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The role of methods in maintaining orthodox beliefs in health research

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Abstract

Views about correct ways of obtaining knowledge develop from socially constructed tenets and beliefs. The dominant beliefs about how health research should be conducted are derived from the biomedical model of human health. The beliefs are maintained by traditions developed in support of the orthodox model and by power relationships. This paper examines the impact of the orthodox views of the biomedical model on the research methods used to investigate population health issues. Experimental design is the "gold standard" for research in the biomedical model. Beliefs about the superiority of experimental research have affected most types of health research. The role that methods assume in maintaining the orthodoxy is examined. Acceptance in other health disciplines of the attitudes of the dominant paradigm and limited options for research and training in alternatives to the orthodoxy became major influences reinforcing orthodox beliefs about health research.

Keywords: Orthodox beliefs; Causation; Experimental design; Statistical modeling; Methods

Introduction

Generally accepted views about correct ways of obtaining knowledge arise from socially constructed tenets and beliefs (Kuhn, 1962; Wolpe, 1994; Zito, 1983). Orthodox views about health and correct ways to conduct health research are maintained by traditions that are developed in support of dominant beliefs and by power relationships (Engel, 1977; Gillet, 1994; Lewontin, 1991). The impact of orthodox views on the research methods used to investigate phenomena, and, in turn, the role that methods assume in maintaining the orthodoxy, is less often considered. This paper will summarize developments shaping western orthodox views about human health that are relevant for considering their impact on research methods. A brief overview of the historical roots of the dominant biological paradigm provides background for understanding orthodox beliefs about how to gain knowledge about human health. It will be argued that orthodox methodology arising from the dominant paradigm has impeded innovation in health research. The paper will focus on quantitative research investigations dealing with population health issues.

Knowledge about human health: The dominant orthodoxy

As socially constructed fields of learning, dominant beliefs in a discipline are based on an underlying ideology. An ideology is an organized body of views that seek to monopolize ways of thinking and speaking about the world (Zito, 1983). The dominant tenets and beliefs about how health research should be conducted are derived from the biomedical model of human health (Engel, 1977; Gillet, 1994). The biomedical model functions like a traditional natural science, attempting "to isolate distinct and identifiable diseases which are causally produced by some underlying patho-physiological condition which can be isolated, verified and monitored" (Gillet, 1994, p. 1127). In this biological paradigm, explanations for psychosocial, psychological,

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and behavioral aberrations are sought in biochemical and neurophysiological causes. The model builds on the philosophical concept of reductionism which holds that complex phenomena are derived from single principles or causes; and mind–body dualism, a doctrine that separates the mental from the somatic (Engel, 1977).

In the biomedical research model, it is believed that the highest form of knowledge is provided by experimental research. Seeking the simplicity inherent in reductionism, the focus is experimental manipulation of hypothesized causes—germs, genes, chemicals, or other substances, that are easily isolated and monitored. The model deals most comfortably with single microorganisms, biochemical agents, or structural defects (Gillet, 1994). This arises naturally from the origins of modern scientific medicine in the germ theory of disease. After discoveries of microorganisms involved in specific diseases, germ theory replaced pre-scientific concepts of miasmas, or disease causing vapors, which characterized causal beliefs throughout most of the 19th century (Susser & Susser, 1996).

Soon after the advent of germ theory, it was learned that infection with microorganisms is generally not sufficient to explain the onset and course of an infectious disease (Dubos, 1965; Duncan, 1988). In microbiology, relative virulence is the concept used to discuss differential susceptibility to infection. Virulence is defined as the relative capacity of an infectious agent to overcome available host defenses. Complex processes involving variations in both the disease agent and the host determine relative virulence. Normal host defenses function to protect the individual from the multitude of potentially disease-producing organisms to which there is constant exposure. Relatively nonpathogenic organisms, however, may infect persons who are "immunocompromised". This means that "minor alternations of host defenses may create major differences in the apparent virulence of some pathogens" (Sparling, 1983 p. 637).

While knowledge about relative virulence displays multicausal processes, the concept is derived from the perspective of the pathogen or disease agent. The agent focus in research on infectious diseases was transferred to other areas of health research, including clinical, epidemiological, and even some social science research on health. Traditional epidemiology recognized, at least theoretically, the importance of the environment and the condition of the human host in the occurrence of diseases. Still, germs or other disease agents maintained a central position in research on specific diseases. Even in the triad model of classical epidemiology, "the single element of the agent is represented as if it were equal in importance to the variety of relevant factors in the host and the multitude of environmental influences" (Duncan, 1988, p. 36). Vast bodies of research show that the excessive weighing allocated to single agents as causes of disease is not warranted.

