

## Theory as Mediating Variables: Why Aren't Community Interventions Working as Desired?

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**PURPOSE:** This paper discusses the role of theory in explaining why recent community intervention trials for chronic disease prevention are not achieving the level of desired behavioral effects and related outcomes.

**METHOD:** Literature review and analysis are used to derive an explanation.

**RESULTS:** All interventions (e.g., school nutrition education) effect change in behavioral outcomes (e.g., dietary behaviors) through mediating variables. Selected from the social and behavioral theories, these mediating variables can be environmental (e.g., increased availability of the targeted food) or intrapersonal (e.g., increased self-efficacy for eating the targeted foods). The percentage of variance of the outcome variables accounted for by the mediating variables has been modest to low. This places one limit on how much change interventions can achieve in outcomes. Another limit is imposed by the ability of the interventions to produce change in the mediating variables, which also has been weak.

**CONCLUSIONS:** More basic research should examine: (i) the relationships between mediating variables and behavior; and (ii) how interventions effect change in mediating variables. One possible six phase process for developing such research is described.

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**KEY WORDS:** Community Interventions, Health Education, Mediating Variables, Theory, Evaluation, Review.

### INTRODUCTION

A number of recent community and school intervention trials for chronic disease prevention have resulted in null or small effects (1-8). Although many important lessons have been learned from these trials (9) and some desired results have been found, the positive outcomes have been weak in comparison to the resources involved, including substantial funding, multiple years of intervention, large samples, use of state-of-the-art theory, sophisticated statistical models, and the expertise of leading health promotion researchers. Commentators have pointed to the difficulties in conducting such trials (10); argued that secular change has been substantial and perhaps has imposed an upper limit on behavior change, which is an inherently slow process (11); recognized the low power of many community trials (12); demonstrated that treatment delivery is complex (13) (Hearn M, Baranowski T. Doing what comes naturally:

Elementary school teacher fidelity to a behavior change nutrition curriculum. *J Nutr Educ.* (submitted)); recognized the modest reliability and validity of many behavioral and psychosocial measures that limit the ability to detect relationships (15); and admonished investigators to target more carefully the health problem, employ marketing strategies, and use an incremental approach to program design and development (15).

Biological, medical and behavioral scientists investigate similarly complex systems. The biomedical sciences, however, have proportionally more investigators working on elucidating mechanism, often called the "preclinical" phase, than on intervention. Biological and medical interventions generally target the mechanisms underlying a particular problem (often at multiple levels, e.g., organ, hormonal, cellular, subcellular). Perhaps health promotion researchers should similarly focus more on understanding underlying mechanisms and demonstrating that they can affect these mechanisms before developing treatment programs. Like a recent paper by Hansen and McNeal (16), this paper argues that: (i) interventions work by means of mediating variables; (ii) current theoretical models from which mediating variables are obtained often do not account for substantial variability in the targeted outcomes; (iii) interventions have not been shown to substantially effect change in the mediating variables; and, together, (iv) these factors impose limits on the effectiveness of the interventions. As a result, priority

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#### Selected Acronyms and Abbreviations

TRA = theory of reasoned action  
SES = socioeconomic status

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should be placed on research that enhances our understanding of the relations between theoretical variables and outcomes and the impact of interventions on these mediating variables.

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#### MEDIATING VARIABLE MODEL

The pathways from intervention to behavioral outcomes through mediating variables and without them are graphically depicted in Figure 1A. The statistical methods for determining mediating relationships have been discussed elsewhere (16-18). A mediating variable would account for the effect of an intervention if a positive relationship between the intervention and outcome were rendered nonsignificant after statistically controlling for the mediator (18). Figure 1A is a simplified graphic since there can be many mediating processes (e.g., several psychosocial variables: outcome expectations, self-efficacy, and modeling) or cascading sequences of mediating processes (e.g., a child's self-efficacy for asking for fruits and vegetables affects the likelihood that the child will ask for fruits and vegetables at home, which affects the availability of fruits and vegetables at home, etc.). Fundamental to this discussion is the belief that the important characteristics of behavior, mediating variables, and interventions can be adequately quantified.

