

Emotion Regulation Contagion: Stress Reappraisal Promotes Challenge Responses in Teammates

Christopher Oveis and Yumeng Gu
University of California, San Diego

Joseph M. Ocampo
University of California, Berkeley

Emily J. Hangen
Harvard University

Jeremy P. Jamieson
University of Rochester

The current research examined the interpersonal dynamics of emotion regulation in a stressful collaborative context. Little is known about how regulating one's own stress responses impacts teammates. In this article, we propose that individual efforts to regulate emotions can impact teammates for the better. We tested hypotheses arising from this claim using a dyadic experiment ($N = 266$) that assessed in vivo physiological stress responses during collaborative work (a face-to-face product design task) and then individual work (a product pitch to evaluators). Throughout the experiment, the manipulated teammate was randomly assigned to reappraise their stress arousal, suppress their emotional displays, or receive no instructions. The nonmanipulated teammate received no instructions in all experimental conditions. Stress reappraisal benefited both teammates, eliciting challenge-like physiological responses (higher cardiac output, lower total peripheral resistance) relative to the suppression and control conditions. These effects were observed during both collaborative and individual work. A mediation model suggested that face-to-face interpersonal effects of stress reappraisal fed forward to promote nonmanipulated teammates' improved stress responses during individual performance. Moreover, manipulated teammates' displays of positive and negative affect emerged as potential mechanisms for improvements in nonmanipulated teammates' stress responses in moderation analyses. Thus, participants benefited by interacting with a person who reappraised their stress as functional. This work has theoretical implications for the interpersonal dynamics of emotion regulation, and relevance for applied settings is also discussed.


Keywords: reappraisal, suppression, challenge, threat, teams

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Team performance contexts are stressful and ubiquitous. From group projects in educational settings to joint projects in organi-

zations, teams are presented with acute task demands (e.g., developing a marketing plan on a deadline), and team members must work together to marshal resources to produce optimal results under evaluative pressure. In stressful situations, individuals frequently seek to regulate their affective responses by changing cognitive processes, including reappraising situational factors (Gross, 2002), adopting a self-distanced perspective (Kross & Ayduk, 2011), or altering appraisals of bodily responses (Jamieson, Hangen, Lee, & Yeager, 2018). In fact, intervention techniques have been developed with the express intention of helping individuals regulate their affective responses in stressful situations via cognitive change (e.g., Brooks, 2014; Crum, Salovey, & Achor, 2013; Jamieson, Nock, & Mendes, 2012). However, little is known about how individuals' efforts to optimize their own stress responses might impact their teammates. For instance, if I engage reappraisal processes to improve my affective response in a stressful team performance context, might my teammates also benefit? Or might my regulatory efforts disrupt team cohesion and communication? To answer questions along these lines, the current research built on recent advances in emotion regulation theory and used the biopsychosocial (BPS) model of

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 Christopher Oveis and Yumeng Gu, Rady School of Management, University of California, San Diego; Joseph M. Ocampo, Department of Psychology, University of California, Berkeley; Emily J. Hangen, Department of Psychology, Harvard University; Jeremy P. Jamieson, Department of Psychology, University of Rochester.

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Correspondence concerning this article should be addressed to Christopher Oveis, Rady School of Management, University of California, San Diego, Wells Fargo Hall 4W120, 9500 Gilman Drive, MC 0553, La Jolla, CA 92093. E-mail: oveis@ucsd.edu

challenge and threat as an organizing framework to test the interpersonal dynamics of a stress reappraisal manipulation.

The BPS Model of Challenge and Threat

Scholarship on stress reappraisal grew out of the literature on the BPS model of challenge and threat, which provides a theoretical framework for understanding the interplay between cognitive, physiological, and motivational processes underlying stress responses in acutely demanding contexts (for reviews, see Blascovich & Mendes, 2010; Jamieson, Crum, Goyer, Marotta, & Akinola, 2018; Mendes & Park, 2014). A core contribution of challenge and threat theory is specifying the psychological processes underpinning stress responses in performance contexts. Specifically, in challenge and threat theory, appraisals of demands (e.g., perceptions of uncertainty, danger, and required effort) and resources (e.g., perceptions of familiarity, knowledge, skills/ability, dispositional factors, and social support) interact to determine challenge- and threat-type responses (Mendes & Park, 2014). Challenge states are experienced when appraisals of coping resources exceed perceived situational demands. Alternatively, threat manifests when perceived demands exceed resources.

Another central aim of challenge and threat theory is elucidating the biological pathways underlying different stress response patterns (see Mendes & Park, 2014 for a review). Informed by models of physiological toughness (Dienstbier, 1989), challenge and threat theory focuses on the sympathetic-adrenal-medullary (SAM) and hypothalamic-pituitary-adrenal (HPA) axes. Both challenge and threat responses are theorized to stimulate the SAM axis, but threat also activates the HPA axis (see Blascovich, 2013, for a review). Downstream in the cardiovascular system, challenge and threat responses are associated with specific patterns of physiological reactivity, which are used to index challenge and threat responses in vivo during acute stress (e.g., Blascovich, Mendes, Hunter, & Salomon, 1999; Hangen, Elliot, & Jamieson, 2019; Jamieson & Mendes, 2016). After individuals are engaged with a stressful situation, to differentiate challenge from threat, researchers often examine changes in cardiac output (CO) and total peripheral resistance (TPR; see Seery, 2011 for a review). CO indexes the amount of blood pumped through the cardiovascular system per minute, and TPR indexes resistance in the peripheral vasculature. Challenge is marked by an increase in cardiac efficiency (i.e., increased CO) combined with reduced resistance in the peripheral vasculature. This response pattern helps to deliver oxygenated blood to the brain and periphery to facilitate active coping. Threat, on the other hand, reduces cardiac efficiency as increases in vascular resistance limit blood flow to the periphery in anticipation of damage or defeat (see Figure 1).

These patterns of physiological responding have important downstream consequences. For instance, challenge and threat states exhibit differential motivational orientations: Whereas challenge is generally associated with approach motivation, threat elicits avoidance (e.g., Jamieson, Valdesolo, & Peters, 2014). Moreover, challenge and threat responses have direct consequences for health and well-being. In the short-term, threat impairs decision-making (Kassam, Koslov, & Mendes, 2009) and leads individuals to miss opportunities for growth (e.g., Crum et al., 2013). Over the long term, repeated physiological threat responses are associated with increased rates of cardiovascular disease, re-

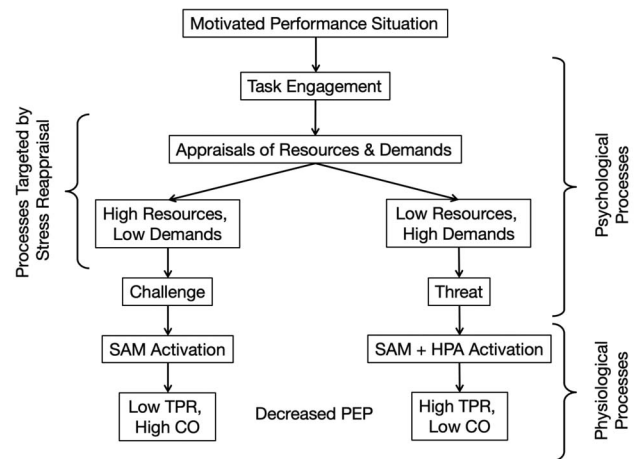


Figure 1. Overview of the psychological and physiological processes of the biopsychosocial (BPS) model of challenge and threat. Stress reappraisal seeks to promote challenge responses via highlighting the adaptive benefits of stress. SAM = sympathetic-adrenal-medullary axis; HPA = hypothalamic-pituitary-adrenal axis; PEP = preejection period; TPR = total peripheral resistance; CO = cardiac output.

duced immune function, and cognitive impairment (e.g., Jefferson et al., 2010; Lundberg, 2005; Matthews, Gump, Block, & Allen, 1997).

Stress Reappraisal

Because appraisal processes directly inform stress responses in the BPS model of challenge and threat, manipulating or modifying resource and demand appraisal processes has the potential to improve physiological stress responses and cognitive and behavioral outcomes. Indeed, a growing body of evidence from the emotion regulation and intervention literatures provides support for the idea that acute stress responses and performance outcomes can, indeed, be improved by modifying appraisal processes (Beltzer, Nock, Peters, & Jamieson, 2014; Brady, Hard, & Gross, 2018; Hangen et al., 2019; Jamieson, Mendes, Blackstock, & Schmader, 2010; Jamieson, Mendes, & Nock, 2013; Jamieson et al., 2012; Jamieson, Koslov, Nock, & Mendes, 2013; Jamieson et al., 2016; John-Henderson, Rheinschmidt, & Mendoza-Denton, 2015; Liu, Ein, Gervasio, & Vickers, 2019; Moore, Vine, Wilson, & Freeman, 2015; Rozek, Ramirez, Fine, & Beilock, 2019; Sammy et al., 2017; Yeager, Lee, & Jamieson, 2016).