As infections became less important as the dominant type of disease, attempts to link a pathogen with each specific disease shifted to efforts to identify other types of disease agents. These studies often involved testing hypotheses about single causes to predict specific pathological deviations, with attention centered on biological and chemical agents. The specific-cause biological paradigm of the biomedical model has even dominated research on the complex processes involved in human aging. It is not uncommon for cellular aging to be cast as an opposing explanation to exposure to health damaging influences over the life course for age-related increases in the prevalence of diseases. For example, specific biological alterations in antitumor defenses with aging are investigated as alternative, rather than supplemental, hypotheses to exposure to carcinogens, nutrition, and other joint or intervening influences affecting positive relationships between age and most cancers (Dean, 1997; Miller, 1991).

Documented weaknesses in the dominant orthodoxy

It is well known among biologists (Dubos, 1965; Lewontin, 1991; Lewontin, Rose, & Kamin, 1984; Sparling, 1983; Strohman, 1997), clinical practitioners and researchers (Engel, 1977; Gillet, 1994; Martin, Danner, & Holbrook, 1993; Miller, 1991), public health specialists (Davison, Macintyre, & Davey Smith, 1994; Rose, 1992, 1985; Susser & Susser, 1996), and epidemiologists (Duncan, 1988; Rothman, 1986; Vogt, 1992) that diseases seldom develop from single causes. Yet, as outlined above, far more research is focused on disease agents than on host defenses and the interactions among biological, psychological, and social influences involved in the protection or breakdown of human health. A problem-ridden paradigm for all types of conditions, when the disease agent perspective is transferred from infectious to chronic diseases, the weaknesses of the model are even more sharply apparent (Davison et al., 1994; Duncan, 1988; Gillet, 1994; Ory, Abeles, & Lipman, 1992).

The relative capacity of an infectious or any other disease agent to overcome available host defenses is determined by complex processes that operate at different levels of causal influence (Dubos, 1965; Finlay & Falkow, 1989; Sparling, 1983). Lewontin (1991) stresses the importance of distinguishing between infectious disease agents and causes, emphasizing that the logic applies equally to other single agents (carcinogens, specific genes, behaviors) as causes of specific diseases. That diseases are not simply caused by single agents or factors is clearly illustrated by the multicausal processes involved in the functioning of even the most

basic biological components of health: the genes (Kishino & Waddell, 2000; Lichtenstein et al., 2000; Strohman, 1997; Waddell & Kishino, 2000). The genes of individuals interact with each other and environmental influences to shape variations among individuals within species. Knowledge of the complete molecular structure of every gene in an organism would not assure the prediction of what that organism would become (Lewontin, 1991). These points are illustrated by recent findings from research on cancer in cohorts of monozygotic twins, who share all genes, and dizygotic twins, who share about 50% of their genes (Lichtenstein et al., 2000). Comparing the cancer rates of twin pairs, the researchers concluded that environmental causes account for most cancers, while genetic factors make a relatively minor contribution to the occurrence of most neoplasms. These researchers pointed out that genes may be modified by environmental influences, and that environmental influences may operate differently in the presence of specific genetic conditions.

Recently documented increases in osteoporosis among men provide another example of the complexity inherent in disease processes. Traditionally considered a disease of women, a strong increase in osteoporosis has been documented among men since 1970 (Sørensen, 2000). Factors such as exercise and diet are considered responsible for its growing prevalence. Since genetic aspects of osteoporosis existed prior to 1970, it appears that modern lifestyles account for changes in the onset and course of this disease in both men and women. The serious gaps in knowledge about coronary artery disease provide another example of how little is known about interactions among genes and their products and environmental influences in the development of disease. Experts in molecular biology point out that research on genetic predisposition to disease needs to recognize both the contribution of multiple genes and the myriad influences from the life history of individuals (Strohman, 1997).

The inability to reduce diseases to simple cause and effect mechanisms is even more readily seen in diseases that have etiologies that appear quite individualistic. Gillet (1994) uses chronic allergic rhinitis to illustrate the individualistic features of inflammatory diseases. He points out that inflammatory reactions involving biochemical processes in the lining of the nasal passages favor chronic infections with organisms that would normally be cleared from these passages. It has been found that the allergic factors stimulating the inflammatory reactions are multiple and shifting in individual patients. Thus, not only are multiple causal factors involved, but the necessary and the sufficient factors for the condition to occur change both across persons and within the same person. The immune system is functioning or malfunctioning in complex ways that are not understood.

