Aggression and Violent Behavior 29 (2016) 79–88

Contents lists available at ScienceDirect

Aggression and Violent Behavior

The role of dynamic risk factors in the explanation of offending

Tony Ward *, Clare-Ann Fortune

Victoria University of Wellington, New Zealand

ARTICLE INFO

Article history:
Received 30 March 2016
Received in revised form 9 June 2016
Accepted 16 June 2016
Available online 20 June 2016

Keywords:
Dynamic risk factors
Explanation of crime
Risk prediction

ABSTRACT

The focus of this paper is on what role dynamic risk factors should play in the development of explanations of crime. Following a discussion of the nature of explanation we propose that in their current form dynamic risk factors should not be regarded as causes of crime because they cannot be coherently conceptualized as causal mechanisms. We then examine the issue of how best to ascertain whether risk factors are causes and a number of methodological guidelines are suggested to assist in this evaluation process. Finally, we conclude that dynamic risk factors are valuable predictors of recidivism and that, additionally, suitably reconstructed they *can* serve an important methodological function in identifying the causes of crime and reoffending.

Contents

1. Introduction

Dynamic risk factors are the children of risk prediction. They were identified to help practitioners assess risk of recidivism and to set treatment targets likely to reduce reoffending (Andrews & Bonta, 2010). This

* Corresponding author at: School of Psychology, Victoria University of Wellington, PO Box 600, Wellington, New Zealand.
E-mail address: Tony.Ward@vuw.ac.nz (T. Ward).

http://dx.doi.org/10.1016/j.avb.2016.06.007
1359-1789/

resulted in the development of intervention programs designed to modify the characteristics of individuals and their environments associated with crime. The *predictive* nature of their legacy lies in their ability to provide reliable information about the likelihood of future reoffending. In this respect, dynamic risk factors are useful complements to static risk factors such as age, gender, and history of offending, and add incremental validity to recidivism prediction (Hanson & Harris, 2000). Their *treatment* utility resides in the fact that practitioners increasingly rely on the identification of dynamic risk factors to direct correctional assessment and interventions. In their book, *The psychology of criminal conduct*, Andrews and Bonta (2010) use the term "criminogenic needs" to refer to those dynamic risk factors that "when changed, are associated with changes in the probability of recidivism" (p. 49). In other words, they are viewed as potential causal factors that if effectively targeted by cognitive behavioral techniques will reduce reoffending rates. In a similar vein, Mann, Hanson, and Thornton (2010) identify a number of dynamic risk factors, which they believe to be prima facie causes of sexual offending and validated predictors of recidivism. They refer to these factors as *psychologically meaningful risk factors*, and propose that: "the causal factors for sexual recidivism will ultimately be drawn from variables similar to those included in our list. We believe that it is these variables that should be emphasized in treatment" (p. 210).

The theoretical legitimacy of incorporating dynamic risk factors into the domain of treatment depends on their causal status. There is little point in regarding them as intervention targets or using them to monitor treatment changes unless it is assumed that individually or collectively they are able to explain why individuals reoffend, presumably by virtue of their causal powers. For example, it is assumed that self-regulation deficits in some individuals' convicted of sexual offences cause them to engage in risky behaviors that ultimately result in further offences (Thornton, 2013; Ward & Beech, 2015). While the causal mechanisms resulting in offending are usually unspecified, an *association* between the presence of self-regulation deficits and sexual recidivism is viewed as a marker of causality. The assumption that dynamic risk factors are potential causes of offending is also evident in their integration into forensic case formulations, essentially conceptual models which depict a person's offence related presenting problems, the mechanisms that generate them, and their interrelationships (Hart, Sturmey, Logan, & McMurran, 2011). There have been two recent comprehensive reviews on the impact of changes in dynamic risk factors within treatment and concerning its impact on the social reintegration process (e.g., Cording, Christofferson, & Grace, 2016; Polaschek, 2016). Both of these papers note that while there does appear to be evidence that changes occur in the appropriate direction in dynamic risk factors, they raised concerns about their construct validity.

Thus, dynamic risk factors have a dual status. They are viewed as: (1) useful predictors of reoffending and measures of risk status, and (2) potential causes of reoffending, capable of serving an explanatory role as well as a predictive one. It is a simple and powerful conceptualization that has streamlined forensic and correctional research, program development, and the delivery of treatment.