The mediating variable model highlights the importance of theory in understanding community intervention results, since the mediating mechanisms for behavioral science are theoretical variables. The theories used to design the interventions specify the mediating processes. If interventions are demonstrated to have an effect ( $R^2_{I-B}$  in Figure 1A) that is not accounted for by mediating variables, theory has incompletely specified the mediating processes and needs further development. Elsewhere, this has been called "the law of indirect effect" (16). As in the biological sciences, as more mechanisms are identified, more points or types of intervention are possible.

The relationship of theoretical variables to behavior can be represented formally as Behavior =  $f$  (Theoretical Variables). This function,  $f$ , does not specify the number of variables nor the kinds of relationships (e.g., linear, additive, multiplicative, etc.). How well a theory predicts a behavior may be specified by the percentage of variance in the behavioral outcome accounted for by the theoretical variables ( $R^2_{M-B}$  in Figure 1A).  $R^2_{M-B}$  can be as small as zero or as

large as 1.0. An implication is that better theories have substantially higher  $R^2_{M-B}$  values.

In most behavioral theories behavior is affected by many variables, not only by the relatively small number of mediating variables. For example, behavior is partially determined by genetics (19) and other probably random effects over which we have little or no control (e.g., life events). When other variables account for a large percentage of variance in the behavior, there is less variance in behavior for mediating theoretical variables to predict. The upper limit of the proportion of variance in behavior accountable for by theoretical variables is unknown at this time, but is certainly less than 1.0.

Since the major goal of intervention is change ( $\Delta$ ) in behavior, these relationships can be expressed in terms of change (see Figure 1B). Thus, change in theoretical variables that are strongly related to behavior should result in more change in the behavior  $R^2_{\Delta M-\Delta B}$ . Since interventions work through mediating processes (16),  $R^2_{\Delta M-\Delta B}$  provides an upper limit to the level of change interventions can achieve. For example, an intervention based on a theory that can explain 70% of the variance in an outcome (i.e.,  $R^2_{M-B} = 0.7$ ) should permit more change in the outcome behavior than a theory accounting for 20% (i.e.,  $R^2_{M-B} = 0.2$ ).

A recent review of 21 studies cross-sectionally predicting consumption of dietary fat found the highest  $R^2_{M-B}$  with behavioral dependent variables to be 0.36; most values were in the range of 0.2 to 0.3 (20). An extensive review article (21) revealed that the theory of planned behavior substantially predicted behavioral intention (averaged  $R^2 = 0.409$ ) but was less predictive of behavior (averaged  $R^2 = 0.340$ ), and this predictiveness varied substantially by behavior (from averaged  $R^2 = 0.156$  for clinical, screening behavior up to averaged  $R^2 = 0.423$  for HIV/AIDS related behaviors). Predictiveness also varies by the theory employed and the demographic characteristics of the sample to which the theories are applied (22-29). The one exception to generally low predictiveness of theoretical models is where the investigators studied very narrow categories of foods (e.g., milk, regular sodas) (29). This pattern of findings suggests that existing theoretical or basic research accounts for relatively small percentages of variance in the target behaviors.

Knowing the mechanism, however, does not imply a knowledge of how to intervene effectively. The relationship of intervention to mediating variables  $R^2_{I-\Delta M}$  must also be considered. Figures 1A and 1B make no statement about what kinds of interventions are identified nor how many mediating variables are included. From a functional perspective, better interventions should attain more change in mediating variables. Reports of the impact of the intervention on mediating variables are seldom found in the literature. The ability of interventions to effect change in mediating variables places another limit on the impact of an intervention on the outcome behavior of interest.