Knowledge about health and disease as people age is seriously constrained by research focusing on narrow hypotheses. The greater prevalence of diseases among old people is not explained by hypotheses about declines in immune functioning with aging (Miller, 1991). There are inconsistencies in the evidence from research studies examining aging declines in biochemical aspects of immune functioning or "immune senescence". Many old people maintain immune responses at comparable levels of vigor to those observed in young people (Candra, 1993; Saphire, Rudolph, Hackleman, & Stone, 1993). Furthermore, it appears that complex remodeling of immune functions with age, in contrast to a unidirectional deterioration, is possible (Franceschi, Monti, Sansoni, & Cossarizza, 1995; Hirokawa et al., 1994).

The examples cited in this section provide only a limited selection of the wide-ranging evidence that multicausal processes are involved in the onset and course of disease. It may be concluded that at this time knowledge about health and aging will be most readily expanded by research that identifies and explains interactions among multiple influences.

Maintaining the orthodoxy

How can it be, in the face of extensive scientific and clinical evidence showing that diseases result from complex multicausal processes, that research on human health continues to focus so heavily on single factors that most often are physical or biological agents? The most easily identifiable explanation for the continued dominance of the orthodox biomedical paradigm is control over institutions and resources. In recent times, this dominance has been compounded by the power of pharmaceutical companies and other vested interests influencing and limiting the research agenda. With extensive concentration of research effort and resources on testing drugs and identifying genes or other agents involved in specific diseases, it is difficult to focus attention on mapping how social, behavioral, psychological, and biological variables interact in disease processes. Numerous drugs, many with serious side effects and unknown long-term consequences, especially for people taking multiple drugs or who have various genetic and lifestyle profiles, are being made available and marketed aggressively.

Genetic aspects of some relatively rare conditions have been identified, but the complex knowledge needed to understand more prevalent diseases remains illusive. Discussing the excessive claims arising from the Human Genome project, Holtzman and Marteau (2000) remind us that social structure, lifestyle, and environmental influences account for much larger proportions of disease than genetic differences. They conclude that

the genetic mantle now clouding medicine will prove to be like the emperor's new clothes. The implications of these developments for health promotion, understanding aging, and the effectiveness and ability to fund health care systems are generally ignored.

The dominant biological paradigm has become so entrenched that knowledge challenges to the belief system have had little impact on orthodox views. The developments illustrate how orthodoxies develop ideologies that define standards in the professional, educational, and funding institutions that gradually assimilate the ideology (Engel, 1977; Gillet, 1994; Kuhn, 1962; Wolpe 1994). While it is known that firmly entrenched orthodoxies maintain excessive power and influence over an extended period of time, reinforcing influences that inadvertently serve to maintain the orthodoxy are less well understood. Although generally not recognized, the research methods used to study health phenomena are contributing factors which reinforce orthodox beliefs.

Research methods in the biomedical model

The 19th century discoveries leading to a general acceptance of the germ theory of disease promoted a science of medicine to replace what was viewed as disorganized, unscientific, anecdotal medical practice with research and knowledge based on biological understanding (Duncan, 1988; Rosen, 1958; Susser & Susser, 1996). Scientific medicine developed parallel with, and was fundamentally affected by, the rapid raise in prestige and power of the positivistic science that dominated western thinking at the turn of the century.[1]

In the positivistic paradigm, the scientific method provides knowledge by singling out empirically observable entities in order to predict an outcome of interest. A distinction between knowledge derived from empirical observations and abstract concepts is one of two central dogmas of positivistic science. The other is reductionism (Quine, 1962). These two dogmas established a split between abstract reasoning and empirical observation. Empirical science was to identify cause and effect relationships that are sequential and replicated. Since in this paradigm, if the cause does not occur the effect cannot occur, causation is deterministic. The split between abstract reasoning and empirical observations in a deterministic science was consistent with the mind–body dualism accepted by the Church.[2]

The powerful historical roots shaping the tenets and beliefs that emerged in the scientific biomedical model created an air of certainty that continues to pervade attitudes about how to conduct health research. The model holds that certain knowledge is obtained only from observations made in experimental research. This means that experimental design is the "gold standard" for obtaining knowledge about causation. Experimental design requires the random allocation of research subjects to experimental and control groups. The belief is that causation can only be determined when a test factor can be manipulated in an experimental group that is compared with a control group that has not been exposed to the test factor. This design allows findings for the outcome variable of interest to be collected and compared. It is believed that the random allocation of research subjects creates groups that are comparable in every aspect with the exception of the influence of the test factor. The goal is to predict an outcome in the experimental group that is not found in the control group and thus can be attributed to the test factor.