The focal goal of stress reappraisal is to increase perceptions of resources by defining the stress response itself as a coping resource (e.g., Jamieson, 2017). Contrary to other stress management approaches, stress reappraisal is not aimed at convincing individuals that stressful situations are not demanding. That is, reappraising stress does not decrease perceptions of effort required to address stressors. The focus of stress reappraisal manipulations on resource appraisals is an important mechanistic distinction when individuals encounter acutely stressful situations that cannot be avoided or mitigated, such as team performance situations. For example, in many vocational contexts, employees are assigned to work in teams to solve problems or develop products. These demanding situations have important consequences for performance reviews,

promotions, and sustained employment and are generally unavoidable. However, people who reframe the stress responses they may experience as functional have the potential to optimize their stress responses and potentially improve their task performance.

In addition, targeting stress arousal processes is an important distinction when seeking to measure effects of stress reappraisal in teammates because research has demonstrated that arousal can be socially transmitted (Thorson, West, & Mendes, 2018). For example, sympathetic arousal linkage has been observed in married couples discussing conflict (Levenson & Gottman, 1983), in mothers and infants when the mothers experienced social stress (Waters, West, & Mendes, 2014), and in cross-racial conversation partners while getting acquainted (West, Koslov, Page-Gould, Major, & Mendes, 2017). However, research has yet to examine the interpersonal effects of stress reappraisal—that is, whether stress responses can be improved via stress reappraisal instructions administered to an interaction partner.

Interpersonal Emotion Regulation Dynamics

Emotion regulation—attempting to influence one’s own or others’ affective responses (Gross, 1998)—is prevalent (English & John, 2013), and has direct implications for health decisions and outcomes (DeSteno, Gross, & Kubzansky, 2013; Ford, Karnilowicz, & Mauss, 2017). Since its conception, emotion regulation research has tapped into cognitive change mechanisms, and reappraisal processes in particular, to regulate affective responses (Gross, 2002). More specifically, cognitive reappraisal strategies seek to alter affective responses by “changing the way one thinks” (McRae, Ciesielski, & Gross, 2012). Reappraisal, however, is not a unitary process, but rather refers to a class of approaches that exhibit substantial variability, including reappraising attributes of the situation, one’s placement in a situation, or bodily responses, to name a few (McRae, Jacobs, Ray, John, & Gross, 2012; Vishkin, Hasson, Millgram, & Tamir, 2020). The cognitive change processes of stress reappraisal focus on changing the underlying meanings of stress and the utility of stress responses (for a review, see Crum, Jamieson, & Akinola, *in press*). This regulatory aim is closely aligned with the emphasis on valuation processes in the extended process model (EPM) of emotion regulation (Gross, 2015).

Valuations assess whether an event/experience/situation is perceived as being “good for me” versus “bad for me” (Gross, 2015). Importantly for regulating stress responses, reappraising stress as functional and adaptive promotes the valuation that stress can be “good for me.” In this way, stress reappraisal may be applied to the EPM framework, which is important for considering the possibility that stress reappraisal can spill over to impact teammates in stressful situations. Notably, the EPM highlights that emotions are regulated within *dynamic systems*. Emotions (and stress) are not experienced in a vacuum; rather, temporal and social dynamics play an important role in determining how emotions are generated and regulated. For instance, helping people reappraise their stress responses in a social interaction setting can feed-forward to regulate affective processes in subsequent noninteraction settings (Jamieson et al., 2012).

In addition to studying temporal dynamics of emotion regulation, another burgeoning area of research emphasizes the *interpersonal* dynamics of emotion regulation processes (English & John,

2013; Reeck, Ames, & Ochsner, 2016; Zaki & Williams, 2013). Although research has yet to examine whether people might directly benefit from intraindividual regulation strategies enacted by teammates, extant research does suggest that emotion regulation processes, broadly construed, influence interaction partners’ affective responses (Butler & Randall, 2013). For instance, one line of research demonstrates the (negative) consequences of emotion suppression for social partners in nonperformance contexts (Butler et al., 2003; Peters & Jamieson, 2016; Peters, Overall, & Jamieson, 2014). Accompanying models, such as the Temporal Interpersonal Emotion Systems (TIES), highlight the importance of affective interconnectedness in interpersonal relationships (Butler, 2011). That is, emotional experiences that occur in the context of social interactions and/or ongoing relationships may be conceptualized as interpersonal emotion systems (Butler, 2015). Thus, teammates’ emotional experiences in a collaborative performance task context can be construed as an interpersonal emotion system, and the regulatory strategies enacted by one person have the potential to directly impact their teammate’s functioning within the dynamic system. However, to date, research has yet to examine whether stress regulation techniques, such as stress reappraisal, can directly benefit social partners’ affective responses in stressful contexts.

Potential Mechanisms of Stress Reappraisal-Facilitated Contagion

The current research leverages theoretical advances in emotion regulation dynamics to test whether stress reappraisal facilitates contagion of stress regulation processes. More specifically, we posit that *intrapersonal* stress reappraisal can operate as a mechanism by which teammates exhibit improved stress physiology during joint performance. Targeting this aim has implications for the study of teams, social groups, and organizations, in which people coordinate to accomplish tasks, achieve goals, and meet deadlines. This aim is distinct from prior work on interpersonal emotion regulation, such as how people regulate their own emotions via social interactions (Williams, Morelli, Ong, & Zaki, 2018; Zaki & Williams, 2013), how people aim to regulate others’ emotions (Gneezy & Imas, 2014; Netzer, Van Kleef, & Tamir, 2015; Zaki & Williams, 2013), or the intrapersonal social consequences of regulating one’s own emotions (Butler et al., 2003; English & John, 2013; English, John, Srivastava, & Gross, 2012; Srivastava, Tamir, McGonigal, John, & Gross, 2009).

Following previous scholarship (Elfenbein, 2014), we use the term *contagion* to refer to an outcome (here, physiological stress responses) becoming similar across people via some mechanism (here, an emotion regulation manipulation). Affect contagion occurs through a variety of processes—Elfenbein (2014), for example, outlines 10—including those that involve imitation of a target person, taking the perspective of a target person, or appraisal processes related to exposure to a shared stimulus or a target person’s behaviors. Importantly, contagion is multiply determined—that is, several pathways can operate in concert to produce contagion of outcomes between people. This is particularly true for contagion occurring during face-to-face social interactions in which multiple channels of information (e.g., visual, speech, and context factors) may be attended to and processed with the potential to impact interpersonal outcomes. To examine how stress reappraisal could facilitate contagious responses in teammates, the

present research focused on three potential mechanisms. However, we emphasize that the three outcomes examined here should not be considered the only processes through which reappraisal contagion might function, and we caution against overinterpreting any null effects as evidence that a focal process is irrelevant for contagion effects.

First, we considered whether stress reappraisal facilitates contagion by altering social perceptions of the reappraiser. Two fundamental dimensions of social perception are perceptions of warmth and competence (Fiske, Cuddy, & Glick, 2007); it is possible that stress reappraisal could lead teammates to view the reappraiser as more competent (because of improved performance on tasks) or warm (because of enhanced positive affect and/or less anxiety). Perceiving one's teammate as more competent or warm may, in turn, increase appraisals of social coping resources, leading to more challenge-like stress responses.

Second, we considered whether stress reappraisal facilitates contagion by improving social connection between the reappraiser and their teammate. Whereas suppression is associated with social costs, reappraisal strategies tend not to impair relationship factors or impede social connection (English et al., 2012; English & John, 2013; Impett et al., 2012; Impett, Le, Kogan, Oveis, & Keltner, 2014; Srivastava et al., 2009). Improved social connection between teammates could lead to enhanced contagion via myriad pathways. For example, greater levels of closeness are associated with increased empathy (Cialdini, Brown, Lewis, Luce, & Neuberg, 1997), and increased social connection may lead to a richer representation of the close counterpart's experiences (Preston & de Waal, 2002). Improved social connection could thus heighten imitation of the stress reappraiser, accuracy or tendency in sharing a stress reappraiser's perspective, or the degree to which one feels socially supported by one's stress reappraising teammate, to name a few possible pathways toward contagion of stress regulation processes.

Third, we considered how stress reappraisers' affect displays might serve as mechanisms of contagion. One route through which affect displays could serve as a mechanism would be if stress reappraisers expressed more positive affect (PA) and/or less negative affect (NA). If teammates perceive this displayed affect, this could lead teammates to share in the reappraisers' affective experiences, facilitating a shared stress response profile. A second, distinct, route by which reappraisers' affect displays might facilitate contagion would be if stress reappraisers' displays of PA or NA had an outsized impact on teammates' stress responses because reappraisers' affect displays were viewed as more authentic. That is, stress reappraisers experiencing more challenge-like physiological responses and exhibiting positive affect may be perceived as more authentic by their teammates, relative to suppressors or controls experiencing threat-like physiological responses who "try to put on a good face" and force PA displays. And stress reappraisers' NA displays may be perceived as more authentic because they are more likely to be related to observable, external causes (such as poor teamwork), rather than internal stress processes. Indeed, suppression, but not reappraisal, is linked with perceived inauthenticity in previous research (English & John, 2013; Impett et al., 2012). In contrast, reappraisal techniques centered on changing perspective, such as stress reappraisal, are well-suited to preserving perceived authenticity (Anderson, Chen, & Ayduk, 2019). In addition, perceiving affect displays, specifically, as authentic

enhances perceptions of the expresser as trustworthy, a desirable team member, and a leader (Slepian & Carr, 2019). In the present research, we hypothesized that stress reappraisers' affect displays, when present, would map onto stress responses of their teammates, because their affect displays would be viewed as authentic, leading teammates to be more likely to be influenced by those displays.