Studies reporting  $r_{I-\Delta M}$  are not easily summarized. Esti-

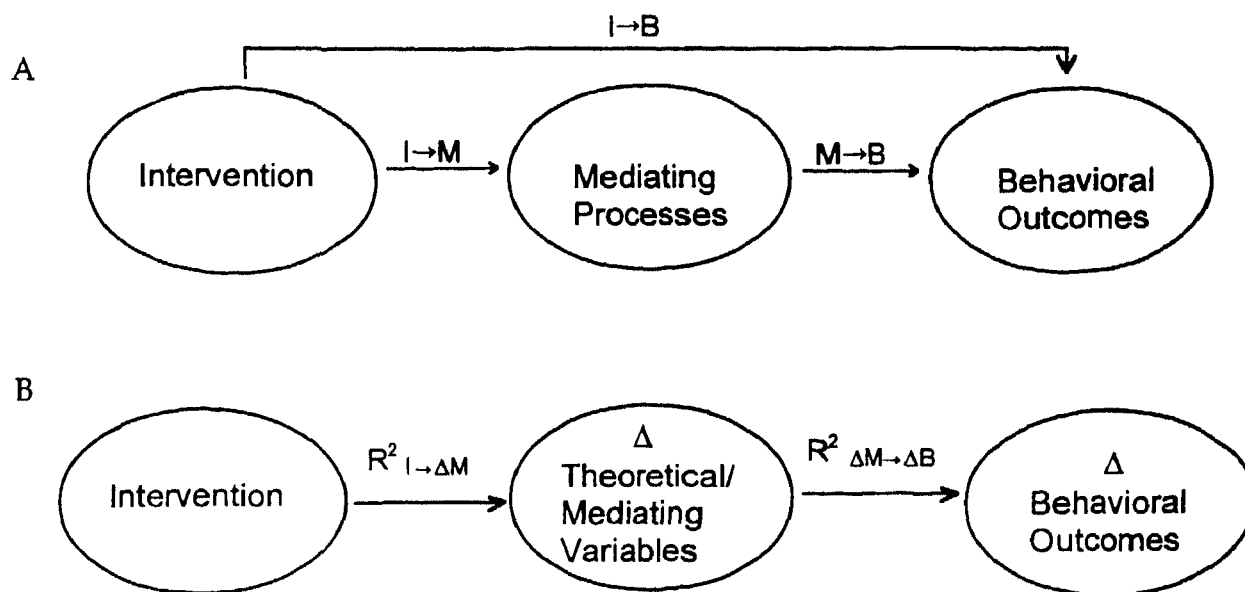


FIGURE 1. General framework for relating intervention to behavior (A) and mediating variable analysis of behavior change programs (B).

measuring the percentage of variance in the mediating variable due to the intervention is not clear cut or not reported in most of this literature. For example, one of the earliest studies of mediating variables demonstrated that a school-based multicomponent heart disease prevention intervention had statistically significant but weak effects on five mediating variables (30). A more recent related study with a much larger sample and more sophisticated statistical analyses demonstrated an impact at the end of the first year on all eight mediating variables, with the greatest change occurring in the group that had the most intensive (family involvement) intervention or condition (31). This condition, however, resulted in no more behavior change than a less intensive condition (8). In neither of these studies did the authors close the loop from mediator to behavioral outcome, apparently assuming that changed mediating variables resulted in behavior change. A school-based pilot intervention had only a small effect on fruit and vegetable preferences among a larger group of mediating theoretical variables (32), and none of these mediating variables were related to the small outcome effect. Another dietary study among adults demonstrated small increases only in negative-affect self-efficacy for reducing dietary fat, one among several hypothesized mediating variables (33). Change in negative-affect self-efficacy, however, was related to lower lipid values only at the assessment immediately after the intervention and was not related to the increases in cholesterol values at follow-up assessments.