Belief in the superiority of the experimental model arises quite logically from the two dogmas of positivism. Causal evidence about test factors (agents) can be determined only by empirical observation. The agents studied are separated (reduced) from larger entities, the causal processes in which they are embedded, in the belief that the ability to manipulate a biological or

[1] The 20th century witnessed first the development and later the rejection of a philosophy of knowledge, logical positivism, based in the view that all knowledge is derived from verifying empirical observations about phenomena without the intrusion of mental conjecture (Suppe, 1977a, b). This scientific paradigm was developed and refined by a group of philosophers and mathematicians that came to be known as the "Vienna Circle". David Hume had established philosophical views articulated in positivism long before the type of mathematical logic that characterized the work of the members of the Vienna Circle. In the 18th century, Hume developed two key principles that became the "continuing and cardinal points of empiricist doctrine" (Barrett, 1962): there should be a distinction between truths derived from ideas and truths found in empirical sciences about matters of fact, and the reduction of ideas to sensory experiences. According to Hume, causation is sequential and exhibits regularity. The cause is always followed by the effect, and if the cause does not occur, the effect cannot occur. In this view, causation is deterministic. These principles provided the foundation for concepts about necessary and sufficient causes of events, operationalized by John Stuart Mill in the 19th century, and exerting strong influence on the causal thinking behind the biomedical model.

[2] In his widely discussed paper outlining the need for a new medical model, Engel (1977) concluded that the decision by the Church in the middle ages to allow dissection of the human body was a major historical influence on the development and strength of the model. He considers this concession by the Church to study the internal units of the human body a tacit injunction against a corresponding investigation of the mind and behavior, which were to remain the domains of the Church. At the same time, investigations of the anatomical units of the body were consistent with the mechanistic science of the day. With mind–body dualism firmly established, a reductionistic approach to understanding human health was supported by the dominant institution of the time.

chemical agent to predict an independent statistical effect on an outcome will ultimately provide useful knowledge about diseases in humans.

This model ignores the problem that predicting an independent causal effect is quite different from understanding the context of causal relationships. Predictions made on the basis of a statistical effect observed for a factor on an outcome are not sufficient for explaining causation. It is now known that interactions among many influences will overwhelm statistical predictions about single factors (Holtzman & Marteau, 2000; Martin et al., 1993; Scriven, 1962, 1959; Suppe, 1977b).

Most discussions about the superiority of experimental design in the biomedical model use examples of treatments, usually drug treatments, to illustrate points about the importance of randomization to avoid confounding in testing for the effects of the manipulated agent. Indeed, it is important to assure that unknown influences do not account for or hide the effects of a drug treatment. However, since health maintenance and the development of diseases are inherently multicausal phenomena, detecting a statistical effect of one factor provides very limited information that can be misleading. Even when the goal is to test for treatment effects, important knowledge may be lost if the model and methods do not allow the elaboration of the conditions that determine the effectiveness of the treatment. Thus a statistical effect in a treatment group that is not found in the control group may indeed be "real", while the actual effects of the treatment may differ considerably for various members of the group.

Two major influences supporting the orthodoxy are the acceptance in other health disciplines of the attitudes and models of the dominant paradigm, and the limited options for research and training in alternatives to the orthodoxy.

Accepting the beliefs and models of the orthodoxy

As mentioned above, in research on human health, the biomedical experimental model and/or beliefs about the superiority of the model and the importance of emulating the model were transferred from laboratory research to clinical and population studies. Since experimental design is rarely possible and often unethical in clinical and population health research, the goal became to approximate it with quasi-experimental designs.

In the biomedical model, observational studies are considered a lower form of research inquiry than experimental studies (Edwards, 2000; Pocock & Elbourne, 2000; Rothman, 1986). In observational research, subjects, rather than being allocated to experimental and control groups, are studied in natural settings or in samples selected to represent general or special populations. The inferiority status assigned to observational studies is one of the major orthodox beliefs of the biomedical model. This belief is perpetuated in spite of the knowledge provided by observational research in astronomy, anthropology, geology, and other disciplines. Biology is replete with observational research that provided major scientific advances such as knowledge about evolution and discoveries associated with the mapping of the human genome. Furthermore, there is evidence from overviews of evaluation research that observational studies provide estimates of treatment effects similar to those found in randomized controlled trials and that randomized trials have produced contradictory results in studies of the same clinical treatment (Benson & Hartz, 2000; Concato, Shad, & Horwitz, 2000).

