population interventions to reduce cholesterol provide an example that illustrates the consequences of basing community health policy and programmes on findings from empirical research without developing a logically sound theoretical basis for identifying inconsistencies and contradictions in the findings. The use of theory to guide research to support health promotion action is discussed.

Key words: health promotion; informed action; theory

INTRODUCTION
The field of health promotion is infusing new thinking into population health research and the policy making process for health. Originating in a critique of traditional health education (Green and Raeburn, 1988), the concept of health promotion has evolved into a strategy for improving health that goes beyond individual behaviour to include the physical, social and economic environments in which both health and behaviour are shaped (World Health Organization (WHO), 1984; Kickbusch, 1986). The core of the strategy is to stimulate action on the root causes of ill health in communities.

What then, it might be asked, has theory, a core element of ‘basic’ research, to do with health promotion? Since health promotion research is supposed to support action, it is, in the minds of many, limited to ‘action’ research. When focused on community health, action research involves attempts to change policies, environments, professional services or the personal behaviour of individuals in order to improve health. Frequently it involves implementing and assessing the effects of interventions. Clearly, action and/or action research based on faulty ‘basic research’, will lead to uncertain problem identification or incorrect assumptions that misdirect policy (Orosz, 1994).

A prerequisite for action that addresses the root causes of poor health in communities is valid knowledge about the forces that protect and damage health in daily life. Characterized as a ‘knowledge challenge’ (Kickbusch and Dean, 1992; Labonte, 1994), a health promotion strategy involves assuring a valid knowledge base to inform the action. Without entering the deep and muddy waters of debates about the differences between basic and applied research, this paper asserts that theory is essential to valid and meaningful research, however conceptualized. Since the central concerns of theory building have
to do with explaining phenomena in both the most comprehensive and the most precise way possible (Selltiz et al., 1976; Suppe, 1977a), it might be concluded that theory is the most fundamental prerequisite of a policy science, and that the absence of theory and effective theory building are serious weaknesses of much of the existing research available for the policy making process for improving the health of populations.

In order to explore these issues, a detailed example will be used to illustrate the consequences of basing community health policy and programmes on findings from empirical research without developing a logically sound theoretical basis for identifying inconsistencies and contradictions in the findings. Based on this example, the use of theory to guide basic and applied research to support health promotion action will be discussed.

COMMUNITY INTERVENTIONS TO REDUCE CHOLESTEROL

In the 1980s, major health policy initiatives directed toward reducing cardiovascular disease involved population interventions to reduce cholesterol blood levels. A well-documented higher risk of death from coronary heart disease among persons with elevated levels of blood cholesterol formed the basis of the policies focused on activating people to lower blood cholesterol. The goal was to contribute to improving public health by shifting the distribution of blood cholesterol concentrations for entire populations (Consensus Conference, 1985; Study Group, 1987). Extensive public funds were used in many countries on interventions and health education focused on getting people to change their diets, obtain blood cholesterol measurements and even use drugs to reduce cholesterol levels. After extremely costly population based programmes had been in place for some time, it became known that not only high, but also low levels of blood cholesterol are associated with disease and death—that there is a ‘U-shaped curve’ in the relationship between blood cholesterol and mortality.

Muldoon and his colleagues (1990), conducted a meta-analysis, a quantitative review of the evidence, on findings from primary prevention trials of the type that had been used to justify this community health policy. The purpose of the meta-analysis was to determine the effects of lowering blood cholesterol on total and case specific mortality. In order to enhance the comparability of the findings and reduce statistical effects from combining data from different studies, rigorous criteria were developed for accepting experimental trials into the study. Each project had to have: (i) been a randomized clinical prevention trial of serum cholesterol reduction; (ii) included a treatment group that received instructions for a diet and/or drugs to reduce cholesterol and a control group; (iii) resulted in the lowering of cholesterol in the intervention group relative to the control group; and (iv) reported both total and cause specific mortality in the results of the trial. Six randomized trials totaling 24 847 male participants could be accepted on the basis of these criteria.