Using structural equation modeling, a multicomponent intervention accounted for 44% of the variability in a set of mediating variables, after controlling for baseline vari-

ables, but these mediating variables accounted for only 9% of the variability in smoking onset, the outcome of interest (34). Another study that targeted the rate of weekly smoking by adding a media component to a school intervention revealed apparently weak intervention effects; however, the percentage of variability in mediators accounted for by the intervention was not reported (35). In another program, the intervention decreased the risk of smoking onset among those at high levels of four predisposing variables, but increased the risk of smoking with those at low levels of those variables (36). That is, what were first considered to be mediating variables turned out to be moderating variables. A multicomponent multiple risk behavior (cigarettes, alcohol, marijuana) intervention demonstrated an impact of the intervention on several (but not all) hypothesized mediating variables. Of eight hypothesized mediating variables, only one or two actually mediated the impact, and the specific mediating variables differed across risk behaviors (37). Thus, knowledge of how to impact mediating variables, and which are the most appropriate mediating variables, is in its early stages of development.

If the mediating variables predict behavior, then correlation coefficients reflecting the paths from the intervention to the mediators and from the mediators to the outcome behavior can be multiplied to equal the direct path of the intervention on the outcome behavior, and leads to the following inequality:

$$r_{IM} \cdot r_{MB} \leq r_{IB},$$

if the correlation between intervention and behavior is at least zero, once the mediating variable(s) is controlled for.

This inequality puts the intervention, mediating variables, and outcomes together and permits an analysis of the limits of an intervention on outcome. If the intervention is perfectly related to the mediating variables (i.e.,  $r_{IM} = 1.0$ ) then the upper limit on the effect of the intervention on behavior is the correlation of the mediating variables to the behavioral outcome (i.e.,  $r_{MB} = r_{IB}$  provided  $r_{IB/M} \geq 0$ ). If the mediating variables are perfectly related to the outcome ( $r_{MB} = 1.0$ ), then the limit to the effect of the intervention is the correlation of the intervention to the mediating variable (i.e.,  $r_{IM} \leq r_{IB}$ ). As the sampling of articles demonstrates, neither set of correlations involving mediating variables approaches 1.0, so both factors limit the relationship. If  $r_{IB} \geq r_{IM} \cdot r_{MB}$ , then the intervention is having an effect on the outcome behavior other than through the mediating variables, and further research needs to ascertain the nature of the mediating variables. The square of the product specifies the upper limit on  $r_{IB}^2$  due to the mediating variables.

As an example of the full model, we can generously allow  $r_{MB} = 0.5$  and  $r_{IM} = 0.5$ , so that the product of the correlations is at best 0.25, and the percentage of variance in the outcome behavior accounted for by the intervention is  $(0.25)^2$  or 6.25%, which is rather modest. When the effectiveness of an intervention requires a cascading sequence of effects among mediating variables (e.g.,  $I \rightarrow \Delta M_1 \rightarrow \Delta M_2 \rightarrow \Delta M_3$ ), then this multiplicative relationship becomes even more restrictive on the intervention's effect on outcomes. While any improvement in behavior is desired, the point is that the multiplicative relationship between  $r_{MB}$  and  $r_{IM}$  places limits on how much change can be achieved, requiring high component values for the component relationships to attain substantial change in behavior. Elsewhere, this has been referred to as the law of maximum expected potential effect (16).

## DISCUSSION

This brief review suggests two major reasons why interventions are not attaining the desired levels of change in behavioral outcomes. First, current theories do not fully predict behavior or behavior change. Second, interventions are not substantially effecting change in the mediating variables.