Nevertheless, acclaimed as the gold standard, the experimental paradigm has fundamentally affected the way that observational studies are conducted in population health research. A primary goal is to assure that confounding variables do not distort the "independent" statistical effect of the study variable on the outcome variable. Confounders are all of the influences that purportedly would be removed by random allocation of the sample members into experimental and control groups. When random allocation to experimental and control groups does not occur, it is necessary to control for the confounders statistically in order to draw conclusions about the causal influence of the study variable. Thus the belief that causation can be assessed by testing for the ability of a factor to independently predict an outcome, controlling for other factors, a central tenet of the biomedical model, took root in the analysis of data drawn from samples of populations. Since the prediction occurs in group data and not for every single individual, the outcome must be called "statistical risk" rather than a constant and invariable causal effect.

The risk factor concept, and the methods used to identify statistical risks for specific factors, have dominated quantitative population health research in recent times. Risk factor research has been extremely successful in identifying risks statistically correlated with specific diseases and/or mortality. This success, along with the barriers to causal discovery in this way of conducting research, are responsible for the escalation of risk factors and for the confusion that plagues information about specific risks. In the existing bodies of research on factors (agents) related to morbidity and mortality in research on population health, little is understood about the predictions made for specific factors after the effects of other influences have been removed statistically (Susser & Susser, 1996; Taubes, 1995).

It is quite common that contradictory findings about risk factors are accompanied by unwarranted specula-

tive discussions about possible reasons for the contradictions. The risk concept itself, and the way methods are used to identify statistical risks, are rarely questioned. Since the model focuses on the "independent" statistical prediction of a study variable on an outcome, the ways that other variables function with or alter the influence of the test factor generally receive little attention and may even be deliberately avoided so as not to disturb the prediction.

Contemporary research challenges

The deterministic beliefs on which positivism builds are no longer compatible with contemporary science. Causal thinking has moved beyond beliefs about single causes that predict an outcome to the study of dynamic systems. It is now recognized that rules for determining when an event is necessary and sufficient for the occurrence of an outcome over-simplify causal processes. The outcome may have alternate causes, or the event predicting the outcome may occur simultaneously with a causal influence that has not been discovered, or under a specific set of conditions (Edwards, 2000; Scriven, 1959; Suppe, 1977a, b).

Health research limited to predicting statistical effects of single factors cannot address the challenges raised by modern advances in knowledge about causation. What were considered random variations producing uncertainty in predictions may be real differences arising from the effects of earlier conditions or from interactions among complex forces (Dubos, 1965; Gillet, 1994; Holtzman & Marteau, 2000; Lewontin, 1991; Martin et al., 1993; Scriven, 1962, 1959). It has been documented that interactions among influences can have profound effects on the ways that specific variables are related to outcomes. A major weakness of risk factor research is that it relies on risk ratios that relate exposure to outcome with no elaboration of intervening pathways (Susser & Susser, 1996). Separating components of causal processes from the other variables affecting an outcome will inevitably produce inconsistent findings and inhibit knowledge about how a test variable operates in causing an outcome. Thus experimental design and risk factor research have similar weaknesses. The elaboration of multiple causes and modifying influences is neglected or even precluded in most standard approaches to research design and the statistical analysis of data.

It must first be assured that a predictor is indeed a determinant, and under what conditions causal influence occurs. Knowledge about causal processes, rather than single predictors, allows the identification of misleading correlations and biological deviations that are co-morbidities rather than causes. Biological markers are not causes. They are outcome variables that result from interactions among variables that perhaps cannot or should not be acted upon in isolation.

Limitations in health knowledge available from traditional experimental and quasi-experimental research have been discussed in many academic journals and books (e.g., Dean, 1993, 1996, 1997; Dean, Kreiner, & McQueen, 1993; Dean & Hunter, 1996; Pearce, 1990, 1996; Rose, 1985; Smith & Torrey, 1996; Susser & Susser, 1996; Taubes, 1995; WHO, 1992). In 1995, issues of inconclusive and contradictory research findings on risk factors were taken up in the journal *Science*. Experts in risk factor research interviewed by a science journalist acknowledged that systematic errors, bias, and confounders that can overwhelm statistical variation are ignored in the methods often used to study risk factors. The Dean of the School of Public Health at Harvard, commenting on the problems, said: "We are fast becoming a nuisance to society. People do not take us seriously anymore, and when they do take us seriously, we may unintentionally do more harm than good" (Taubes, 1995). Already, in 1985, Geoffrey Rose, writing about the limits of the risk factor model, noted that even though methods based on the relative risk concept are not measures of etiological outcome, they have essentially precluded other methodological approaches.