The results of the meta-analysis showed that while each trial reported evidence of success in lowering blood cholesterol levels, the evidence of any reduced coronary heart disease (CHD) mortality was weak, and overall survival among men who underwent lipid lowering treatment was not improved. Furthermore, mortality not related to illness was nearly twice as high in the intervention groups as in the control groups, a finding consistent for all six studies whether or not the cholesterol lowering treatment was carried out with drugs or dietary education. These highly consistent and disturbing results were reported about the same time that other investigators were uncovering similar findings (Holme, 1990; Strandberg et al., 1991).

The evidence from studies of cholesterol reduction was presented for evaluation in a US National Heart, Lung and Blood Institute expert conference in 1990 (Conference Report, 1992). The experts reviewed and discussed existing data on the left-hand limb (association between low cholesterol and mortality) of the U-shaped curve, the term that is used to represent the greater mortality found among persons with both high and low blood cholesterol levels. Presenting results of a statistical overview of available cohort studies with findings involving 68 406 deaths, a study of unprecedented size, it was documented that the higher mortality rates associated with low blood cholesterol held both across studies and for a diverse range of causes. Especially high excess death rates from digestive system conditions and from less common diverse causes were found, but also cancer, respiratory system and injury deaths were associated with low cholesterol.

The findings of higher risk of death from causes
other than CHD held for both men and women. At the same time, however, the second major finding of the conference report was the 'surprising observation' that for women 'high blood cholesterol is not associated with all-cause mortality nor even with cardiovascular mortality' (Hulley et al., 1992). Three major conclusions were drawn from the findings presented in the expert conference:

• the association between low blood cholesterol and noncardiovascular deaths indicated the need to review policies aimed at shifting entire population distributions of blood cholesterol to lower levels;
• the lack of association between high blood cholesterol and cardiovascular deaths in women indicated that, except for those with coronary disease or other high risk of CHD death, 'it no longer seems wise to screen for and treat high blood cholesterol in women';
• the findings in primary prevention trials of cholesterol intervention that the increase in non-CHD mortality rates is similar to the decrease in CHD death rates indicated that, except for people who already have coronary disease, it is unwise to treat high blood cholesterol with drugs.

IMPORTANT OF KNOWING AND USING RELEVANT RESEARCH LITERATURE

Actually, it turns out, as noted by Hulley and his colleagues (1992), that this 'U-shaped curve' had been reported for two decades. Moreover, Muldoon and his colleagues (1990) had noted that, although large primary prevention trials had found evidence suggesting that lowering serum cholesterol concentrations reduced the incidence of coronary events, predominantly myocardial infarction, only one had found mortality from heart disease lowered significantly after cholesterol reduction.

If one examines research literature other than that from controlled clinical trials, evidence that would have foreseen these findings goes back to at least 1962 (Groen et al.). In an investigation conducted in Holland, cholesterol research was placed in a lifestyle context, with the result that the findings differed considerably from simple predictive correlations between measures of cholesterol and CHD mortality. The study involved comparative analyses of behavioural and health variables in populations of Benedictine and Trappist monks living in cloistered monasteries.

It was found that blood cholesterol levels were much higher in all age groups for the Benedictine monks compared to the Trappist monks. The essentially vegetarian diet of the Trappist monks is much stricter than that of the Benedictines. This is evidence that diet can affect blood cholesterol, also shown in the cholesterol intervention trials. Cardiovascular disease incidence was, however, in spite of the higher cholesterol levels of the Benedictines, the same in both monk populations, and much lower than in the general male population of comparable age in Holland. The findings show that even if blood cholesterol is a contributing factor in cardiovascular disease, it is not a sufficient cause. Social and psychosocial processes, as either component causes or modifiers of disease processes, appear to be the determining influences. This study illustrates that it is research on lifestyle, understood as patterns and ways of living (Coreil et al., 1985), rather than cholesterol or any other risk factor that is needed for understanding health and disease.

Geoffrey Rose (1985), in a discussion of problems and limitations of the risk factor model of research on population health, unintentionally, but powerfully, illustrated the potential problems that would arise from action connecting cholesterol and heart disease in a simple causal model. He used findings from the Framingham Study, a comprehensive longitudinal investigation of health outcomes in a US population sample, to juxtapose the serum cholesterol curve of the persons in the cohort study who developed CHD over the curve of those who did not develop heart disease. The curves showed a comparable range and peak in the cholesterol values of those who did and did not develop disease, rendering the powerful relative risks produced from the data unimpressive.