Current Research

To examine the contagious effects of emotion regulation on stress responses, we designed a novel dyadic paradigm in which teammates engaged in two incentivized, demanding tasks: (a) collaborative work in which teams designed a new product, marketing plan, and pitch for the product; and (b) an individual performance task in which each team member pitched the product to evaluators. The manipulated teammate was randomly assigned to reappraise their stress arousal (stress reappraisal), suppress emotional displays (expressive suppression), or a control condition. The other, nonmanipulated teammate received no instructions and was unaware of instructions delivered to their partner. This design allowed us to determine if stress reappraisal implemented by one person could influence their teammate's stress responses when working together and afterward when working individually. A mediation model was used to determine whether reappraisal's interpersonal challenge-threat effects during face-to-face collaborative work could account for later effects during individual work. Finally, three potential interpersonal mechanisms of emotion regulation contagion—competence perceptions, social connection, and affective displays—were examined.

Method

Sample Size Determination

An *a priori* power analysis was used to determine sample size. Effect size for the main effect of condition on physiological stress responses was estimated based on previous emotion regulation research assessing challenge and threat responses with *in vivo* cardiovascular measures in dyads (Peters et al., 2014). Using an anticipated effect size of $d = 0.59$, G*Power indicated that 46 participants per cell (i.e., 276 total participants) would be necessary to achieve a target power level of .80. In anticipation of potential data loss, data collection was set to terminate *a priori* after data were collected from 300 participants (150 dyads).

Participants

Three hundred undergraduate students participated in same-gender, same-race/ethnicity dyads and received course credit. Members of each dyad did not know each other prior to the study. To achieve the desired sample size, we collected the data at two different research labs—71 dyads were run at University of California, San Diego, and 79 dyads were run at the University of Rochester—using identical procedures, physiological systems, and standard data scoring and analysis procedures using identical software; the research was approved by the institutional review boards of both universities.

Thirty-four participants were excluded due to unusable physiological data primarily due to movement artifacts and problems

with sensors ($n = 28$), experimenter error ($n = 4$), or because participants knew each other prior to the study ($n = 2$). Thus, the final sample ($N = 266$; 159 women, 107 men; $M_{\text{age}} = 20.86$, $SD_{\text{age}} = 2.99$, range = 18–45; 150 Asian, 114 White/Caucasian, two Latino/a) consisted of 44 control, 48 suppression, and 43 reappraisal dyads; in four of these dyads, one teammate's data was excluded as described above.

Design

Each dyad was randomly assigned to the control, suppression, or reappraisal condition. Within each dyad, one participant was randomly assigned to be the manipulated teammate and the other participant was randomly assigned to be the nonmanipulated teammate. Only the manipulated teammate received the experimental manipulation (see the *Experimental manipulation* section); the nonmanipulated teammate received no special instructions.

Procedure

Overview. Each critical portion of the protocol is italicized in this overview section (see [Figure 2](#)); more detail about each italicized phrase is provided in a subsequent section.

Upon arrival, participants were escorted to individual, private testing rooms, where they completed intake questionnaires, had physiological sensors affixed, and rested quietly for a 5-min *baseline recording*. After baseline, the manipulated teammate received the *experimental manipulation*, and then the manipulated and nonmanipulated teammate were brought to a single testing room and introduced. The two teammates first completed the 6-min *collaborative work task*, in which they engaged in stressful, face-to-face teamwork involving the design of a product pitch. Next, each participant completed a Trier-style *individual performance task*, in which they each presented a 3-min portion of a product

pitch to evaluators. This procedure allowed us to assess how challenge-threat contagion during face-to-face teamwork may support stress responses later during related individual work.

Baseline recording. While physiological signals were checked, each participant individually acclimated to the laboratory for 5 min and completed intake questionnaires. Participants then rested quietly while seated alone in a room for a 5-min baseline recording. After this, participants completed self-report measures on a tablet computer (see [online supplemental materials](#)).

Experimental manipulation. Only the manipulated partner received emotion regulation instructions, which were provided on a piece of paper and then read aloud by the experimenter. In the reappraisal condition, the manipulated teammate was instructed to reappraise their arousal during the collaborative and individual performance tasks as functional and beneficial, rather than harmful ([Jamieson et al., 2010](#)):

Before you begin developing the product pitch with your partner we'd like to provide you with some brief instructions designed to maximize your performance. People often feel stress in collaborative performance situations. Research shows that this stress does not harm performance, but rather helps people perform well. In fact, people who experience signs of elevated stress arousal, such as a racing heart, are more creative, collaborate more effectively, and deliver better product pitches compared to people who are calm and experience no stress arousal. So, please do not be concerned if you notice you are stressed during the collaborative task today. Simply remind yourself that your body's stress responses are helping you perform as well as possible.

In the suppression condition, the manipulated teammate was instructed to suppress all emotional displays and behaviors during the collaborative and individual performance tasks (e.g., [Peters & Jamieson, 2016](#)):

Before you begin developing the product pitch with your partner we'd like to provide you with some brief instructions designed to maximize your performance. People often feel stress in collaborative performance situations. Research shows that one way to ensure the interaction remains "on task" is to limit your displays of emotion. So, when developing your product pitch with your partner and during your presentation to the evaluators, try your best not to let your emotions show. In other words, behave in such a way that your partner and your evaluators do not know what emotions you are feeling. Remaining stoic will help limit feelings of anxiety and ensure your partner and the evaluators do not judge you negatively because of your emotional displays.

In the control condition, the manipulated teammate received no instructions. After the collaborative work task and prior to the individual performance task, the manipulated teammate received the manipulation instructions for a second time to refresh the experimental manipulation.

Collaborative work task. During the 6-min collaborative work task, the teammates designed a bicycle, a marketing plan for the bicycle, and a pitch for the bicycle. The goal of this design was to provide an ecologically valid, consequential, and stressful context in which the two teammates needed to constantly interact to produce a creative work output. The teammates were informed that the pitch would have two parts: Part 1 would describe the product and its features, and Part 2 would describe the marketing strategy, including advertising and budget (see [online supplemental mate-](#)

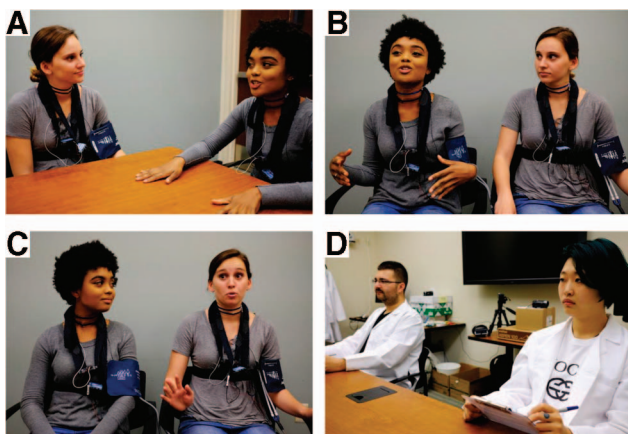


Figure 2. Procedure overview. The manipulated teammate received reappraisal, suppression, or control instructions (the nonmanipulated teammate received no special instructions). The two teammates then completed the collaborative work task (depicted in Panel A), during which they designed a product, marketing plan, and pitch. Next, each teammate completed the individual performance task by presenting Part 1 (B) or Part 2 (C) of the product pitch to evaluators who provided no positive verbal or nonverbal feedback (D). All teams were matched on gender and race/ethnicity. See the online article for the color version of this figure.

rials for more details). Critically, the teammates were told that they would not learn which teammate would deliver Part 1 or 2 of the pitch until after the collaborative work task. Thus, participants needed to work together during this period, rather than individually working on only the part they would present. The pitch was incentivized to increase engagement and evaluative pressure: Participants were informed that \$200 would be awarded to the best-performing team.

Individual performance task. Each teammate was randomly assigned to deliver Part 1 (3 mins) or Part 2 (3 mins) of the product pitch. This individual performance task was modeled on the Trier Social Stress Test (Kirschbaum, Pirke, & Hellhammer, 1993), which reliably elicits threat responses in individuals. To enhance signal quality, participants remained seated during the pitch and delivered their part to two evaluators (one male and one female) trained to withhold cues (verbal and nonverbal) of positive feedback.

Physiological Measures

The following signals were collected during baseline, collaborative work, and individual performance: electrocardiography (ECG) with a Lead II configuration, impedance cardiography (ICG) with band sensors, and blood pressure. ECG and ICG signals were sampled at 1,000 Hz, and integrated with an MP150 (Biopac Systems Inc., Goleta, CA). Blood pressure readings were obtained from the brachial artery on the non-dominant arm using an ambulatory system (Colin Medical Instruments, San Antonio, TX) and were taken at 2-min intervals during each recording period. ECG and ICG signals were ensemble into 1-min averages using Mindware software (IMP v3.0.21; Mindware Technologies, Gahanna, OH). Trained coders visually examined all B, Q, and R points for artifacts and corrected erroneous placements.

Analyses were conducted on the following measures: preejection period (PEP), CO, and TPR. These responses were used to distinguish challenge and threat states. Reactivity scores were computed by subtracting scores taken during the final minute of baseline (the “most relaxed” portion) from those collected during the first minute of the collaborative work task or the first minute of a particular participant’s portion of the individual performance task (the “most reactive” portion). This is a common approach in the social stress literature (e.g., Jamieson et al., 2012; Peters & Jamieson, 2016; Peters et al., 2014).