The low predictiveness of theory for behavior ( $R_{M \rightarrow B}$ ) specifies the vital importance of theory in the behavior change process and the necessity to invest substantially more effort into the further development and refinement of theories. One rarely sees a relationship between  $\Delta M$  and  $\Delta B$  in the literature. More commonly these relationships are estimated from cross-sectional data:  $M \rightarrow B$ . A possible problem in current theory is that the relationships currently estimated by cross-sectional data are really due to some common third antecedent variable and are really not caus-

TABLE 1. Tasks to be accomplished in each of the phases in the development of a health education intervention

Phase I.	Development of new ideas <ul style="list-style-type: none"> <li>• Clear conceptual statement of new ideas based on theory &amp; established results</li> <li>• Operationalization of construct(s)</li> <li>• Demonstration of reliability and construct validity</li> </ul>
Phase II:	Explanatory Research <ul style="list-style-type: none"> <li>• Necessary <ul style="list-style-type: none"> <li>Demonstrates the ability of the construct/measure to predict target behavior in some group of people</li> </ul> </li> <li>• Desirable <ul style="list-style-type: none"> <li>Establish the groups in which the construct/measure has and does not have predictive ability (e.g., age, gender, ethnicity, socioeconomic status (SES), psychographics)</li> <li>Test competing models on the same behavior</li> <li>Conceptually and empirically integrate concepts to develop more comprehensive models</li> <li>Assess stability/change of predictiveness at times of life transition</li> <li>Assess tracking across transitions</li> <li>Fully explore, conceptually and empirically, interaction or synergy between variables</li> <li>Carefully relate variables to process models of change</li> <li>Assess the relationship of naturally occurring change in theoretical variables to change in outcomes</li> </ul> </li> </ul>
Phase III.	Effecting change in mediating variables <ul style="list-style-type: none"> <li>• Necessary <ul style="list-style-type: none"> <li>Select mediating variables whose change is highly predictive of change in the target behavior in target group</li> <li>Develop and refine an intervention component to effect mediating variable change in target group</li> <li>Test progressively the extent to which the intervention affects the target mediating variable, then other related mediating variables related to the behavior of interest</li> <li>Build an overall intervention by developing, testing, and refining other complementary intervention components to affect mediating variable(s)</li> </ul> </li> <li>• Desirable <ul style="list-style-type: none"> <li>Demonstrate the effectiveness of the intervention on mediating variables in multiple groups (e.g., age, gender, ethnicity, SES, psychographics)</li> </ul> </li> </ul>
Phase IV.	Efficacy outcome intervention <ul style="list-style-type: none"> <li>• Necessary <ul style="list-style-type: none"> <li>For those phase III interventions demonstrated to effect change in mediating variables to some desired level in a target group, demonstrate that it affects outcome under field conditions that maximize exposure to the intervention</li> <li>Demonstrate extent to which mediating variables mediate outcome</li> <li>Assess how much change in outcome behavior is associated with a unit change in the mediating variable</li> </ul> </li> <li>• Desirable <ul style="list-style-type: none"> <li>Demonstrate the efficacy of the intervention across multiple groups (the generalizability of the intervention)</li> </ul> </li> </ul>
Phase V.	Effectiveness outcome intervention <ul style="list-style-type: none"> <li>• Necessary</li> <li>• Desirable <ul style="list-style-type: none"> <li>Demonstrate effectiveness in multiple channels</li> <li>Demonstrate effectiveness with multiple groups</li> </ul> </li> </ul>

continued

TABLE 1. *Continued*

Phase VI. Dissemination
<ul style="list-style-type: none"> <li>• Necessary For those Phase V interventions demonstrating effectiveness to a desired level in a target group as employed by research staff, demonstrate the effectiveness of the intervention when employed by others with the same target group, and assess the fidelity of the implementation</li> <li>• Desirable Assess dissemination to other groups</li> </ul>

ally related, or relations obtained in cross-sectional data are functionally different in longitudinal studies.

One may increase the potential for impact by selecting theories that show higher predictiveness of behavior. For example, the theory of reasoned action (TRA) has demonstrated substantial predictiveness, particularly when the behavior is eating a relatively restrictive category of foods (29). This suggests that interventions should be mounted to influence TRA variables in regard to restrictive categories of foods.

Clearly identifying those groups by demographic characteristics (e.g., gender, age, socioeconomic status, literacy), stages of change, psychographic factors (38), or health status (39), in which existing theories have high predictiveness may provide clues as to why these theories are not predictive in other groups. This would clearly define areas for intensive theoretical work.

