



Here we go again: Bullying history and cardiovascular responses to social exclusion

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HIGHLIGHTS

- Previous research has linked social exclusion with blunted cardiovascular activity.
- I examined whether history of being bullied would moderate responses to exclusion.
- As predicted, chronic bullying victims showed blunted responses to exclusion.
- The blunting reflects a sympathetic reduction not a parasympathetic increase.
- This suggests that bullying victims develop a regulatory response to social stress.

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ABSTRACT

Previous research suggests that social exclusion—both acute and chronic—may be associated with a pattern of blunted cardiovascular responding. But it is unknown to what extent acute and chronic exclusion interact. That is, what happens when victims of long-term social rejection encounter an instance of exclusion later in life? The goal of the present study was to test whether prior experience being bullied would alter cardiovascular responses to an acute experience of social exclusion. Participants took part in a short online chat, during which they were either included or excluded from the conversation. Consistent with hypotheses, all participants showed an increase in sympathetic activity in the exclusion condition, but this response was significantly blunted among those with more chronic history of bullying victimization. No differences were observed for parasympathetic activity. This pattern suggests that a history of chronic victimization magnifies the cardiovascular “blunting” shown previously among victims of ostracism. This line of work suggests that bullying victims may develop regulatory mechanisms in response to social threats, and this may ultimately provide valuable information for helping victims become more resilient.

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1. Introduction

The victims of childhood and adolescent bullying are at higher risk for a number of outcomes, including depression, anxiety and suicide risk (see [8], for a review). Several recent studies have also examined the *long-term* consequences of being bullied. Findings from studies using both college student [14] and community [5] samples suggest that memories of adolescent teasing are positively associated with adult levels of anxiety, depression, and loneliness. Further, more extensive history of victimization during adolescence is associated with both increased self-reported stress [12] and increased use of avoidant coping strategies [13]. In addition, Newman et al. [13] report that avoidant coping partially mediates the link between victimization history and

current stress, suggesting that chronic victims may have prolonged stress because they did not learn effective strategies for coping with daily stress. Taken together, findings from these studies support the idea that bullying history has a lasting impact on stress and coping processes.

A growing body of evidence has linked chronic stress exposure to changes in cardiovascular stress responses. In a review of 19 studies, Gump and Matthews [21] reported that chronically stressed participants have altered levels of reactivity to episodes of acute stress. In some cases, this manifested as blunted reactivity, and in other cases as delayed recovery once the stressor was over. Only one study to date has applied this framework to the study of bullying victimization. After pre-selecting participants for a presence or absence of bullying history, Hamilton et al. [9] asked participants to prepare a speech that would be delivered in front of a group of faculty members. Males with a history of frequent bullying showed blunted blood pressure responses to this stressor, relative to males who had never been bullied.

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These findings mirror work in both human [21] and animal [22] studies of social stress, learned helplessness, and depression.

One way to make sense of these findings is by understanding how the brain might process the experience of being bullied. Although this question has not yet been tackled directly, analogies can be drawn to the work on social exclusion and ostracism. Because they threaten fundamental human motives [1,6], even short-term social exclusion and ostracism have been shown to almost universally induce negative mood, social withdrawal, and feelings of lost control (e.g., [3]; for review, see [19]). Several recent studies have extended this work to physiological processes, arguing that it would make evolutionary sense to have an “alarm” response to social exclusion. Eisenberger et al. [4] found that when participants were ostracized via a computer-controlled ball tossing game, they showed increased activity in the dorsal anterior cingulate cortex (dACC) and right ventral prefrontal cortex (RVFPFC). What makes these regions notable is that they are also involved in alarm responses to physical pain.

In a 2010 article, Moor, Crone, and van der Molen took this investigation one step further, studying the peripheral body responses associated with exclusion. Previous work had suggested that the ACC—part of the alarm system—was involved in parasympathetic control over heart rate. Thus, Moor et al. [11] hypothesized that social rejection would trigger the ACC, which would trigger the parasympathetic system, and lead to a slowing of heart rate. Results were consistent with this hypothesis: When participants experienced rejection (especially unexpected rejection), they showed a momentary increase in the *inter-beat interval* (IBI), meaning that the time in milliseconds increased between onset of individual heartbeats. This suggests that the parasympathetic system may kick in as an attempt to regulate negative affect in the face of rejection (cf., [16,17]).

Taken together, these studies suggest that social exclusion—both acute and chronic—may be associated with a blunted physiological response. But it is unknown to what extent acute and chronic exclusion interact. That is, what happens when victims of long-term social rejection encounter an instance of exclusion later in life? The goal of the present study was to test whether prior experience being bullied would alter cardiovascular responses to an acute experience of social exclusion. This study has the potential to expand existing knowledge in two areas: the lasting impact of being bullied, and moderators of the impact of social exclusion.