PEP is an index of sympathetic arousal and measures the time from the start of left ventricle contraction to the opening of the aortic valve. Shorter PEP intervals indicate greater contractile force of the heart and greater sympathetic activation. CO is the amount of blood ejected from the heart during one minute. An increase in CO indicates improved cardiac efficiency and is typically observed in approach-oriented challenge states. TPR is a measure of overall vascular resistance (calculated here as mean arterial pressure/CO * 80). An increase in TPR suggests a reduction of blood flow to the periphery, and accompanies threat states, whereas vasodilation (i.e., reduced TPR) facilitates delivery of oxygenated blood to the brain and periphery and is suggestive of challenge states.

Measures of Potential Interpersonal Mechanisms

Three potential interpersonal mechanisms of contagion in this context were measured: the nonmanipulated teammate’s perceptions of the manipulated teammate’s warmth and competence, the nonmanipulated teammate’s sense of social connection with the manipulated teammate, and the manipulated teammate’s behavioral displays of positive and negative affect.

Nonmanipulated teammate’s perceptions of the manipulated teammate’s warmth and competence. After the collaborative work task (just prior to the individual task), the nonmanipulated teammate rated the manipulated teammate’s competence (“competent/capable”) and warmth (“warm/friendly”) on 1 (*not at all*) to 5 (*very much*) scales (Cuddy et al., 2009).

Nonmanipulated teammate’s sense of social connection with the manipulated teammate. After the collaborative work task (just prior to the individual task), the nonmanipulated teammate rated how connected they felt to the manipulated teammate using the Inclusion of Other in the Self Scale (IOS; Aron, Aron, & Smollan, 1992). In each of seven options, two circles were presented with various amounts of overlap representing the connectedness of the two teammates. The nonmanipulated teammate chose one option to indicate the degree of connectedness that they perceived in the relationship with their teammate.

Manipulated teammate’s expressed positive and negative affect. To test whether contagion might manifest via emotions expressed by the manipulated teammate, independent raters coded emotional expressions during the 6-min collaborative work task. For each of 12 consecutive 30-s segments of the collaborative work task, 11 trained coders (blind to hypotheses and experimental condition) rated the extent to which the manipulated teammate expressed each of three positive emotions (“energized/excited/enthusiastic,” “proud/good about themselves,” and “happy/pleased/joyful”; $\alpha = .86$) and three negative emotions (“anxious/nervous,” “guilty/embarrassed/ashamed,” and “angry/irritable/frustrated”; $\alpha = .92$) on 1 (*not at all*) to 5 (*very much*) scales. Coders had access to both audio and video, and their ratings accounted for expressions in verbal and/or nonverbal channels (including face, voice, gaze, gesture, and posture). The 11 coders overlapped on 5% of the corpus of video recordings and showed adequate to excellent interrater reliability in their ratings—alphas: energized (.92), proud (.87), happy (.88), anxious (.83), guilty (.84), and angry (.63). Composites for PA expressed and NA expressed by the manipulated teammate across the entire collaborative work task were retained for analysis.

Results

Analysis Plan

To account for the nesting of participants in dyads, we built a two-level multilevel linear model (MLM) using the lme4 and lmerTest package in R. These models failed to identify significant variance at the level of the dyad during collaborative work—PEP: $\chi^2(1) = 2.17, p = .141$; CO: $\chi^2(1) = 0.64, p = .422$; and TPR: $\chi^2(1) = 2.96, p = .086$ —and during individual performance—PEP: $\chi^2(1) = 0.70, p = .404$; CO: $\chi^2(1) = 1.11, p = .291$; and TPR: $\chi^2(1) = 0.34, p = .557$, suggesting no need to model that nesting (see [online supplemental materials](#) for dyadic analyses).

Thus, primary analyses tested hypotheses separately on manipulated and nonmanipulated participants using analyses of variance and *a priori* planned contrasts, as described below.

Baseline

No baseline physiological differences (for PEP, CO, or TPR) were observed among the experimental conditions, all $ps > .17$ (see [online supplemental materials](#) for more details).

Collaborative Work Task

PEP reactivity. The collaborative work task (see [Figure 3](#)) elicited sympathetic arousal, as evidenced by PEP reactivity. Collapsing across conditions, participants showed a significant decrease in PEP ($M = -9.93$, $SD = 10.94$), $t(253) = -14.46$, $p < .001$, 95% CI $[-11.28, -8.58]$. No condition effects were observed for manipulated, $F(2, 122) = 0.49$, $p = .613$, or nonmanipulated teammates, $F(2, 126) = 0.18$, $p = .834$.

CO reactivity. For manipulated teammates, emotion regulation condition impacted CO reactivity (see [Figure 3A](#)), $F(2, 122) = 5.17$, $p = .007$. Those engaging in stress reappraisal exhibited greater increases in CO ($M = 0.33$, $SD = 0.76$) relative to both the suppression— $M = -0.15$, $SD = 0.86$; $b = 0.48$, $t(122) = 2.69$, $p = .008$, 95% CI $[0.13, 0.83]$, $d = 0.59$ —and control conditions— $M = -0.19$, $SD = 0.82$; $b = 0.52$, $t(122) = 2.88$, $p = .005$, 95% CI $[0.16, 0.88]$, $d = 0.65$ —which did not differ from each other, $b = 0.04$, $t(122) = 0.22$, $p = .823$.

The same pattern emerged for nonmanipulated teammates. Emotion regulation condition impacted CO reactivity, $F(2, 126) = 5.94$, $p = .003$. The reappraisal condition ($M = 0.24$, $SD = 0.64$) produced significantly higher CO reactivity relative to both the suppression— $M = -0.36$, $SD = 0.79$; $b = 0.60$, $t(126) = 3.42$, $p < .001$, 95% CI $[0.25, 0.95]$, $d = 0.83$ —and control conditions— $M = -0.14$, $SD = 1.03$; $b = 0.38$, $t(126) = 2.10$, $p = .037$, 95% CI $[0.02, 0.74]$, $d = 0.45$ —which did not differ from each other, $b = -0.22$, $t(126) = -1.21$, $p = .227$.

TPR reactivity. For manipulated teammates, emotion regulation condition impacted TPR reactivity (see [Figure 3B](#)), $F(2, 121) = 8.31$, $p < .001$. The reappraisal condition ($M = 57.17$, $SD = 57.17$) exhibited significantly lower TPR reactivity relative to both the suppression— $M = 172.40$, $SD = 159.45$; $b = -115.23$, $t(121) = -3.96$, $p < .001$, 95% CI $[-172.78, -57.67]$, $d = -0.83$ —and control conditions— $M = 141.07$, $SD = 118.00$; $b = -83.89$, $t(121) = -2.85$, $p = .005$, 95% CI $[-142.11, -25.65]$, $d = -0.73$ —which did not differ from each other, $b = 31.34$, $t(121) = 1.09$, $p = .280$.

For nonmanipulated teammates, emotion regulation condition also impacted TPR reactivity, $F(2, 124) = 20.96$, $p < .001$. The reappraisal condition ($M = -15.52$, $SD = 131.30$) produced significantly lower TPR reactivity relative to both the suppression— $M = 184.87$, $SD = 146.67$; $b = -200.40$, $t(124) = -6.28$, $p < .001$, 95% CI $[-263.60, -137.19]$, $d = -1.44$ —and control conditions— $M = 133.43$, $SD = 167.42$; $b = -148.96$, $t(124) = -4.53$, $p < .001$, 95% CI $[-214.04, -83.87]$, $d = -0.99$ —which did not differ from each other, $b = 51.44$, $t(124) = 1.59$, $p = .114$.

Individual Performance Task

The individual performance task (see [Figure 4](#)), which was modeled after the Trier Social Stress Test ([Kirschbaum et al., 1993](#)), was used to examine how emotion regulation condition influenced stress responses when faced with negative social evaluative feedback.

PEP reactivity. The individual performance task elicited sympathetic arousal, as evidenced by PEP reactivity. Collapsing across all conditions, participants showed a significant decrease in PEP ($M = -23.85$, $SD = 13.59$), $t(248) = -27.70$, $p < .001$, 95% CI $[-25.55, -22.16]$.

For manipulated teammates, emotion regulation condition impacted PEP reactivity, $F(2, 124) = 3.16$, $p = .046$. Manipulated teammates in the reappraisal condition ($M = -27.93$, $SD = 12.25$) showed significantly lower PEP reactivity (i.e., more SNS arousal) than manipulated teammates in the suppression condition— $M = -21.09$, $SD = 13.76$; $b = -6.84$, $t(124) = -2.51$, $p = .013$, 95% CI $[-12.23, -1.45]$, $d = -0.52$ —here, neither reappraisal, $b = -3.37$, $t(124) = -1.20$, $p = .233$, nor suppression, $b = 3.46$, $t(124) = 1.26$, $p = .209$, differed from the control condition ($M = -24.55$, $SD = 11.79$). For nonmanipulated teammates, emotion regulation condition did not influence PEP reactivity, $F(2, 116) = 0.002$, $p = .998$.