Rose felt that research focused on individuals constrains knowledge about the causes of diseases. With roots in clinical practice, the purpose of both case control and cohort epidemiological studies, he noted, is to discover how sick and healthy individuals differ. He pointed out that the use of relative risk as the measure of aetiological force in this paradigm has almost excluded the use of any other approach in epidemiology, even though relative risk 'is no measure at all of aetiological outcome or of public health importance' (p. 32). Rose reminded us
that, while considerable information is available about the characteristics of individuals susceptible to various diseases, 'most non-infectious diseases are still of largely unknown aetiology'.

These considerations led Rose to the conclusion that research should focus on populations rather than individuals—ecological comparisons in contrast to relative risk approaches. Using CHD as an example, he pointed out that even with screening to detect early disease, there is weak ability to predict the future for individual patients. His suggested solution, looking at population averages and attempting to shift the curve of whole populations on specific risk factors is, however, the approach that was found to be dangerous in the overview of research findings on the U-shaped curve with regard to cholesterol related mortality. Rose's recommendation, aimed at preventing cardiovascular disease in populations instead of detecting disease at an early stage in individuals, was still centred in a risk factor cause and effect model of disease.

Both relative risk approaches to specific aetiology and ecological studies examining group differences on specific phenomena look away from the differential impact of causal influences in the presence of other causes and over periods of time. Rose's synthesis of findings pointing to the limitations of the relative risk paradigm in a sense replicate those of the research in Holland described above, but without the theoretical force of the lifestyle study for suggesting contributing causes.

THEORY, THE NEGLECTED COMPONENT

The evidence provided by the cholesterol studies, whether they stem from randomized trials, case control, cohort or ecological studies, centre on statistical effects and predictions. Detecting a statistical effect of a factor that remains after other influences are removed by randomization or statistical 'control' does not provide the information needed for understanding the statistical connection between the two variables. Relationships between variables that have been tested and replicated may be true, but remain so unspecified that they do not provide meaningful knowledge (Merton, 1949). It is necessary to learn the conditions under which statistical correlations hold, are modified or disappear to understand causal processes determining health in the real world. A theoretical logic for transferring statistical correlations into causal processes and for investigating inconsistencies and contradictions in the findings, such as that developed by Rosenberg (1968), is absent in the risk factor model of disease. This problem is a major barrier for understanding the meaning of statistical correlations between specific factors and disease.

The cholesterol example is illustrative because results available in the research literature contained theoretical insight suggesting alternative directions long before the costly and perhaps dangerous interventions led those responsible for them to conclude that the policy needed to be changed. One wonders at the power given statistical predictions in this body of work, as if the statistical connections between two factors had real meaning for the health outcomes of individual people.

Even without an awareness of research findings such as those from the investigation in Holland and those presented by Rose, or without stopping to think about the reality constraints of statistical correlations, one wonders why cholesterol was given such unique power as a cause of CHD. Should not cholesterol be regarded as a biological marker expressing a state of physicochemical processes in the individual at a given time, an outcome or at least an intermediate outcome, rather than a cause? What theoretical logic was presented and tested that could grant cholesterol the causal status justifying intervention affecting the health of general populations and the use of huge amounts of limited public health resources?

The purpose of scientific theory and methods is to expand knowledge about causal processes. All research designs and statistical models have limitations for testing theories. The details of how scientific methods fall short fill numerous texts on research methodology. Research granting a methodological approach a primacy that hides its limitations, and without theoretical frameworks to illuminate the limitations, should never be the basis of health policy and action. The overwhelming consensus is that methods appropriate for given research questions (fallible as they are) should be used to test theories (Stolzenberg and Land, 1983). This means that both action and basic research in pursuit of knowledge to inform health policy would always be an interplay between expanding theory and improving methods (Dean, 1993).
CHANGING VIEWS ABOUT THE ROLE OF THEORY

The meaning and importance of theory have changed periodically throughout the history of science. Long traditions of separating theory from empirical observations had serious consequences on scientific research, including that conducted in population based sciences (Bernert, 1983). The weaknesses of atheoretical empirical work are now widely recognized (Faust, 1984; Maclure, 1985; Dean, 1993). Suppe (1977b) discussing major contemporary views on theory building, reached the conclusion that only in primitive science, where the development of theories is neglected, does the verification of empirical predictions maintain a central position. A major function of theory is to organize and integrate information for discovery (Shapere, 1977).