Quantitative elaboration of complex relationships

Recognizing that the tools regularly used are not adequate for researching complex problems, Smith and Torrey (1996) emphasized that research for understanding the dynamic systems affecting individuals and societies requires theories of dynamic processes and data, and methods sufficient for testing the theories. They recognized that new methodologies are needed to study nonlinear, dynamic systems, and that quantitative and qualitative methods need to be more systematically integrated to advance new theory.

Contextual frameworks that recognize the complexity inherent in causal relations, such as sufficient and component causes (Rothman, 1986) and causal background context (Edwards, 2000), have been suggested for health research. Taking up the shortcomings of a strict doctrine of causality, Rosenberg (1968) created a framework for investing antecedent, intervening, conjoint, and conditional relationships. The methodological challenge is to find ways to examine the complexity in the analysis of data.

Over the past several decades, options have become available that facilitate the quantitative elaboration of complex influences in analyses that examine how relevant variables in a statistical problem relate to each other (Arminger, 1993; Cox & Wermuth, 1996; Edwards, 2000; Greenland, Pearl, & Robins, 1999; Wermuth, 1993; Whittaker, 1990, 1993). Graphical

models are a broad class of statistical model portraying association structures in conditional independence graphs. Graphical chain models may be considered a series of regression models, which, in contrast to simple regression models, are concerned with identifying relationships among all study variables, including those usually considered explanatory or control variables (Didelez, Pigeot, Dean, & Wister, 2002). Thus, with graphical models, the context of a relationship between a hypothesized cause and outcome can be studied. These newer methods facilitate both the analysis of complex relationships and the identification of hidden relationships, in some applications without the need for limiting parametric assumptions. This makes it possible to consider antecedent and intervening relationships that affect statistical connections between test variables and outcomes.

Mathematical and statistics journals have published articles on graph theory and applications for over two decades (see Cox & Wermuth, 1992; Darroch, Lauritzen, & Speed, 1980; Lauritizen & Wermuth, 1989). Yet, in spite of their potential for improving knowledge, these methods may be rejected in applications for health research and in the peer review of manuscripts for publication. This occurs because the methods do not fit orthodox beliefs, or because reviewers are not familiar with concepts and methodology used to explain the methods. At the same time, old methods are often accepted without question in spite of documented weaknesses and limitations. Nevertheless, innovative researchers are making progress in using new methods to advance knowledge.

Recognizing the lack of sound methodology to infer causal gene relationships, Waddell and Kishino (2000) used cluster inference methods and graphical models to study relationships among genes. Discussing the complex conditional distributions in cancer cell lines and their dependence on mutation status in these cell lines, they concluded that clustering based on partial correlations usefully identifies sets of genes for graphical modeling or other approaches to uncovering causal relationships which are currently clouded in complexity.

There are also innovative approaches to measuring outcomes. Neil-Dwyer, Lang, Smith, and Iannotti (1998) used a temporal graphical chain model to study the direct and indirect effects of possibly adverse influences on outcomes of subarachnoid haemorrhage. They found associations among the influences that helped to explain outcome, and determined that age was not related to either risk group or outcome.

Ruggeri, Bigger, Rucci, and Tansella (1998) used graphical chain models to study antecedent and intervening variables affecting mental health outcomes. They found that measures of psychological state, disability, and functioning at initial assessment predicted outcomes, with greater improvement in the more severely ill. At the same time, higher costs included in the model as an intervening variable predicted poorer outcomes at follow-up. It was concluded that the graphical models provided a useful methodology for understanding outcomes.

Graphical models have been used in a variety of research endeavours to gain knowledge about how multiple influences affect outcomes and how intervening variables change the statistical effects of predictors. They have been used to study chromosome mapping (Edwards, 1992), risk factors for coronary thrombosis (Whittaker, 1993), neonatal and post-neonatal mortality (Mohamed, Diamond, & Smith, 1998), the prognosis of head injuries (Sakellaropoulus & Nikiforidis, 1999), and nonhealth applications such as credit behavior (Stanghellini, McConway, & Hand, 1999).