CO reactivity. For manipulated teammates, emotion regulation condition impacted CO reactivity (see [Figure 4A](#)), $F(2, 124) = 6.81$, $p = .002$. The reappraisal condition ($M = 1.59$, $SD = 1.46$) produced significantly higher CO reactivity relative to both the suppression— $M = 0.53$, $SD = 1.30$; $b = 1.06$, $t(124) = 3.48$, $p < .001$, 95% CI $[0.46, 1.66]$, $d = 0.77$ —and control conditions— $M = 0.70$, $SD = 1.5$; $b = 0.89$, $t(124) = 2.82$, $p = .006$, 95% CI $[0.27, 1.51]$, $d = 0.60$ —which did not differ from each other, $b = -0.17$, $t(124) = -0.56$, $p = .577$.

For nonmanipulated teammates, emotion regulation condition also impacted CO reactivity, $F(2, 119) = 5.75$, $p = .004$. The reappraisal condition ($M = 1.11$, $SD = 1.38$) produced significantly higher (more efficient) CO reactivity relative to both the suppression— $M = 0.53$, $SD = 1.30$; $b = 0.67$, $t(119) = 2.41$, $p = .017$, 95% CI $[0.12, 1.21]$, $d = 0.55$ —and control conditions— $M = 0.17$, $SD = 1.39$; $b = 0.94$, $t(119) = 3.27$, $p = .001$, 95% CI $[0.37, 1.51]$, $d = 0.68$ —which did not differ from each other, $b = 0.28$, $t(119) = 0.98$, $p = .331$.

TPR reactivity. For manipulated teammates, emotion regulation condition significantly influenced TPR reactivity (see [Figure 4B](#)), $F(2, 110) = 17.68$, $p < .001$. The reappraisal condition ($M = -4.39$, $SD = 191.82$) produced significantly lower TPR reactivity relative to both the suppression— $M = 217.69$, $SD = 163.39$; $b = -222.08$, $t(110) = -5.62$, $p < .001$, 95% CI $[-300.37, -143.80]$, $d = -1.25$ —and control conditions— $M = 182.02$, $SD = 160.06$; $b = -186.41$, $t(110) = -4.61$, $p < .001$, 95% CI $[-266.63, -106.20]$, $d = -1.06$ —which did not differ from each other, $b = 35.67$, $t(110) = 0.92$, $p = .361$.

For nonmanipulated teammates, emotion regulation condition significantly influenced TPR reactivity ($F(2, 111) = 17.06$, $p < .001$). The reappraisal condition ($M = -3.13$, $SD = 175.12$) produced significantly lower TPR reactivity relative to both the suppression— $M = 205.22$, $SD = 171.30$; $b = -208.36$, $t(111) = -5.05$, $p < .001$, 95% CI $[-290.08, -126.64]$, $d = -1.20$ —and control conditions— $M = 214.00$, $SD = 203.97$;

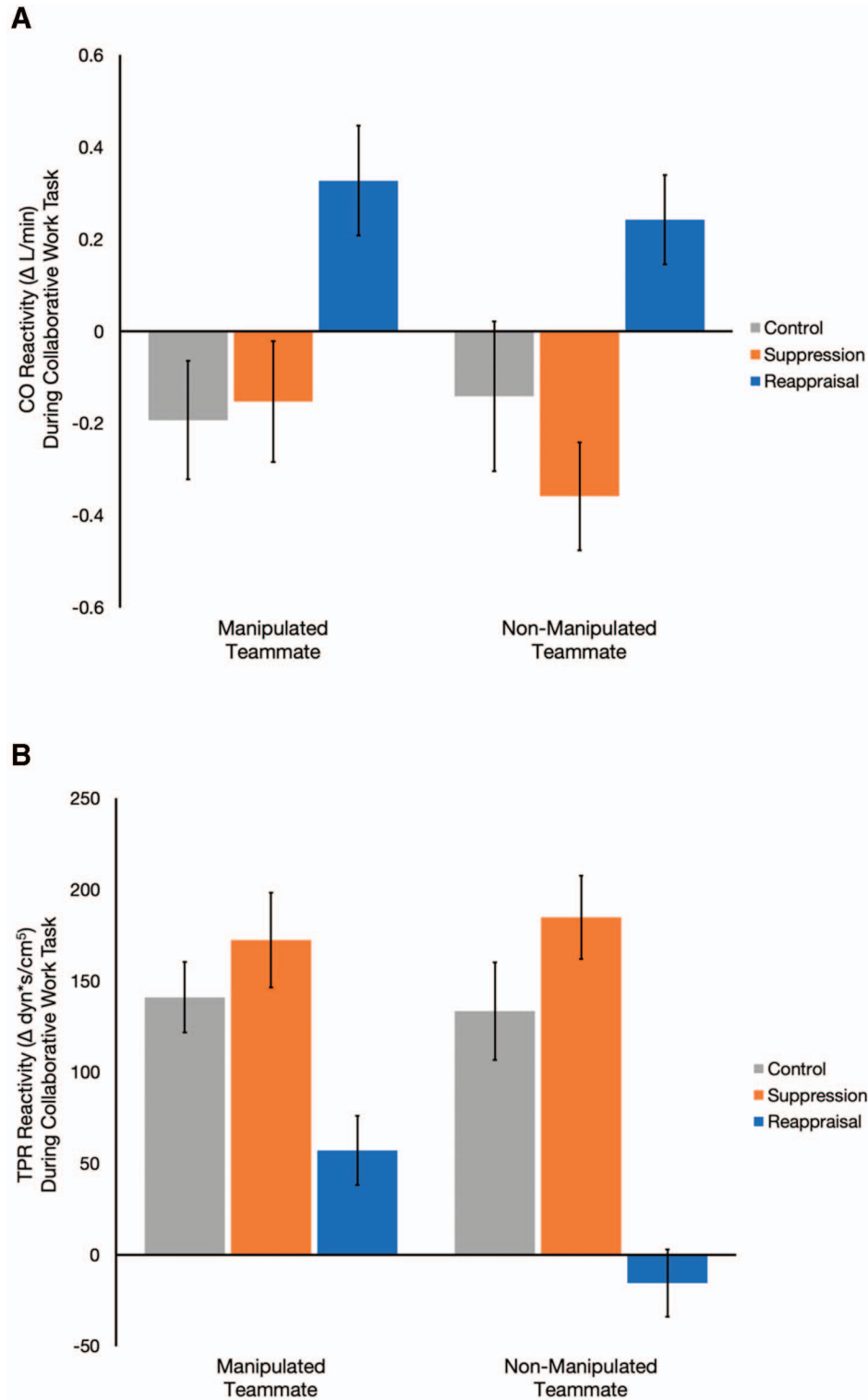


Figure 3. During collaborative work, manipulated and nonmanipulated teammates benefited from the stress reappraisal manipulation. When one teammate was manipulated to reappraise their stress, both the manipulated teammate and the nonmanipulated teammate showed more efficient cardiac output (A) and total peripheral resistance (B). Error bars represent one standard error. TPR = total peripheral resistance; CO = cardiac output. See the online article for the color version of this figure.