The above observations do not mean that developing and using theory guarantees 'truth' or even necessarily the advancement of knowledge. Theory, an aid to human cognitive limitations, helps to organize information in the body of knowledge for further inquiry (Faust, 1984). When not tested and modified appropriately, theory can become transformed from a scientific thesis about reality relationships into a rationale for a belief system.

Competing theories are also important for gaining new knowledge. The interpretation of observations considered true within the framework of a particular theory will be only partial truths, or even false, in the context of another theory. Gillett (1994) illustrates this with the example of the corpuscular theory of light being considered false when wave theory became dominant, but regaining importance with quantum theory. The revived importance of corpuscular theory was not, however, considered to discredit wave theory. Both became part of a new understanding of particles and the development of a new theory of light. This example is useful because it illustrates that different theories, sometimes opposing or contradictory and sometimes complimentary, as well as the modification of theory, are core aspects of advancing scientific knowledge. Referring back to the cholesterol example, the point is that empirical observations are always partial representations conveying certain truths and concealing others.

USING THEORY IN HEALTH PROMOTION RESEARCH

A great deal of health promotion research has been limited to the health related behaviour of individuals. When some form of theoretical framework is used in research on behaviour and health, it is generally based on theories developed by social psychologists. These theories are used quite loosely and rarely modified or rejected when very little of the 'variance' in the behavioural practice is explained. It has been argued that these models are more 'idea-sets' than the type of theories usually developed to provide scientific explanation (Research Unit in Health and Behavioural Changes (RUHBC), 1989). Influenced both by risk factor epidemiology and social psychology, this work provided the knowledge base for health education. In recent years, more studies have focused on social environments and structural determinants of health (Milio, 1986). Starting slowly as a critique of traditional health education, the shift gained force with the evolution of the field of health promotion.

The term 'health promotion', generally traced to the Lalonde Report (1974), remained for some time a vaguely conceived umbrella term very often used in relation to quite traditional research and programmes (McQueen, 1994). It was after the WHO (1984), Health Promotion: A Discussion Document on the Concept of Principles, and the Ottawa Charter (WHO, 1986), launched 2 years later at the first International Conference on Health Promotion, that the process of establishing research, education and publication structures moved rapidly forward.

Much has been done to shift the health promotion research agenda. Still, a great deal of the research conducted in the field continues to rely on the risk factor tradition, methodological approaches and the social psychological thinking from which the field emerged. New theoretical thinking is present in the field conceptually and implicitly, but making theory explicit and a strong force guiding research and explanation has not yet occurred. While health promotion cannot lay claim to well-developed theories of its own, even in its infancy, this young field developed a strong conceptual base that provides a framework for theory development. Theories embedded in the concepts and principles of health promotion (WHO, 1984) and the Ottawa Charter (WHO, 1986) can guide the various types of research on institutions, populations and individuals that are
needed to provide knowledge for health promoting action.

The health field concept (Lalonde, 1974), modified on the basis of the Ottawa Charter (Raeburn and Rootman, 1989), summarizes domains for research and action. Other models and conceptual frameworks have been put forward as well. The characteristics that best describe the types of research needed to provide the knowledge for health promoting action are contextualism and dynamism (Dean et al., 1993). The emphasis on environments, communities and policy opens the way for theories of multilevel influences that can expand knowledge about the causal processes that people are exposed to in the contexts of daily living.

Bringing together theory and knowledge from diverse sources is a central idea in Shapere's (1977) concept of theoretical domains. In his thinking, theory and empirical research are mutually interdependent. The development of a theoretical domain in research to promote health would entail discovering causal processes by exploring and then fitting together moderating influences instead of trying to reduce complexity to a simple connection between two factors. A statistical relationship between two variables can only be a starting point for understanding the context in which the two variables relate to each other, and for identifying the conditions that modify the relationship.

