Psychology of Sport and Exercise 19 (2015) 95–102

Contents lists available at ScienceDirect

Psychology of Sport and Exercise

journal homepage: www.elsevier.com/locate/psychsport

Habit as mediator of the relationship between prior and later physical activity: A longitudinal study in older adults

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ARTICLE INFO

Article history:
Received 2 May 2014
Received in revised form
20 March 2015
Accepted 21 March 2015
Available online 28 March 2015

Keywords:
Theory of planned behavior
Habits
SRHI
SRBAI
Physical activity
Mediation analysis

ABSTRACT

Objectives: Habit has been proposed as an explanation of why prior behavior is a good predictor of later behavior. This study examined whether habit mediates the relationship between prior and later physical activity (PA), within the framework of the attitude-social influences-efficacy (ASE) model and the theory of planned behavior (TPB).
Design: A longitudinal design was used.
Methods: A total of 1976 older adults completed questionnaires on ASE/TPB constructs and PA at baseline, intention at three months, habit at six months, and PA at twelve months.
Results: Path analyses showed that habit significantly mediates the relationship between prior and later PA, after ASE/TPB variables were taken into account.
Conclusions: Habit is a partial solution to the question why prior PA is a good predictor of later PA. It is recommended to incorporate habit into the ASE/TPB model.

Introduction

Although physical activity (PA) is an important contributor to physical and mental health (e.g. Chodzko-Zajko et al., 2009; Durstine, Gordon, Wang, & Luo, 2013; Vogel et al., 2009), a large proportion of adults aged 50 years or older are currently insufficiently active to obtain the health benefits associated with PA (World Health Organization, 2011). In order to be able to develop effective PA interventions for this age group, insight into the determinants of PA is indispensable. Two theoretical models that describe the processes wherein health behaviors are shaped and that have often been used to guide intervention development are the attitude-social influences-efficacy (ASE) model (De Vries, Backbier, Kok, & Dijkstra, 1995; De Vries, Dijkstra, & Kuhlman, 1988) and the theory of planned behavior (TPB) (Ajzen, 1991).

The ASE model contends that health behavior is governed by intention to act and self-efficacy, while intention, in turn, is determined by attitudes (i.e. pros and cons), social influences (i.e. social norms, modeling, and social support) and self-efficacy (De Vries et al., 1995). The TPB is largely comparable to the ASE model, although small differences do exist (De Vries & Mudde, 1998). One such difference concerns the inclusion of previous behavior in the model. Whereas the ASE model is open to include previous behavior (De Vries et al., 1995; De Vries & Mudde, 1998), the TPB rejects this suggestion, based on the assumption that the influence of prior on later behavior is mediated by the model's constructs (Ajzen, 1991). Nonetheless, both models are used without further distinction throughout this article, firstly because both models are conceptually closely related and complete each other in operationalization of the core concepts, and secondly because the research question addressed in this article is equally relevant for both models.

Meta-analytic studies on applications of the ASE/TPB model in a PA context have revealed that attitude, social influences and

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http://dx.doi.org/10.1016/j.psychsport.2015.03.006
1469-0292/

self-efficacy on average explain 42–46% of variance in intention, and that self-efficacy and intention on average explain 24–36% of variance in PA behavior (Godin & Kok, 1996; Hagger, Chatzisarantis, & Biddle, 2002; McEachan, Conner, Taylor, & Lawton, 2011). These numbers, although quite substantial, support the proposition that the variables in the model do not sufficiently predict and explain intentions towards PA and PA behavior (Conner & Armitage, 1998).

Based on the dictum that prior behavior is a good predictor of later behavior (Ajzen, 2011; Sutton, 1994; Triandis, 1977), prior PA has often been added to the ASE/TPB model to improve prediction of later PA (e.g. Abraham & Sheeran, 2004; Bozionelos & Bennett, 1999; Brickell, Chatzisarantis, & Pretty, 2006; Godin, Valois, & Lepage, 1993; Jackson, Smith, & Conner, 2003; Norman & Smith, 1995; Wang, 2011). Meta-analyses have shown that prior PA contributes 10–19% (Hagger et al., 2002; McEachan et al., 2011) to the prediction of later PA, in addition to the ASE/TPB variables. These findings contradict the assumption of especially the TPB model that the effect of prior on later behavior is fully mediated by the model's constructs (Ajzen, 1991, 2002). The residual impact of prior behavior has also been demonstrated in other health domains, such as alcohol consumption (Conner, Warren, Close, & Sparks, 1999), breast self-examination (Lechner, De Nooijer, & De Vries, 2004), sleep hygiene (Kor & Mullan, 2011), and breakfast consumption (Wong & Mullan, 2009).