In a methodological study (Didelez et al., 2002), graphical methods were compared with logistic regression in research on coping with chronic disease. While many findings were the same with both analytic approaches, there were differences that illustrate the potential of methods that identify indirect, partial, and hidden relationships. For example, the logistic regression found a strong, highly significant independent relationship between the type of illness and successful coping with three chronic conditions. Using graphical methods, it was shown that exercise, mutual aid, stress reduction, and perceived seriousness were responsible for (explained away) the relationship between the type of illness and coping. Exercise was positively related to coping regardless of the type of illness. The relationship between exercise and coping was stronger and highly significant for those persons who did not use mutual aid compared to those who did, but was not significant for those who reported extremely serious chronic conditions. It was concluded that graphical chain models can be used to expand and refine the information provided by traditional regression models, moving from the simplification of complex processes to the expression of their inherent multidimensional nature.

This does not mean that these methods are panaceas for causal understanding. The methods must be used appropriately and rigorously to avoid the dangers inherent in model selection from the large numbers of possible models in multivariate analytical problems. While looking away from multiple and moderating influences to highlight statistical effects of single test factors provides incomplete, really primitive information, statistical modeling demands careful analytic work by the researcher to avoid mis-specification of statistical connections. Just as with good qualitative research, indeed serious research of any kind, it is the knowledge of the researcher, logic and theory that must guide model selection in a statistical analysis. Statistical techniques are only tools for use in sound academic work and knowledge development.

Limited options for research and training in alternatives to the orthodoxy

The problems arising from adopting the beliefs of the orthodoxy are intensified when methods take on a force of their own. The result is that essentially no options become available for many researchers to learn about newer methods even though they may be far more relevant and useful for their research. The occasional options that do arise for learning about the new methods may be taken over by the power of the status quo. Research then becomes driven by orthodox thinking and traditional methods, in spite of their weaknesses, rather than being guided by the development of theory and the substantive issues in the existing body of research on a topic. When this happens, old ways of doing things are passed along to subsequent generations of researchers in methods courses that become stylized. Teachers who become experts in making traditional ways of thinking and conducting research seem certain and uncomplicated become popular. Since bringing up weaknesses in traditional approaches, or presenting new methods that may not be as easily understood, creates challenges to this popularity, questioning the old ways becomes heretical. Thus, traditional beliefs are defended, projects based on the traditional beliefs and methods are funded, and innovation is constrained.

In health research, these dynamics mean that discussions about limitations in the experimental model for understanding causal processes receive little attention. Traditional statistical methods for the analysis of population health data in research based on the paradigm may be taught and accepted with little questioning. It is not uncommon for standard regression models to be used with little concern for either violation of the measurement and mathematical assumptions on which the validity of the models depend or for the limits of the methods for analyzing the complexity inherent in most statistical relationships. These commonly used models may require parametric assumptions that are violated or they may implicitly assume, incorrectly, that relationships found do not differ for subgroups of the population (Greenland et al., 1999).

Beyond the barriers of orthodox beliefs

In the era dominated by the experimental model and quasi-experimental designs, statistical relationships found in research in this paradigm were inadvertently given a hardness and power that are not warranted. It is easily forgotten that the variables selected and how they are measured determine what is found, that simple changes in the values assigned to variable categories or the removal/addition of an uncertain statistical relationship can change the findings fundamentally. When variables that contribute causal influence, or those that intervene to moderate statistical associations among variables, are only controlled as parallel factors called confounders, interactions among the variables in the model are ignored. The results of these analyses will generally be inconsistent and may be misleading. The conceptual models presented in many studies are multicausal, but the statistical methods used to analyze the data tend to be appropriate only for simple or single cause models (Arnetz, 1996).

Research applications now available document the untapped promise inherent in statistical models that can elaborate complex relationships. To move beyond the barriers of orthodox beliefs, the analysis of data needs to focus on the complexity inherent in causal processes; a complexity which is often represented in theoretical and conceptual models. For example, aging is an area of study clouded in uncertainties arising from the neglect of research on complex relationships. Neil-Dwyer et al. (1998) found that age was not independently related to either risk group or outcomes of subarachnoid haemorrhage. Similarly, it is known that measurable declines in immune functioning, resulting in mild to moderate immune deficiency with advancing age, are not an adequate explanation for age-related increases in disease occurrence (Miller, 1991). The inconsistencies in evidence about aging declines in biochemical aspects of immune functioning, findings that many old people maintain immune responses comparable to young people (Candra, 1993; Saphire et al., 1993), and evidence of complex remodeling of immune functions (Franceschi et al., 1995; Hirokawa et al., 1994), suggest interactions among psychological, behavioral and environmental influences, and physiological variables that need elaboration.