Developing the context in a theoretical domain examining how lifestyles affect health would involve fitting together findings from different types of studies, as well as integrating different levels of influence into investigations of lifestyle and health. The contexts in which people live their daily lives involve social conditions and opportunities for learning and practising life skills. Social norms and formal legal statutes, as well as the resources that shape both personal skills and supportive environments, make up the domain in which lifestyles are formed and maintained. It is necessary to study the formal and informal rules governing the context in a research domain (Gillett, 1994).

Specific health related behaviours would have limited meaning or relevance in a lifestyle research domain. Theories would postulate how ways of living protect or damage health. Translated into empirical research, patterns of behaviour (their separate, joint and interactive effects on health) in the context of daily life, would be studied with complementary types of research methodologies. Cholesterol levels might be studied to obtain knowledge about the causal processes leading some people with high cholesterol to develop heart disease while others remain healthy. This would involve identifying how intervening influences such as general behavioural patterns, stress, supportive environments and available resources affect the health of people with family histories of high cholesterol and/or heart disease.

Thus in a lifestyle framework, meaningful research would seek to understand the influence of living situations, and of cultural and subgroup learning and expectations on behavioural practices. Another important area of inquiry would be the progressive nature of health damaging habits for some people. 'Addiction' research needs to study environmental demands and the moderating effects of personal skills and of support available in social networks.

Time, generally neglected in population health research, is another major force determining the contextual nature of causation. Lifestyle theory, developing the interplay of environmental context and behavioural patterns would need to be tested with methods capable of disentangling the effects of age, period and cohort aspects of causal processes (Riley, 1993). The contribution of any specific influence is not necessarily stable over time, and there may be social group differences in the relative stability of influences. Take, for example, the impact of diet at different periods of life. A deficiency in childhood of nutrients needed for normal development will have far more serious consequences for health, functioning and longevity than the same deficiency in late life (Barker, 1988).

For other influences, the impact at different periods of the life course may reverse. The physiological caustic effects of a substance such as alcohol may be far less for young active people with rapid metabolism than for old people who are less active and whose metabolic processes have altered character. At the same time, alcohol presents other dangers to young people relative to older people. The role of peer pressure is especially strong in youth, and may contribute to overuse or dangerous use of alcohol or other mood altering substances among young people relative to mature adults. Accidents, especially those involving alcohol and/or speeding in motor vehicles, are a major cause of death and disability for young males. Variations in how influences affect outcomes need to be understood for effec-
Using theory to guide policy

The field of health promotion has already taken great strides in shifting the research agenda to subjects more in tune with real community health needs. It is now important to assure that the sometimes heavily rhetorical discussions on the subject of health promotion do not impede progress. In order to continue and build on the contributions already made to public health, the field of health promotion 'must now move beyond the rhetoric of its developmental years' (McQueen, 1994). 'Action' research and other forms of research to inform health policy are not new. They have always existed in the field of public health. Throughout an extended period in this century, research became dominated by the biological experimental paradigm, and indeed action research in that tradition did form the basis of community health policy. The policies focused on shifting the cholesterol curves of general populations resulted from action research accepted in the policy making process.

Changing the focus of the health debate and the renewed emphasis on the environmental determinants of health are important contributions of the field. Community health programmes have, however, received high priority in the past only to be dismantled, and even within the field of health promotion research and programmes have not always risen above the individualized risk factor approaches (Green, 1994).

Research simply shifting the focus from 'risk factors' to 'risk conditions' is not sufficient for improving knowledge. The risk factor model itself has built in limitations. Like so many dysfunctional dichotomies that are being challenged in contemporary science, arguments about the micro–macro determinants of health, and their counterpart, the nature–nurture division, are both reductionistic approaches (Lewontin et al., 1984; Lewontin, 1991). Both fail to acknowledge the multidimensional and interactive nature of the causal processes that shape health and functioning.

CONCLUSION

Theory for guiding health promotion research needs to build in the complexity involved in real causal processes that shape health over time. Relationships among influences can then be explored with the range of research designs, qualitative as well as quantitative, for studying research issues, and with analytic approaches that are capable of studying direct, indirect and moderating relationships.

'The 1990s represent the watershed for health promotion. Now health promotion must show its utility to the skeptics, as well as those who have had their consciousness raised by its rhetoric' (McQueen, 1994, p. 336). Action that is not based on valid knowledge will discredit the field. Theory based in the concepts and principles of health promotion can guide research to new knowledge about health and health related quality of life.

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