It has been argued that residual effects of prior behavior on later behavior are due to shared method variance, as they are often assessed using the same measurement instrument (Ajzen, 1991, 2002). By contrast, two studies have shown that residual effects of prior behavior also exist when using different measurement instruments, indicating that these effects are not solely attributable to shared method variance (Conner et al., 1999; Verplanken, 2006). Furthermore, a statistical test by Bamberg, Ajzen, and Schmidt (2003) did not provide support for the shared method variance explanation. Should prior behavior thus be interpreted as a variable to be incorporated into the ASE/TPB model? Caution is warranted in giving prior behavior the same status as other ASE/TPB variables (Conner & Sparks, 2005). The ASE/TPB model, namely, is a causal model, meant both to predict and explain behavior (Sutton, 1998). Although it is beyond doubt that prior behavior often has predictive value, it is theoretically inadequate to contend that individuals perform a behavior *because* they have performed it in the past (Conner & Sparks, 2005). On an explanatory level the question thus remains why prior behavior predicts later behavior (Fishbein & Ajzen, 2010). This question is referred to as the *residual variance problem* (Ajzen, 2002).

Habits have often been proposed as a solution to the residual variance problem (e.g. Aarts, Verplanken, & Van Knippenberg, 1998; Sutton, 1994). Habits are defined as automatically enacted behavioral patterns in response to a context that consistently covaried with past performance (Wood & Neal, 2009). Although habits as an explanation of the residual variance problem may sound quite appealing, the mere existence of residual effects of prior on later behavior is not evidence of the existence of habits (Ajzen, 2002). Any configuration of factors that exerted an effect in the past and that continues to influence behavior at present could explain the residual effect (Ajzen & Fishbein, 2000). In order to accept habit as a valid explanation of the residual variance problem, Ajzen (2002) has set the conditions that, first, habit must be measured with a theory-based instrument that does not solely equate habit with past behavioral frequency, and, second, habit must mediate the relationship between prior and later behavior. From a practical point of view, meeting these two conditions implies incorporating habit into the ASE/TPB model and taking habit into account when developing interventions based on this model.

Although several calls have been made to test habit's hypothesized mediating role in the relationship between prior and later behavior (e.g. Smith et al., 2007), to our knowledge, only two studies conducted this test. In the first study, on travel mode choices, Bamberg et al. (2003) did not find support for the mediation hypothesis. In this study the response frequency measure of habit (see Verplanken, Aarts, Van Knippenberg, & Van Knippenberg, 1994) was used. This measure has been criticized for measuring generalized intentions or prior behavior generalized across situations, rather than habits (Ajzen, 2002). The Self-Report Habit Index (SRHI) (Verplanken & Orbell, 2003) overcomes this criticism. This reliable and valid instrument covers three features of habits, namely repetition, automaticity, and expression of one's self-identity. In the second study, Verplanken (2006) used this measure of habit and found that habit mediated the relationship between prior and later snacking behavior. Both mediation studies were based on the causal steps approach (Baron & Kenny, 1986), in which the mediation effect is logically inferred, rather than directly estimated (Hayes, 2013).

The present study aims to perform path analyses to examine whether habit mediates the relationship between prior and later PA within the framework of the ASE/TPB model, applying, in accordance with Hayes' (2013) recommendation, a direct estimate of the mediation effect. It is hypothesized that habit is a mediator of the relationship between prior and later PA.

Methods

The study was registered at the Dutch Trial Register (NTR920) and approved by the Medical Ethics Committee of Maastricht University and the University Hospital Maastricht. Informed consent was obtained from all participants.

Participants and procedures

This study was part of a clustered randomized controlled trial testing the efficacy of two interventions (i.e. a basic intervention targeting psychosocial determinants of PA and a *plus* intervention targeting both psychosocial determinants and PA opportunities in the environment in which the older adults lived) aimed at promoting PA behavior in adults, aged 50 years or older. These interventions proved to be effective in increasing levels of PA (days per week) at three (Cohen's *d* effect size $d_{basic} = .20$, $d_{plus} = .20$), six ($d_{basic} = .30$, $d_{plus} = .35$), and twelve months ($d_{basic} = .18$, $d_{plus} = .18$) after baseline measurement when compared to control participants (Van Stralen, De Vries, Mudde, Bolman, & Lechner, 2009, 2011).

Via six randomly selected Municipal Health Councils, 8500 Dutch adults aged 50 years or older, were invited to participate in the study. A total of 1976 adults (23%) agreed to participate and completed the baseline questionnaire. Of these participants ($M_{age} = 63.63$, $SD = 8.61$, 43% male, 51% meeting the PA recommendation) 30% were assigned to the control condition, 33% to the basic intervention condition, and 37% to the intervention plus condition. Retention rates at three, six, and twelve months were 74%, 71% and 68% respectively. The procedure of the study, including the selection, enrollment and dropout of participants, the distribution and content of the questionnaires, and the interventions are described in detail elsewhere (see Van Stralen et al., 2008, 2011).

Participants of both the control group and the two intervention conditions were included in the present study. To control for the influence of the interventions, all analyses were adjusted for treatment condition by the use of dummy variables. However, in order to eliminate any concern about possible residual intervention effects not controlled for by dummy variables, the analyses were