Behavior likely has many determinants. The same behavior in different situations, e.g., eating lunch at school versus at grandmother's house, is likely to be susceptible to different influences. Some investigators have attempted to address the limits of existing theory by incorporating variables from multiple theories (40, 41). Similarly, some authors have called for a more thorough understanding of what affects the behavior of interest and for targeting interventions at those factors (42). A polytheoretical approach may enhance predictiveness of behavior and thereby increase the possible effectiveness of intervention. Perhaps separate interventions must be demonstrated to effect change in behavior in separate situations and later combined and tested together.

Intensive interventions with patients or those at high risk of disease have resulted in substantial behavior change (43, 44). Such programs may be successful because they are working with more highly motivated populations (because of the screening of potential participants for compliance to a regimen before the start of the trial) or the treatment intensity enables tailoring of the intervention to key behavioral or psychosocial factors. A multivariate analysis of diet after intensive intervention, not change in diet, accounted for only 25% of the variance (43). Process evaluation and behavioral mediating variable analysis of intensive clinical

interventions is an important arena in which to learn about effective components in interventions.

There is also a substantial literature on biological influences on behavior, e.g., genetic, neural, metabolic and sensory influences (45). Genetic factors can influence behaviors through many paths (19), including something as simple as influencing sensitivity to bitter tastes (46). Future research may stratify samples on the relevant genetic factors and assess the influence of psychosocial variables separately in the distinct groups. This may allow us to tailor behavioral interventions to genetic or other biological characteristics.

More research on enhancing the impact on mediating variables, the other limit on outcome effectiveness, is also needed. Some theories do not clearly facilitate behavior change. For example, unlike social cognitive theory, TRA historically was not developed within a change-promoting context and therefore does not clearly specify procedures for facilitating change. Another limitation is that intention is the primary predictor, but it is imperfectly related to behavior. This suggests that other variables mediate or moderate the relationship between intention and behavior. At this time, only attitude to the act and normative beliefs are available to influence intention; additional variables are needed to impact behavior. Triandis proposed that habit is a variable that constrains behavior change (47); however, from the perspective of intervention, this reasoning becomes tautological, since by definition the only way to affect habit is to change the behavior.

Outcome effectiveness research would benefit from investigators' demonstrating both that their selected theoretical variables adequately predict the target behavior and that their interventions effect change in these mediating theoretical variables at an acceptable (as yet undetermined) level. Almost a decade ago, an optimal sequence was proposed in the conduct of cancer control research (48) and in health promotion research (49). The time appears ripe to implement this sequencing in the conduct of behavioral intervention research. Table 1 introduces the general sequence and the necessary and desirable tasks to be accomplished at each phase to advance in the sequence. In contrast to the current process of initially developing comprehensive interventions for outcome evaluation, this sequence is incremental in its approach. The incremental sequence would quicken the feedback of findings into the research community. It would place higher priority and greater emphasis on the role of theory, thereby enhancing the science of health promotion. Research teams contributing to health promotion would not have to possess staff with skills in all the tasks necessary to conduct intervention outcome research. Such research should result in a larger body of basic knowledge and a firmer foundation for building interventions.

Research with existing data sets is needed to assess how much behavior change can be expected from specific increments in the mediating variable(s). This would enable cali-

bration of interventions to achieve sufficient change in mediating variables to expect some minimally acceptable level of behavior change.

Anticipating the problems of multiplicative relationships in cascading effects from intervention to outcome (with the even lower effect sizes from the multiple multiplications), interventions should ideally intervene to encourage change at each of the multiple points in the sequential effects to minimize the low resulting products.

Finally when outcome studies are considered for funding, it is particularly important to include the statistical analysis of mediating variables to establish whether the interventions worked as designed.

The result of these suggestions would be to refocus the research effort toward a better theoretical understanding of behavior and thereby to provide a firmer foundation for more effective interventions.

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