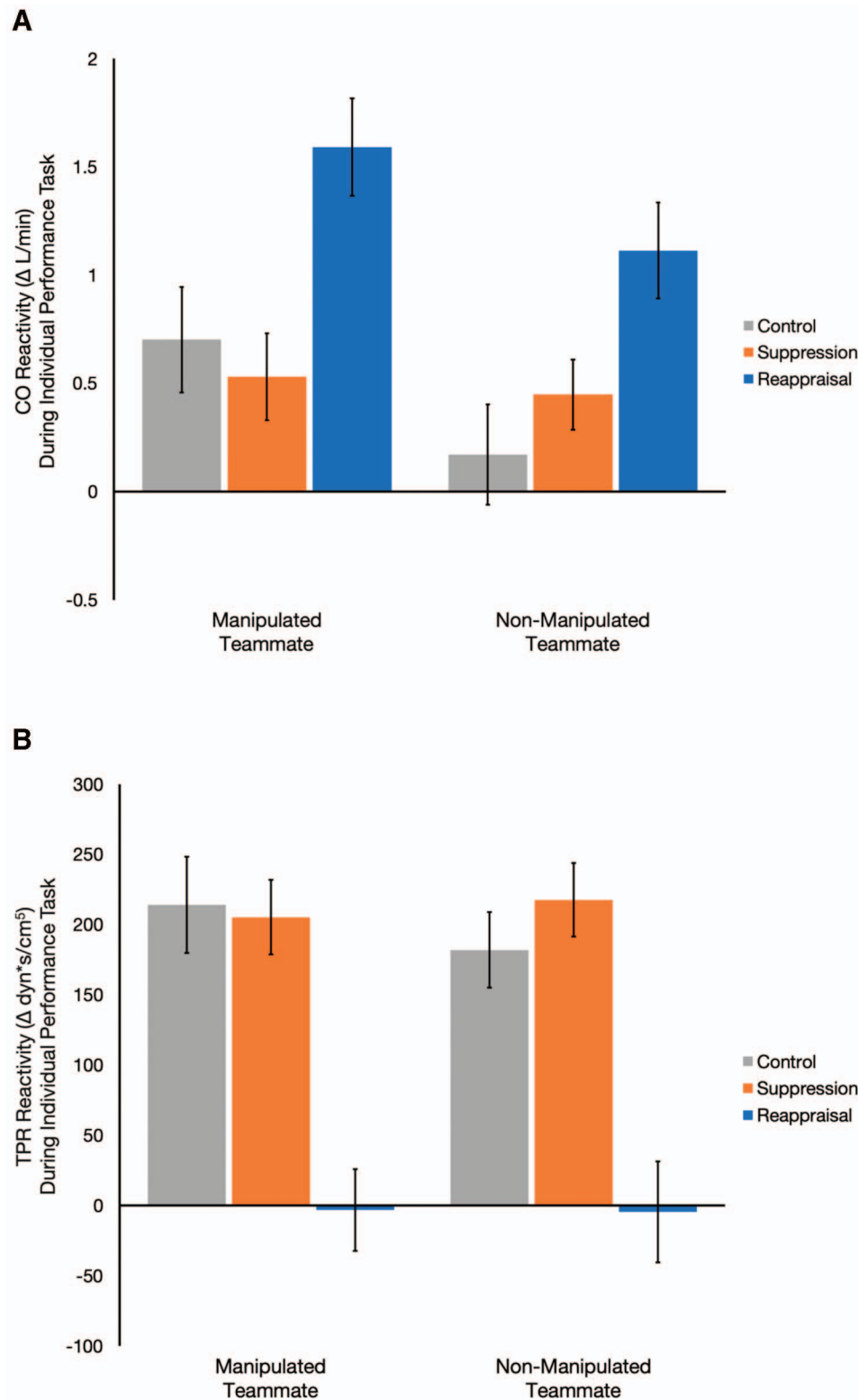


Figure 4. During individual work, a period in which the two teammates were not interacting face-to-face, nonmanipulated teammates continued to benefit from their prior interaction with a stress reappraising teammate. During individual work, both teammates once again showed more efficient cardiac output (A) and total peripheral resistance (B). Error bars represent one standard error. TPR = total peripheral resistance; CO = cardiac output. See the online article for the color version of this figure.

$b = -217.13$, $t(111) = -5.06$, $p < .001$, 95% CI $[-302.16, -132.11]$, $d = -1.14$ —which did not differ from each other ($b = -8.78$, $t(111) = -0.21$, $p = .835$).

Classification Analyses

Past research has shown that physiological reactivity to stress may show two distinct profiles: challenge (higher CO reactivity and low-to-no TPR reactivity) and threat (low-to-high CO reactivity and high TPR reactivity). In this section of results, we used untrained clustering algorithms to assess multivariate empirical clusters of physiological reactivity. We (a) attempted to identify multivariate physiological patterns consistent with challenge and threat, (b) investigated if these physiological clusters reliably distinguish the three conditions from each other using logistic regression, and (c) conducted training-test analyses examining whether training on the manipulated teammates could predict the condition of the nonmanipulated teammates.¹

We first assessed whether challenge and threat patterns of physiological reactivity could be empirically classified based on the multivariate cardiovascular data and found an affirmative answer to this question. We conducted cluster analysis using the k-means algorithm from the flexclust package (Leisch, 2006; v1.3–5 in R v3.5.1) to check for physiological signatures of challenge and threat, consistent with the literature on challenge and threat responses. Indeed, we found that there were two distinct clusters in the physiological data, and these two clusters were consistent with the literature in that Cluster 1 showed increased CO and lower TPR reactivity, whereas Cluster 2 showed the opposite pattern. Confidence in these clusters was confirmed via the Breckenridge (2000) procedure. Each participant was thus classified into one or the other cluster.

We next analyzed how condition affected the probability of cluster assignment to the challenge profile. This was done by using the classifications from the k-means algorithm fit to the entire dataset as the response variable in a logistic regression. Condition significantly improved the model fit compared to an intercept-only model, $\chi^2(2) = 21.12$, $p < .001$. Individual comparisons showed that the reappraisal condition significantly increased the probability of being in the challenge cluster compared to control ($b = 1.19$, $SE = 0.34$, odds ratio [OR] = 3.29, 95% CI_{OR} [1.70, 6.85], $\chi^2 = 12.61$, $p < .001$) and compared to suppression ($b = 1.41$, $SE = 0.39$, OR = 4.11, 95% CI_{OR} [2.03, 10.17], $\chi^2 = 15.81$, $p < .001$).

Third, to investigate whether the physiological signals trained on the manipulated teammates can predict the condition of the nonmanipulated teammates, we used the caret package (Kuhn, 2015; v6.0–80) and e1071 package (Dimitriadou, Hornik, Leisch, Meyer, & Weingessel, 2008; v1.7–0) to perform linear discriminant analysis (LDA). Here, the evidence was more equivocal, likely due to the sample size. The fit to the training set was limited—compared to the no information rate (NIR) accuracy of .34, LDA accuracy of .40 was not significantly better, $p = .116$, because it appears that the suppression and control conditions were difficult to tell apart. Nevertheless, testing the trained model on the nonmanipulated teammates showed a better fit than the no information rate: The accuracy of classification was .52 (95% CI [.43, .62]), which was better than the NIR of .35, $p < .001$. Further, the model was successful in classifying nonmanipulated teammates of reappraisers: 29/40 (72.5%) nonmanipulated teammates of reap-

praisers were accurately classified based solely on their physiological profile, whereas only 24/77 (31.2%) nonmanipulated teammates of suppressors or controls were false positives inaccurately classified as nonmanipulated reappraisers, $\chi^2 = 18.15$, $p < .001$.

Within-Team Comparisons

We did not find evidence that stress reappraisal had a different impact on manipulated versus nonmanipulated teammates' physiological stress responses. No significant differences were observed for manipulated reappraisers versus their nonmanipulated teammates on collaborative task CO, $t(81) = 0.97$, $p = .334$, collaborative task TPR, $t(79) = 1.99$, $p = .051$, individual task CO, $t(79) = 1.53$, $p = .129$, or individual task TPR, $t(70) = -0.52$, $p = .607$. We also explored whether there was a reversal in effectiveness of stress reappraisal on manipulated versus nonmanipulated teammates when going from collaborative task to individual task, but observed no significant Phase \times Role interaction on CO, $F(1, 160) = 0.92$, $p = .340$, or TPR, $F(1, 149) = 2.18$, $p = .142$. However, for this last analysis, we note that the ability to make comparisons of relative effects across collaborative and individual tasks is limited because the individual task was likely more demanding: Across participants, lower PEP was observed during the individual task ($M_{\text{reactivity}} = -23.88$, $SD = 13.66$) compared to the collaborative task ($M_{\text{reactivity}} = -9.89$, $SD = 10.99$), $t(235) = -16.51$, $p < .001$, 95% CI $[-16.04, -12.62]$.

Contagion Mechanisms

We tested two types of mechanisms of contagion. First, we examined whether face-to-face collaborative task effects fed forward to influence nonmanipulated teammates during individual work when teammates were not directly interacting. Second, we tested three potential mechanisms for how stress regulation processes may be transmitted between teammates.

Contagion across time: Individual task effects were mediated by collaborative task effects. After observing beneficial effects of stress reappraisal on physiological reactivity for both teammates across the collaborative and individual tasks (see Figure 5), analyses next focused on elucidating how stress reappraisal produced lasting effects on the nonmanipulated teammate after direct interaction with the manipulated teammate ended. Toward this end, we examined whether the nonmanipulated teammate's physiological reactivity during collaborative work mediated the effect of the manipulated teammate's stress reappraisal (vs. control) on the nonmanipulated teammate's physiological reactivity during individual work.

For CO reactivity, manipulated teammates' stress reappraisal predicted nonmanipulated teammates' collaborative task CO reactivity ($b = 0.45$, $SE = 0.20$, $p = .018$), and nonmanipulated teammates' collaborative task CO reactivity predicted nonmanipulated teammates' individual task CO reactivity ($b = 0.94$, $SE = 0.13$, $p < .001$). A mediation model with 20,000 bootstrap resamples indicated that nonmanipulated teammates' collaborative task CO reactivity mediated the relationship between manipulated teammates' stress reappraisal and nonmanipulated teammates' in-

¹ Full details can be found in the [online supplemental materials](#).

