

How does the Health Action Process Approach (HAPA) Bridge the Intention–Behavior Gap? An Examination of the Model’s Causal Structure

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The health action process approach (HAPA) is attracting increasing interest from researchers who want to predict, explain, and change health-related behaviors. The main aim of this commentary is to examine the causal structure of the continuum version of the HAPA (HAPA-C), and, in particular, to consider the implications of this structure for the claim that the theory helps to bridge the intention–behavior gap. The article discusses, in turn, mediators of the intention–behavior relationship, additional proximal determinants of behavior, moderators of the intention–behavior relationship and implications for intervention. The commentary may contribute to a better understanding of the HAPA-C and to its further development.

L’Approche des Processus d’Action en faveur de la Santé (HAPA) suscite de plus en plus d’intérêt de la part des chercheurs qui souhaitent prédire, expliquer et modifier les comportements relatifs à la santé. L’objet de ce commentaire est l’examen de la structure causale de la version continuum de l’HAPA (HAPA-C) et, en particulier, de prendre en considération les implications de cette structure dans la prétention de la théorie à combler le fossé intention–comportement. L’article aborde successivement les médiateurs de la relation intention–comportement, les déterminants immédiats additionnels de la conduite, les modérateurs de la relation intention–comportement et les retombées pour les interventions. Ce commentaire peut contribuer à une meilleure compréhension de l’HAPA-C et à son futur développement.

INTRODUCTION

The health action process approach (HAPA; Schwarzer, 2008) is attracting increasing interest from researchers who want to predict, explain, and change health-related behaviors. In a detailed review of stage theories (Sutton, 2005), I argued that the HAPA, as tested to date, cannot be regarded as a stage theory like the transtheoretical model (TTM; Prochaska & Velicer, 1997)

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and the precaution adoption process model (Weinstein & Sandman, 1992). I pointed out that the HAPA has a similar structure to the widely used theory of planned behavior (TPB; Ajzen, 2002), that the HAPA can be regarded as an alternative to the TPB, and that future studies should directly compare the two models.

Although not explicitly in response to my comments, Schwarzer (2008) distinguishes between two versions of the HAPA, a stage version in which the stages are explicit and a continuum version in which the stages are implicit (see Weinstein, Rothman, & Sutton, 1998, for an explanation of the differences between stage and continuum theories). While I welcome this development, in my view it is not helpful to refer to the continuum version of the HAPA (call it the HAPA-C) as an implicit stage theory, for the reasons given in Sutton (2005). If the continuum version of the HAPA is regarded as an implicit stage theory, then the TPB and several other widely used social cognition models would also have to be regarded as implicit stage theories, and we would lose the clear and useful distinction between (explicit) stage theories, such as the TTM, and continuum theories, such as the TPB, which are fundamentally different in structure.

The main aim of this commentary is not to directly address the question of whether it is helpful to describe the HAPA-C as an implicit stage theory but rather to examine the causal structure of the model and, in particular, to consider the implications of this structure for the claim that the theory helps to bridge the intention–behavior gap. Some of the points I make are specific to the HAPA-C, and others also apply to similar theories such as the TPB.

As a convenient example, I will use a study of seat belt use among adolescent car passengers, which is labeled Study III in Schwarzer (2008). The study is reported in more detail in Schwarzer, Schüz, Ziegelmann, Lippke, Luszczynska, and Scholz (2007), along with three other longitudinal studies of other health-related behaviors that used essentially the same version of the HAPA-C. The model is shown in Figure 1, with the standardised coefficients obtained from structural equation modeling. A total of 298 students from six high schools in Warsaw completed questionnaires on three occasions over a 7-month period. Motivational self-efficacy, outcome expectancies, risk perception, and intention were measured at time 1, recovery self-efficacy and planning were measured at time 2 (4 weeks later), and seat belt use was assessed at time 3 (6 months after time 2). Seat belt use was measured using three items (“Within the last six months, how often have you used seat belts while traveling on the rear seat/front passenger seat/in a taxi?”).

MEDIATORS OF THE INTENTION–BEHAVIOR RELATIONSHIP

A key feature of this and other recent versions of the HAPA-C is that planning is specified as a mediator of the intention–behavior relationship.

