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Aversive events and aggression Christopher L Groves and Craig A Anderson

There is perhaps no finding in psychology that is more consistent than the human motivation to avoid negative experiences and seek out positive ones. The current review details some of the aggression-related consequences that result from failures to avoid these negative experiences. Attention is paid to the theoretical processes at work that produce such effects. A review is conducted of the empirical literature detailing animal and human studies, in the lab and field. Lastly, we briefly discuss future directions in research that may advance our understanding of such effects.

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Aversive events

Many of our experiences, perhaps most them, can easily be judged as either positive or negative, with varying degrees of magnitude. A great deal of human motivation appears to address the seeking of positive experiences while avoiding negative experiences. In fact, there are few lessons in the study of psychology that are so basic and consistent. Despite the wealth of resources dedicated to these motivations, we sometimes fail to avoid unpleasant or aversive experiences. Here, we discuss the relation between such failures and aggressive responding.

First, we discuss the theoretical accounts of why and how aversive events produce aggressive responses. We then transition into a brief review of the empirical literature that documents the effects of specific aversive events and their effects on aggression. Our goal is to provide an overview of the study of aversive events and aggression, a topic which is integral to a holistic understanding of aggression.

Theories of aggression

One of the earliest theoretical accounts of the relation between aversive experience and aggression is called the frustration-aggression hypothesis [1]. In the book, *Frustration and Aggression*, theorists posited that aggression is the result of frustration, defined as when people are unable to reach goals or do not obtain expected rewards because of some external barrier [2^{••}]. The presence of an 'external barrier' is key to this definition, but emphasis is placed on the notion that the barrier must prevent the obtainment of an expected reward. So, for instance, although poverty serves as a well-known risk factor for aggressiveness [3], its effect should be limited to the degree that those living in poverty are unable to obtain sought after possessions, opportunities, or other goals.

The proposition that aggression is solely the result of frustration certainly led to progress in developing an understanding of aggression, but this hypothesis is not without its limitations. The hypothesis has difficulty, for example, explaining some of the more instrumental forms of aggression (*e.g.*, cutting in line). It is even more difficult for the hypothesis to explain more subtle effects of the mere presence of aggressive stimuli (*e.g.*, the weapons effect; [4,5]).

To solve some of these problems, newer theories were developed, such as cognitive neoassociation theory [6]. This theory focuses on the degree to which negative affect is elicited by the aversive event. This approach to understanding the effects of aversive events helps explain some findings that the frustration-aggression hypothesis could not, such as the role of arbitrariness in the frustrating event's elicitation of aggression [7–9]. For instance, if a bus drives past your stop with a sign stating that it is returning to the garage, the event is less likely to elicit aggression than if it drives by without the implied justification that it needs repairs. Critically, this theory also incorporates a knowledge-structure approach to understanding aggression. By knowledge structures we are referring to the network of interrelated concepts that reside in memory. For example, the concept of 'gun' is more closely related to the concept of 'kill' than the concept of 'cucumber'. These concepts are organized in useful ways such as in scripts, which is when we follow a standardized pattern of responses that are elicited from the social situation (e.g., when at a restaurant, we wait to be seated then order drinks, then food, then we eat, pay, and leave). It relies on research indicating that semantic memory is a network of relationships (with varying degrees of strength) between concepts, scripts, behavioral propensities, and affective reactions. When an aversive

event is experienced, it activates negative affect which, in turn, activates thoughts, feelings, and behavioral responses that are associated with both fight and flight tendencies [6]. In short, the aversive event triggers the activation of relevant knowledge structures that guide behavior.

Other theories focus on the role of attributions in the effects of aversive events on aggression. Excitation transfer theory [10] considers how arousal can increase aggression, especially when the source of the arousal is misattributed. Physiological arousal takes some time to dissipate, often longer than the individual believes. When the individual transitions into a new situation, they may misattribute the arousal that actually was derived from the previous situation to a feature of the new situation. For example, if someone is aroused by a situation (e.g., climbing stairs) and some feature of the subsequent situation is ambiguous, such as a comment that could be interpreted as insulting, the residual stair-climbing arousal may be misattributed to the ambiguous comment, exacerbating the perception of one's own anger and ultimately, one's likelihood of aggressing [11].