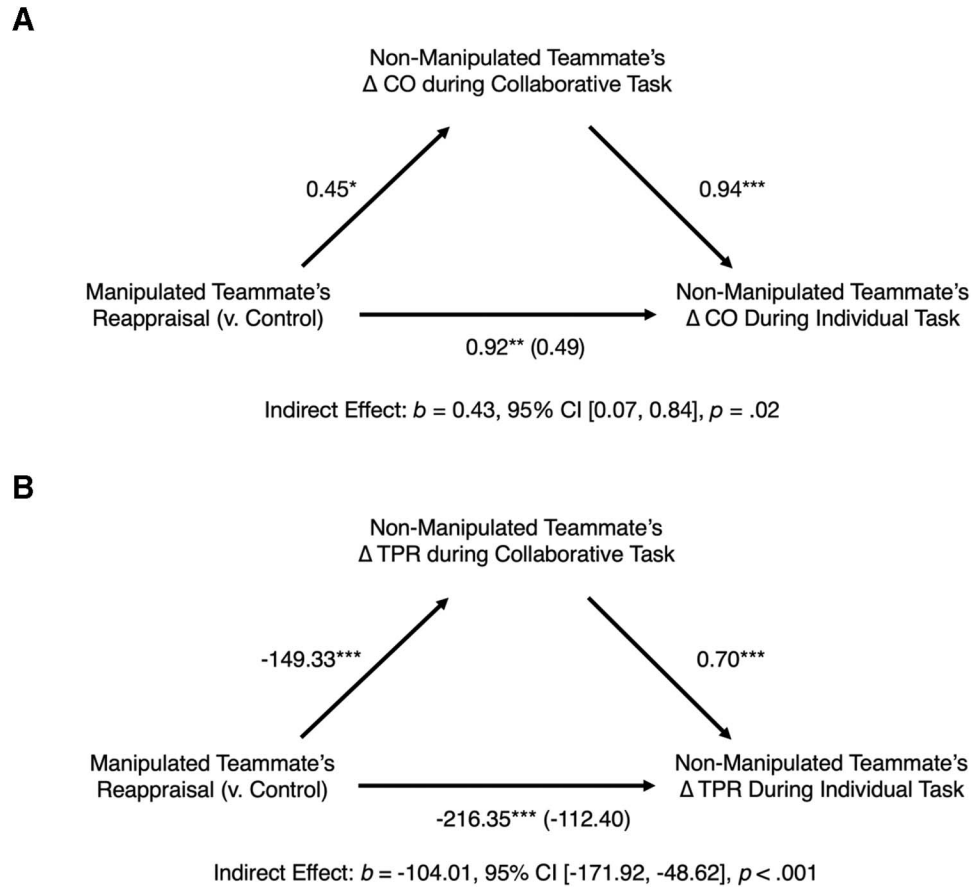


Figure 5. The influence of manipulated teammates' stress reappraisal on nonmanipulated teammates' individual task cardiac output (CO) reactivity (A) and total peripheral resistance (TPR) reactivity (B) was mediated by nonmanipulated teammates' collaborative task CO reactivity and TPR reactivity, respectively. * $p < .05$. ** $p < .01$. *** $p < .001$.

dividual task CO reactivity ($b = 0.43$, 95% CI [0.07, 0.84], $p = .022$).

Similarly, for TPR reactivity, manipulated teammates' stress reappraisal predicted nonmanipulated teammates' collaborative task TPR reactivity ($b = -149.33$, $SE = 34.30$, $p < .001$), and nonmanipulated teammates' collaborative task TPR reactivity predicted nonmanipulated teammates' individual task TPR reactivity ($b = 0.70$, $SE = 0.16$, $p < .001$). A mediation model with 20,000 bootstrap resamples indicated that nonmanipulated teammates' collaborative task TPR reactivity mediated the relationship between manipulated teammates' stress reappraisal and nonmanipulated teammates' individual task TPR reactivity ($b = -104.01$, 95% CI [-171.92, -48.62], $p < .001$).

Contagion between teammates: Tests of three interpersonal mechanisms. We tested three potential mechanisms, as detailed in the following.

Perceptions of competence: Were nonmanipulated teammates' stress responses impacted because they viewed reappraisers as more competent or warm? Emotion regulation condition did not have a significant impact on social perceptions of manipulated teammates in this research—reappraisers: competence $M = 4.23$, $SD = 0.70$; warmth $M = 4.00$, $SD = 0.78$; controls: competence

$M = 3.90$, $SD = 0.84$; warmth $M = 4.00$, $SD = 0.72$; suppressors: competence $M = 4.00$, $SD = 0.77$; warmth $M = 4.00$, $SD = 0.80$; competence $F(2, 122) = 1.87$, $p = .159$; warmth $F(2, 122) = 0.00$, $p = 1.000$; $ps > .148$ for all Tukey's post hoc comparisons between conditions. Nor were nonmanipulated teammates' competence/warmth perceptions of their manipulated teammate significantly related to nonmanipulated teammates' individual task CO²—competence: $b = 0.05$, $t(112) = -0.46$, $p = .612$; warmth: $b = -0.01$, $t(112) = -0.14$, $p = .892$ —or TPR—competence: $b = -0.04$, $t(104) = -0.40$, $p = .691$; warmth: $b = -0.01$, $t(104) = -0.12$, $p = .904$. Thus, we observed no evidence that altered competence/warmth perceptions contributed to stress response contagion in this interactional context.

² For each of the three potential interpersonal mechanisms, the measure pertained to the entire 6-min collaborative task period, extending beyond the period during which collaborative task CO and TPR were measured. Thus, it was appropriate to examine how the three potential mechanisms related to individual task CO and TPR, but not to collaborative task CO and TPR.

Sense of social connection: Were nonmanipulated teammates' stress responses impacted because they felt more connected to reappraisers? Emotion regulation condition did not have a significant impact on nonmanipulated teammates' perceptions of social connection to manipulated teammates (reappraisers: $M = 4.70$, $SD = 1.65$; controls: $M = 4.25$, $SD = 1.56$; suppressors: $M = 4.18$, $SD = 1.76$), $F(2, 122) = 1.19$, $p = .308$; $ps > .322$, for all Tukey's post hoc comparisons between conditions. Moreover, nonmanipulated teammates' sense of connection to their manipulated teammate was not related to nonmanipulated teammates' physiological responses during individual performance, $ps > .333$. Thus, we observed no evidence that social connection—assessed via the IOS—contributed to stress response contagion, but limitations of the measure used may obscure the role of social connection as a mechanism of contagion (see the *Discussion* section).

Affective displays: Were nonmanipulated teammates' stress responses impacted by reappraisers' expressions of PA and NA? We first tested whether individuals engaging in stress reappraisal impacted their nonmanipulated teammates via displaying different levels of PA or NA. No main effects of emotion regulation condition emerged for displays of PA, $F(2, 116) = 0.71$, $p = .500$, or NA, $F(2, 116) = 0.08$, $p = .924$. Tukey's post hoc comparisons showed no significant differences in affective displays by manipulated reappraisers (PA: $M = 2.36$, $SD = 0.57$; NA: $M = 1.50$, $SD = 0.33$) compared to manipulated controls (PA: $M = 2.21$, $SD = 0.50$; NA: $M = 1.54$, $SD = 0.52$) or manipulated suppressors (PA: $M = 2.26$, $SD = 0.59$; NA: $M = 1.53$, $SD = 0.32$), which did not differ from each other (all $ps > .47$). Thus, we observed no evidence that manipulated reappraisers' levels of PA or NA accounted for improved stress responses in their teammates.

Our next test of affective displays as a potential contagion mechanism focused on the hypothesis that manipulated reappraisers' affective displays, when displayed, would have a more pronounced relationship with nonmanipulated teammates' stress responses relative to affective displays by manipulated teammates in the other conditions. Consistent with this hypothesis, for PA displays, we observed a significant Condition (reappraisal vs. control) \times Manipulated Teammates' Collaborative Task PA interaction on nonmanipulated teammates' individual task CO, $F(1, 69) = 14.05$, $p < .001$, 95% CI [0.39, 1.27], $\eta_p^2 = 0.17$ (see Figure 6).³ Simple slopes analysis revealed that manipulated teammates' PA was positively related to nonmanipulated teammates' CO in the reappraisal condition, $b = 0.43$, $t(38) = 2.95$, $p = .005$. In contrast, in the control condition, manipulated teammates' PA was negatively related to nonmanipulated teammates' CO, $b = -0.40$, $t(31) = -2.46$, $p = .019$. Similarly, for NA displays, we observed a significant Condition (reappraisal vs. control) \times Manipulated Teammates' Collaborative Task NA interaction on nonmanipulated teammates' individual task TPR, $F(1, 66) = 5.93$, $p = .018$, 95% CI [0.10, 1.03], $\eta_p^2 = 0.07$. Simple slopes analysis revealed a marginally significant relationship between manipulated teammates' NA and nonmanipulated teammates' TPR in the reappraisal condition, $b = 0.32$, $t(37) = 2.00$, $p = .053$, but not in the control condition, $b = -0.22$, $t(29) = -1.28$, $p = .213$.

We observed no significant Condition (reappraisal vs. control) \times Manipulated Teammates' Collaborative Task PA interaction on nonmanipulated teammates' individual performance TPR,

$F(1, 66) = 0.25$, $p = .622$, nor did we observe a significant Condition (reappraisal vs. control) \times Manipulated Teammates' Collaborative Task NA interaction on nonmanipulated teammates' individual performance CO, $F(1, 69) = 0.37$, $p = .547$.

Discussion

Working in teams to achieve a common goal is stressful. Teammates must marshal resources to actively address task demands, communicate ideas, and coordinate decisions and behaviors. The sympathetic arousal experienced during team performance situations can manifest as approach-oriented challenge responses or avoidance-oriented threat responses. Thus, developing methods to promote challenge responses in teams has the potential to have myriad benefits. However, no research has examined whether strategies meant to optimize stress responses in one team member can benefit their teammates. Toward this end, the research presented here examined the contagious effects of emotion regulation across teammates. More specifically, we tested whether stress reappraisal—an emotion regulation strategy effective in optimizing individuals' stress responses (Jamieson et al., 2012, a finding replicated in a larger sample here)—could improve a teammate's stress responses.