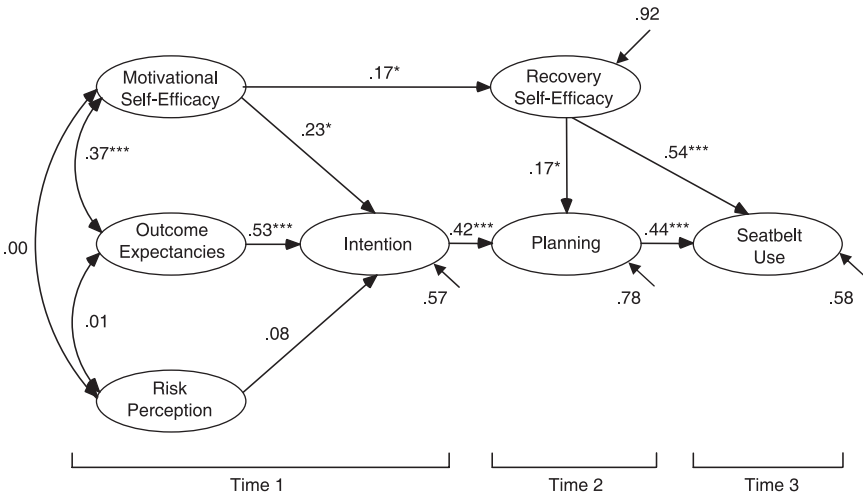


FIGURE 1. Path model to predict seat belt use, from Schwarzer et al. (2007).

Planning here refers to action planning. An example item used in the seat belt study is “I have had my own plan regarding when to fasten the seat belt (i.e. at which moment after getting into the car)”. Incorporating a mediator of the intention–behavior relationship distinguishes the HAPA-C from the TPB. To date, the TPB has not specified mediators of the intention–behavior relationship; in the TPB, intention is regarded as a proximal determinant of behavior (assuming that the behavior is at least partly under the person’s control). In Figure 1, planning completely mediates the effect of intention on seat belt use. Adding planning as a mediating variable bridges the intention–behavior gap in the sense that it explicates the causal mechanism by which intention influences behavior; that is, the model says that intention influences planning which in turn influences behavior. The implication of the model is that this causal process occurs automatically and does not require external intervention: those individuals with higher intention scores will, as a consequence, be more likely to engage in action planning; and those who engage in action planning will, as a consequence, use seat belts more frequently.

It is important to appreciate that including a mediator variable does not enhance the intention–behavior relationship. The size of the estimated causal effect of intention on behavior remains the same whether or not a mediator is included. In Model 1 in Figure 2, *a* is the standardised regression coefficient obtained from regressing behavior on intention. It is equal to the Pearson correlation coefficient in this simple case. Model 2 shows a model in which the intention–behavior effect is completely mediated by a third

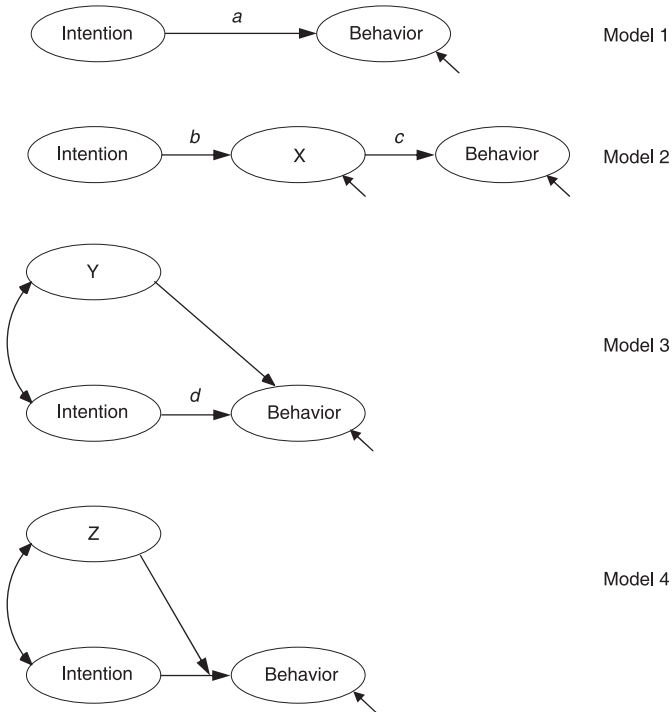


FIGURE 2. Simple path models illustrating the different ways in which an additional variable (X, Y or Z) can affect the intention-behavior relationship.

variable, X. In Model 2, b and c are the standardised regression coefficients (which are again equal to the corresponding correlation coefficients). There is a simple relationship between the three coefficients in Models 1 and 2: $a = b \times c$. Thus, including the mediator does not change the size of the effect of intention on behavior. However, it will generally improve the prediction of behavior: c will be greater than a , and a greater proportion of the variance in behavior will be explained in Model 2 than in Model 1.