Studies limited to specific agents preclude knowledge about the multicausal processes embedded in narrow relationships between a predictor and an outcome. Since available evidence indicates that maintaining health depends on the ability to adapt to or resist stress *and* to repair or replace damaged molecules (Kaplan, 1991; Martin, Danner, & Holbrook, 1993; Vogt, 1992), elaborating antecedent, parallel, and intervening influences will provide knowledge needed for more effective prevention and treatment of cancer and other major diseases.

More qualitative research is needed to provide relevant knowledge about psychosocial and behavioral variables, and to help evaluate quantitative measures before they are included in multivariate analyses. Psychosocial and behavioral variables need to be understood from the perspective of the layperson. Professionally constructed scales may have limited relevance for understanding psychosocial and behavioral variables embedded in causal relationships. In multivariate analysis, complicated scales often confound or

distort causal relationships (Dean, 2000; Dean & Salem, 1998).

These considerations do not mean that the experimental model is not important for some purposes. There are, of course, times when specific phenomena, when separated from the causal processes in which they are embedded, are usefully studied in controlled experiments. Randomized trials provide evidence contradicting findings suggesting that hormone replacement therapy and vitamin E reduce the risk of cardiovascular disease, and that beta carotene lowers the risk of lung cancer (Pocock & Elbourne, 2000). Still, the limits of such research for causal understanding need to be recognized. This is clearly illustrated by current discussions about recent findings that placebos have done as well as, or better than, antidepressant drugs in clinical trials conducted by drug companies (PsycPort, 2002). At the same time, there is evidence that suicide and attempted suicide rates do not differ for drug and placebo treated groups in clinical trials, and that placebo treatment produces changes in the brain that affect depression positively (Leuchter, Cook, Witte, Morgan, & Abrams, 2002). The latter findings are seen to support the continued use of placebo groups in clinical trials (Walsh, Seidman, Sysko, & Gould, 2002; Khan, Warner, & Brown, 2000). However, it would seem that far more useful knowledge would be gained by examining the relationships among placebos, antidepressants, and behavioral and environmental variables to identify direct and indirect effects of variables leading to depression and suicide.

Summary

The tenets and beliefs of the biomedical model have seriously constrained the knowledge available for promoting and protecting human health. Risk factor research on an endless array of disease agents or possible treatments/interventions has inherent limitations. Testing to see if the statistical effects of some variable remain after controlling for the influence of other variables may seriously underestimate or overestimate the true risk of important causal influences. This means that establishing a link between a factor and an outcome by holding constant moderating and mediating influences ignores the real nature of causation. A given study factor may (or may not) be the most important part of the processes affecting an outcome, but it is the interaction of the factor with other causes and with intervening and moderating influences that constitute causation.

In and of themselves, statistical relationships have no reality that extends beyond their usefulness for expanding our understanding of those subjects that are important for research and practice. The statistical analysis of data only provides information about how phenomena relate to each other when the variables are measured, and the data analyzed in specific ways. Stripping away so-called confounders does not alter the reality that the relationship occurs in the context of moderating and mediating causal influences. Both the quality of the measurements and the extent to which an analysis provides information about how relevant influences operate will determine the state of knowledge on a given subject.

It has now been over 20 years since Engel's (1977) seminal paper on the need to move beyond the narrow biological model of human health to a more sound and effective biopsychosocial model. There are pockets of research documenting the importance of social, psychological, and psychosocial variables for health and disease (Adler & Matthews, 1994). As in investigations of biological variables, psychosocial research, in spite of the complex models often displayed, generally examines the effects of specific variables. This occurs because of the limitations of readily available analytic methods and because of limited options for researchers to learn about alternatives. New education and funding priorities are needed to move beyond a biological or psychological or any other narrow model of human health. Challenging orthodox beliefs about health research will improve causal knowledge to better inform health care services.

The dysfunctional dichotomies arising from classical empiricism hang on in the form of false dichotomies that pit nature against nurture or posit micro over macro influences, or the reverse, on behavioral, health, or other types of outcomes. Contemporary life exposes people to interactions among an increasingly complex array of biological and psychosocial influences that can damage health. A biological model that concentrates on biological markers or single components of the causal processes that influence disease will unavoidably misguide health policy and practice (Davison et al., 1994; Dean & Hunter, 1996; Lancet, 1994; Smith & Torrey, 1996). Further research is needed in order to theoretically postulate how variables function together to cause phenomena; to study how measurement affects outcomes; to identify complex relationships and variables that modify causal connections; and to reject or modify a theory when findings suggest this theory can no longer be supported. Qualitative researchers have long decried the neglect of a deeper and more complex causal understanding in much quantitative research.

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