Despite its conceptual elegance we believe that the dual conceptualization of dynamic risk factors is problematic and at best they can only function as *markers* of causality rather than being causes themselves (Ward, 2016; Ward & Beech, 2015; Ward & Fortune, 2016). If this assertion is true, then two questions arise: What if any role should they play in the development of explanations of crime (and reoffending) and how can they assist practitioners in treatment? In this paper we will concentrate primarily on the first question but will briefly address the second one as well. First, we argue that the focus of explanatory theories in the forensic and correctional fields ought to be on causal mechanisms. Second, we propose that dynamic risk factors should not be regarded as causes of crime because they cannot be coherently conceptualized as causal mechanisms, and in fact, are best viewed as composite or summary constructs referring to processes, states, and entities. Third, the question of how to move from risk factor to causal status is examined and a number of methodological guidelines suggested. Fourth, two conceptual frameworks for identifying the causal elements in crime related problems, based on classification research and therapy change mechanisms, is introduced and its application to dynamic risk factors outlined. Finally, we make some recommendations about how the field can facilitate the move from risk predictor to causal mechanism status, drawing from our earlier analyses. Our conclusions are that dynamic risk factors are valuable predictors of recidivism and that they *can* serve an important methodological function in identifying the causes of crime and reoffending. In the remainder of the paper we will speak generally of crime rather than recidivism or the onset of offending. While we appreciate that risk predictors were developed to estimate reoffending rates and status, it is reasonable to assume that they are also indicators of the causes of crime in general (see Andrews & Bonta, 2010).

It is important to understand that our goal is *not* to present a theory of dynamic risk factors or to outline their causal relationships to crime. There is a large body of theoretical and empirical research on the relationship between developmental, distal and proximal risk factors (etc.), and the onset and reoccurrence of offending (e.g., Farrington, 2016; Mulvey et al., 2016; Thornton, 2013, 2016). Our questions are more basic: Is the concept of dynamic risk factors theoretically coherent and in its standard form can it function as an explanatory construct in scientific explanations of crime? The paper is intended to focus researchers' attention on unnoticed theoretical problems in this foundational concept. The hope is that this will open up discussions concerning how to deal with these puzzles and ultimately to progress the field. It is not intended to be the last word on the matter and we respect and appreciate the work by researchers on the topic. It just that we believe our analysis can offer a *different* way of thinking about the relevant issues.

2. Causal mechanisms

Establishing causation requires careful reasoning (Illari & Russo, 2014). First, there needs to be evidence for an association between the outcome – a psychological or behavioral symptom – and a hypothesized causal factor (e.g., depression). Second, it is necessary to rule out a causal relationship in the opposite direction; a cause should precede the occurrence of the effect (e.g., determining that a factor is a cause of a depressive symptom and not a *consequence*). To do this, we might compare our group of interest to one with similar complaints with a known cause. Third, we need to consider whether other, intercorrelated factors may be responsible for the observed association (e.g., socioeconomic status). Of course, even if a direct causal relationship is established, there are limits to what can be inferred. For example, if cause A leads to problem B, this does not necessarily mean that every time B is observed, the cause must always be A. We cannot "reverse" the inference in this way, because there may be other causes of B as well. Finally, ideally a *causal mechanism*(s) that can mediate the pathway from A to B should be inferred and described (Bechtel, 2008; Craver & Darden, 2013; Thagard, 1999). It is not enough to simply state that there is a significant relationship between A and B. To be confident of a causal relationship and to be able to control and explain the outcomes, it is important to know *how* this occurs. Mechanistic explanations serve an important function by making the interactions between causal factors and phenomena much clearer.

Explanation in the life sciences and psychology differs from fundamental sciences such as physics by virtue of their increased focus on mechanisms rather than universal laws (Craver & Darden, 2013; Kaiser & Krickel, 2016; Khalidi, 2013). The reason for this stress on mechanisms is that the functioning of biological organisms is strongly influenced by contextual and local causal factors and therefore universal biological laws that apply across all times and places do not exist (although laws in the form of local generalizations that express causal relationships may be applicable-see Khalidi, 2013). In addition, organisms