ADDITIONAL DETERMINANTS OF BEHAVIOR

Recent versions of the HAPA-C, including the model shown in Figure 1, also specify recovery self-efficacy as a determinant of both planning and behavior. According to Schwarzer (2008), “recovery self-efficacy pertains to one’s conviction to get back on track after being derailed”. An example item from the seat belt study is “I am confident that I am able to resume regular use of seat belts, even if I had failed to use them a couple of times”. The

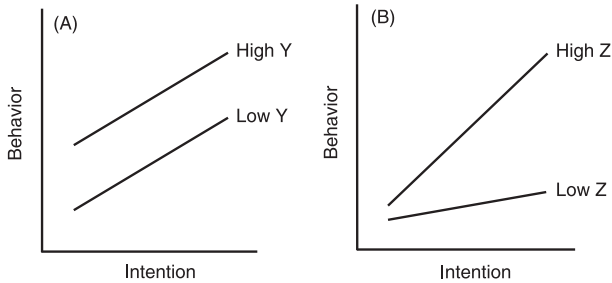


FIGURE 3. Line graphs showing the relationship between intention and behavior for groups of individuals with high and low scores on an additional variable (Y or Z).

effect of adding an additional proximal determinant of behavior can be illustrated by considering Model 3 in Figure 2, in which variable Y is specified as a second proximal determinant of behavior. If, in a real application, intention and Y have positive independent effects on behavior and the two predictor variables are positively correlated, then the estimated causal effect of intention on behavior (given by the standardised coefficient d) will be smaller than if Y is omitted from the model ($d < a$). In this sense, including an additional proximal determinant of behavior does not enhance the intention–behavior relationship. However, it will in general increase the proportion of variance explained in behavior. In other words, the proportion of explained variance will be greater in Model 3 than in Model 1.

There is a sense in which including an additional determinant of behavior explains the intention–behavior gap for some individuals. Figure 3A shows a line graph corresponding to Model 3 in Figure 2, assuming perfect linear and additive relationships. The two lines show the intention–behavior relationship for individuals with high and low scores on Y. Consider a person who scores high on intention but low on Y. Their predicted score on behavior will be lower than another person who has an equally high score on intention but who has a high score on Y. Thus, the first individual is predicted to have a larger intention–behavior gap than the second individual. Additive models are compensatory in this way: A high score on one predictor variable compensates for a low score on another predictor variable.

MODERATORS OF THE INTENTION–BEHAVIOR RELATIONSHIP

The size of the causal effect of intention on behavior may differ depending on the levels of other variables (“moderators”). To date, official versions of the HAPA-C have not specified such moderating variables. However,

planning, which is treated in the HAPA-C as a mediator of the intention–behavior relationship, could plausibly be postulated as a moderator of this relationship: People who form action plans may be more likely to translate their intentions into action. Furthermore, some of Schwarzer’s statements seem to imply moderating effects. For example, in relation to the seat belt study, he says that “The assumption was that intentions to use seat belts are not enough. Students need to have a clear idea, that is, a mental representation of being in a car wearing a seat belt. This is expressed by the planning items (‘when, where, how’)” (Schwarzer, 2008, p. 16).

There is some evidence for the moderating effect of action plans. For example, Jones, Abraham, Harris, Schulz, and Chrispin (2001), in a study on sunscreen use that was partly influenced by the HAPA, found evidence for both mediating and moderating effects of prior planning. A moderating influence of planning is also consistent with the rationale underlying implementation intention interventions, namely that asking people to form detailed action plans helps them to translate their goal intentions into action (Gollwitzer & Sheeran, 2006).

In simple terms, identifying a moderator of the intention–behavior relationship can be thought of as identifying a category of people in which the relationship is relatively strong and a category in which the relationship is relatively weak. Without the moderator, we would be estimating an average effect of intention on behavior. With the moderator, we are estimating different relationships for different levels of the moderating variable. Thus, including the moderator enhances the intention–behavior relationship for some individuals but reduces it for others.

As with the additive model discussed above, including a moderating variable can explain the intention–behavior gap at an individual level. In the line graph in Figure 3B, which assumes a particular form of interaction between intention and an additional variable Z, a person with high intention but low Z is predicted to have a low behavior score (i.e. to show a large intention–behavior gap); by contrast, someone with high intention and high Z is predicted to have a high score on behavior (small intention–behavior gap).