The theories we have described each contribute uniquely to specific types of aggression that could result from aversive experiences. In other words, theories such as excitation transfer theory help explain some types of aggression, whereas cognitive neoassociation theory helps explain other types. For this reason, modern social-cognitive theories incorporate and organize multiple theories into a broader framework that ultimately allows for a more complete understanding of, and ability to predict, aggression. The most recent and comprehensive of these theories is the General Aggression Model (GAM; [12[•]]; see Chapter 3 of this issue). The GAM incorporated the processes described by cognitive neoassociation theory, excitation transfer theory, and the frustration-aggression hypothesis. In addition to these theories, GAM incorporates other cognitive, affective, biological, and personal history factors to help explain aggressive responses (broadly, not solely in relation to aversive experiences). As a brief example, several factors such as beliefs about the likely outcomes of one's plan (outcome expectations), the perceived ability to execute a specific behavioral plan (e.g., fight someone; efficacy expectation), or beliefs about how normal an aggressive response is (normative beliefs), all help shape the likelihood and type of aggressive response. These processes (and others) are explicitly incorporated into the GAM. Such details are beyond the scope of this chapter but can be found elsewhere in this issue (see Chapter 3).

Empirical studies of aversive experiences

Next, we describe several research findings from a range of aversive events that have been show to increase aggressive behavior.

Provocation

Across all aversive experiences, perhaps the single most reliable elicitor of aggression is provocation [12[•]]. Provocations always involve interactions with a (real or imagined) second party and may include insults, physical attacks, thwarting of goals, passive aggression, or relational aggression (e.g., spreading rumors). One way to study the effects of provocation, across its multiple forms, is via meta-analysis. This approach involves the simultaneous statistical examination of a body of literature dedicated to a topic. For instance, in one meta-analysis, a large collection of studies on the effects of provocation on aggression found that provocation increased aggressiveness by nearly a full standard deviation (median Cohen's d = 0.86; [13]). The analysis found another interesting interaction with gender. Males tended to behave more aggressively than females in response to mild forms of provocation, but this gender difference disappeared for severe forms of provocation (*i.e.*, males and females were equally aggressive when severely provoked). This result emphasizes the importance of considering interactions between situations (e.g., the presence of a provocation) and characteristics of individuals (e.g., gender) in explaining aggressive behavior, as outlined in the GAM.

Pain

When asked to think of different types of aversive events, pain is likely to be the first example to come to mind. Research on pain in humans, however, is exceedingly difficult to conduct given the ethical considerations involved. For this reason, much of this research has been conducted on animals. A consistent finding across animal studies is that pain reliably elicits aggressive behavior. Most of these studies involved the use of rats, but similar findings have been observed in hamsters, gophers, monkeys, cats, chickens, snakes and even turtles [14]. In this literature, several factors have been found that reduce or intensify the pain-aggression response in animals (*e.g.*, size of cage, intensity, frequency, and duration of shocks).

Many of these investigations focused on varying the nature of the pain-inducing stimuli. Electric shocks were varied in duration, intensity, and frequency [14,15]. In these studies, researchers identified optimal 'windows' of effectiveness in the eliciting of aggressive responses. If the pain was too low, aggression was low. If pain was too high, aggression also was low. In fact, this finding is somewhat common in the study of aversive events on aggression. Low levels of aversive stimulation fail to effectively arouse aggressive responding, but at extreme levels of such stimulation, the participant is overwhelmed and wants to flee rather than fight back.

Of course, work on animals does not always generalize to humans (see Chapter 1 in this issue). Although ethical considerations preclude a rich and comprehensive study of pain and aggression in humans, researchers have found Download English Version:

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