Specifically, I argue here that a history of being victimized by bullies leads the alarm system to become hypervigilant, scanning the environment for cues to rejection. Because this alarm manifests through parasympathetic activation [11,16,17], I suggest that a history of chronic victimization will magnify the cardiovascular “blunting” shown previously among victims of ostracism [11]. This idea was tested in a sample of college students who provided self-reports of their victimization history, and were either included in or excluded from an online chat room conversation. Cardiovascular responses to the exclusion were measured continuously in two ways, adding an important extension to the earlier study by Moor et al. [11], which measured the interbeat interval (IBI) as a measure of parasympathetic activity. An increase in IBI values indicates a slowing in heart rate, reflecting parasympathetic dominance. However, a decrease in IBI values indicates acceleration in heart rate, reflecting sympathetic dominance. Consequently, heart rate and IBI are actually controlled by both the sympathetic and parasympathetic systems, and slowing may reflect either increased parasympathetic activity or decreased sympathetic activity [2].

In order to verify that responses to social exclusion center on a parasympathetic response, the present study included measures that are linked directly to sympathetic versus parasympathetic activity: 1) the *pre-ejection period* (PEP), representing the time in milliseconds from the beginning of electrical stimulation of the ventricles to the opening of the aortic valve; and 2) *respiratory sinus arrhythmia* (RSA; an index of high-frequency heart-rate variability), representing the rhythmic

fluctuation of the heart rate in sync with breathing rate. Lower PEP values (a shorter time interval) indicate greater cardiac arousal, and thus represent an index of sympathetic activation. Following Berntson et al. [2], PEP values were multiplied by -1 in order to simplify interpretation. Thus, higher $-PEP$ indicates more sympathetic activation, while higher RSA values indicate more variability in heart rate, and thus represent greater parasympathetic activation.

Given the relative shortage of prior data, my hypotheses regarding these measures were partially exploratory. That is, I expected victimization history to moderate responses to acute social exclusion, such that chronic victims would show a *blunted* response relative to non-victims. But this blunting could manifest in one of two ways: *greater parasympathetic* activity, indicated by an increase in RSA during the exclusion period; or *reduced sympathetic* activity, indicated by a decrease in $-PEP$.

2. Materials and method

2.1. Participants

Participants in this study were 57 students¹ (70% F) at a large southwestern university ($M_{\text{age}} = 21.3$; $SD = 4.67$), who received partial fulfillment of a course research requirement. The majority of participants reported their ethnicity as Caucasian (58%); the remainder reported their ethnicity as Hispanic (14%), African-American (9%), and Asian (6%), with an additional 13% reporting “other” ethnicity.

2.2. Materials and procedure

2.2.1. Social exclusion manipulation

Participants were recruited for a study of “online communication,” and completed the experiment in a single session. Following a 2-min baseline recording, they were asked to take part in a 5-min online chat discussion, in which the other participants were pre-scripted and computer-controlled. We developed a conversational script through extensive pilot testing, covering topics that would be familiar to college students such as weekend plans, course schedules, and current pop culture. The presentation of this script was delivered using QuickKeys scripting software, which allowed us to adjust the speed of typing and the timing of sentences.² An additional advantage of QuickKeys was that it allowed the conversation to be paused in the event that participants expressed suspicion. For example, the occasional participant in the *exclusion* condition might type, “Can you even hear me?” into the chat window. The experimenter monitoring the session was able to pause the conversation and respond with “No, we hear you. Anyway, back to what I was saying ...”.

In the *exclusion* condition, there was a brief 30-sec period of introductions in the chat room during which the experimenter would greet the participants (in the role of another participant) and respond to questions. At the end of the introduction period, the experimenter started the conversation script, which involved two computer-controlled “participants” talking and ignoring the real participant.

In the *inclusion* condition, the QuickKeys script was modified to allow the experimenter to converse with the participant. The experimenter played the role of one active member of the chat room, and used a set of keyboard shortcuts to represent a more passive third member, whose comments were limited to statements such as “yes”; “totally”; “lol”; and “OMG.” In order to add variety and reduce suspicion, there were a total of twelve of these stock responses.

¹ Data from seven additional participants was dropped due to suspicion over the chat protocol. These were the first seven participants run, and all attributed their suspicion to the speed of the automated chat. We subsequently adjusted the speed and avoided further problems with suspicion.

² The author is happy to make this script available by request to interested researchers.

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