IMPLICATIONS FOR INTERVENTION

A causal model, such as that in Figure 1, has clear implications for intervention. The principle is that, in order to produce a change in a particular variable, it is necessary to change its specified causes. For example, in order to change seat belt use, we need to change planning and/or recovery self-efficacy; to change recovery self-efficacy, we need to change motivational self-efficacy; to change planning, we need to change intention and/or recovery self-efficacy; and to change intention, we need to change one or more of: motivational self-efficacy, outcome expectancies, and risk perception. Thus,

the variables on the far left of the diagram (the exogenous variables) assume special importance. Changes in any of these variables will work their way through the model and produce changes in seat belt use. (This is another reason why it is not appropriate to think of a model like this as having stages, even implicit ones. The causal processes involved in motivation and volition are part of the same causal model; indeed, put very simply, motivation causes volition.)

It is worth noting at this point that the version of the HAPA-C estimated by Luszczynska and Schwarzer (2003) in their study of breast self-examination specified risk perception as the sole exogenous variable. This implies that, in order to modify any of the variables in the model, it would be necessary to change risk perception.

Returning to the seat belt study, consider the effects of changing motivational self-efficacy. Assume that the target group consists of students like those in the seat belt study who presumably varied at time 1 with regard to frequency of seat belt use (some were using seat belts frequently, others less frequently, others not at all) and intention (some had relatively high scores on intention, some had relatively low scores). Further assume that we are able to increase motivational self-efficacy for a substantial proportion of participants by administering an intervention. According to the model, an increase in motivational self-efficacy would lead to an increase in seat belt use via three pathways: (1) It would lead to an increase in intention, which in turn would lead to an increase in planning, which would in turn lead to an increase in seat belt use; (2) It would lead to an increase in recovery self-efficacy, which would lead directly to an increase in seat belt use; (3) The increase in recovery self-efficacy would also lead to an increase in planning, which in turn would lead to an increase in seat belt use.

The coefficients in Figure 1 provide an estimate of the size of the causal effects. The total (standardised) effect of motivational self-efficacy on seat belt use is $0.147 [= (.23)(.42)(.44) + (.17)(.54) + (.17)(.17)(.44)]$ —much smaller than the direct effects of planning and recovery self-efficacy on seat belt use. Thus, an increase of 1 standard deviation unit in motivational self-efficacy would be predicted to produce an increase of about 0.15 standard deviation units in seat belt use. The corresponding total effects of outcome expectancies and risk perception on seat belt use are 0.098 and 0.015, respectively.

From the coefficients in Figure 1, it is also possible to calculate that the *effective variance explained* by the model (Sutton, 2002b, 2004)—that is, the percentage of variance in seat belt use that is explained by the three exogenous variables (motivational self-efficacy, outcome expectancies, and risk perception)—is about 4 per cent, a striking contrast with the 42 per cent explained by the proximal variables (planning and recovery self-efficacy). From the standpoint of assessing the potential of a theory as the basis for interventions, the percentage of variance explained by the variables on the far left

is more relevant than the percentage of variance explained by the theory as a whole or by the proximal determinants, because interventions have to be applied to the exogenous variables. The relatively low value for effective variance explained is not unique to the HAPA-C; it also applies to the TPB, and potentially to any theory that specifies a causal chain (Sutton, 2002b, 2004).

A different approach to intervention is to “jump into the causal chain” and to try to change a target variable without changing its specified causes. For example, one could target intention by asking participants to develop stronger intentions, or target planning by asking participants to make detailed action plans, or even target behavior directly by asking participants to increase their seat belt use. This approach is not informed by models of the determinants of the variable that is chosen for targeting, although it can be informed by models of the consequences of changing the target variable.

CONCLUSION

This commentary has examined the causal structure of the HAPA-C and discussed the ways in which the model does and does not bridge the intention–behavior gap. I hope that my comments contribute to a better understanding of the theory and to its further development. The constraint on space precluded discussion of several other relevant issues: The role of method and measurement factors in explaining the intention–behavior gap (Sutton, 1998, 2004); the assumptions involved in using non-experimental designs to test theories such as the HAPA-C and the TPB (Sutton, 2002a, 2004); and the need to consider equivalent models in applications of structural equation modeling (MacCallum, Wegener, Uchino, & Fabrigar, 1993).

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