Supporting hypotheses, teams in which one person was instructed to perceive stress as a coping tool (reappraisal condition) exhibited more challenge-like cardiovascular responses (higher CO, lower TPR) relative to suppression condition teams and control teams. Critically, the benefits of stress reappraisal were observed in both manipulated and nonmanipulated teammates. That is, by interacting with a person engaging in stress reappraisal, the nonmanipulated teammate exhibited more adaptive cardiovascular responses indicative of challenge. These findings have direct implications for understanding and improving stress responses in teams. In addition, demonstrating the physiological benefits of a minimal, targeted intervention in a team performance context has potentially broader implications because, relative to threat responses, challenge-type stress responses facilitate delivery of oxygenated blood to the periphery and the brain, reduce attention to negative cues (Jamieson et al., 2012), and are linked with improved decision making (Kassam et al., 2009), approach-oriented behaviors (Beltzer et al., 2014), and slower "brain aging" (Jefferson et al., 2010).

Notably, contagious effects of stress reappraisal were observed in nonmanipulated teammates during face-to-face collaborative work when they were in direct contact with the manipulated teammate, as well as during a subsequent period of individual performance. This pattern suggests lasting effects were produced during the collaborative period that continued to benefit the nonmanipulated teammate even after direct social interaction had ceased. This idea was supported by mediation analyses examining effects across time: Improvements in nonmanipulated teammates' physiological responses during collaborative work mediated condition effects on their physiological responses during individual

³ Nearly identical results were observed when comparing reappraisal to the combined control and suppression conditions. See the [online supplemental materials](#) for these analyses, as well as additional moderation analyses related to the three interpersonal mechanisms addressed in this section.

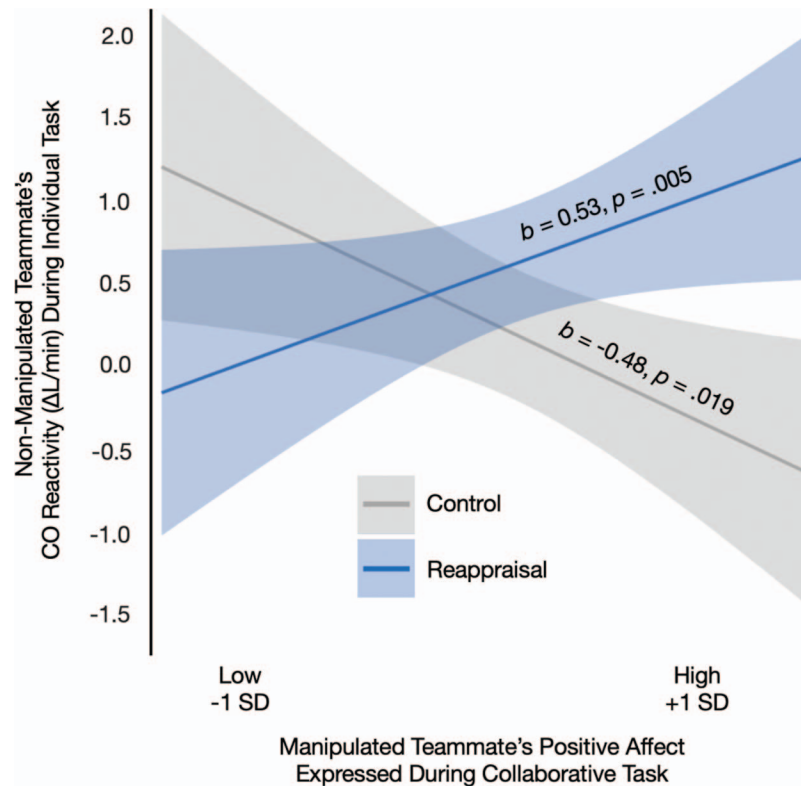


Figure 6. A significant interaction indicated that manipulated teammates' positive affect (PA) displays during the collaborative task related to nonmanipulated teammates' individual task cardiac output (CO) positively in the reappraisal condition, but negatively in the control condition. A similar interaction (not depicted in Figure 6) was observed for manipulated reappraisers' negative affect (NA) displays on nonmanipulated teammates' worsened total peripheral resistance (TPR). Error intervals represent one standard error. See the online article for the color version of this figure.

work. These findings provide evidence that regulating affective processes in one context can “snowball” to influence other contexts—and, in this instance, other people in other contexts.

Moreover, analyses of targeted interpersonal mechanisms of contagion help to illuminate how stress regulation processes and stress responses were transmitted between teammates. Notably, an interaction was observed such that manipulated reappraisers' PA displays predicted improvements in the physiological responses of their nonmanipulated teammates during individual performance, whereas PA displays from manipulated teammates in the suppression and control conditions were not associated with this effect. A similar interaction was observed for NA displays, such that reappraisers' NA displays predicted (albeit marginally) worse physiological responses in their nonmanipulated teammates during individual performance, whereas NA displays from manipulated teammates in the other conditions were not associated with this effect. These findings suggest that, at least in the context studied here, reappraisers' affective displays had a greater impact on their teammates' physiological stress responses compared to affective displays made by manipulated teammates in the other conditions. More generally, the present findings are consistent with previous work showing that a different type of affective display, behavioral tension, promoted physiological linkage in dyads (West et al., 2017).

Although additional contagion mechanisms likely operated alongside the observed affective display effects, these findings represent the first evidence for how emotion regulation processes unfolding in one person can directly impact their teammates' physiological stress responses in the moment and beyond. In addition, the identification of PA displays, specifically, as a potential mechanism of stress response contagion opens the door to future research streams investigating how the regulation of positive affect (Kalokerinos, Greenaway, Pedder, & Margetts, 2014; Mauss et al., 2011; McRae & Mauss, 2016) impacts teammates' stress responses. One possible explanation for this pattern of results is that manipulated reappraisers' affective displays were interpreted by teammates as more authentic than those of manipulated suppressors and controls. For instance, prior dyadic research has observed that suppression, but not reappraisal, predicts perceived inauthenticity (English & John, 2013; Impett et al., 2012, 2014). If reappraisers' PA and NA displays were viewed as more authentic by their teammates, this could account for their relatively greater impact on those teammates relative to the other conditions. However, because no measures of authenticity were used here, these hypotheses need to be tested in future studies.

The present findings offer theoretical contributions to emotion regulation theory (Gross, 1998). First, this research provides a deeper understanding of the interpersonal functions of emotion

regulation. The vast majority of emotion regulation research is conducted in nonsocial contexts; yet, emotions frequently occur in the presence of others (Campos, Walle, Dahl, & Main, 2011; Walle, Reschke, & Knothe, 2017). The present research conceptualized and experimentally examined the contagious consequences of emotion regulation. The most closely related construct in the extant emotion regulation literature is interpersonal modulation (Zaki & Williams, 2013), which refers to (any) actions that have the unintended consequence of modulating others' emotions (specifically). Thus far, evidence of interpersonal modulation effects is rooted in data showing that the social presence of others moderates affect (Coan, Schaefer, & Davidson, 2006). Extending existing models, the present research was the first test of emotion regulation's social consequences for another person's stress responses. This novel contribution demonstrates that reappraising one's own stress has contagious consequences for a social partner, both during a direct social interaction and during an individual performance task in which the teammates were in close proximity, but not interacting.

This research also extends Gross's (2015) conceptualization of multilevel valuation systems in which people appraise different states of the world as good or bad, generating emotion and action. The present work suggests that these valuation systems not only operate within people but can also "spill over" between people. In the terminology of the extended process model of emotion regulation (Gross, 2015), participants in the present research had first-level valuations of stressful situations (the emotion generation system), and second-level valuations of their placement in the situation (demand and resource appraisals) and their bodily responses (arousal). By manipulating one person's second-level valuations with a stress reappraisal manipulation, we observed contagion such that their teammate "caught" their positive outcomes. Thus, second-level valuation systems may not only target one's own first-level emotion generation systems, but also impact the first-level emotion generation systems of others. These findings are consistent with scholarship showing that, across the life span, individuals constantly and actively engage in social appraisal (i.e., social referencing) processes that influence their own emotional experience (Walle et al., 2017).

The present work also informs challenge and threat theory by demonstrating that not only can arousal processes be transmitted across people (Waters et al., 2014; West et al., 2017), but that challenge and threat responses can also exhibit contagion in joint performance contexts. This has important implications for theory development because it suggests the transmission of stress processes could be more nuanced than previously thought (i.e., not only sympathetic arousal, but specific challenge/threat responses). In addition, the present work offers a methodological contribution: The novel product pitch paradigm should be useful to future studies of stress responses in teams.

The present findings have implications for understanding affect contagion within dyads and groups (Barsade, 2002; Barsade & Gibson, 2012; Barsade & Knight, 2015). In fact, research specifically implicates emotion regulation as a factor influencing affect contagion (Elfenbein, 2014). The present work provides empirical support for this claim, and extends theory in the following ways: First, the contagious effects of emotion regulation are likely dependent on social context and regulatory strategy enacted. Second, emotion regulation can influence others even when the regulatory

actions are self-directed. Third, the present research generalizes theorizing to contagious effects on stress responses, rather than valenced affect or discrete forms of affect. In addition, the previous work broadens conceptualization of affect contagion by examining how stress responses can become linked as a function of one person's intrapersonal regulation. These extensions are potentially important because stressful contexts are common across broad classes of groups, and stress responses have clear health consequences.

The idea that improvements in affective responses to stress can transmit to those with whom one interacts is consistent with how other psychological interventions impact others. For instance, the effects of a self-affirmation intervention designed to facilitate minority students' academic achievement "spilled over" to impact other students in the class who were not administered the intervention (Powers et al., 2016). The greater the density of students completing the intervention exercise, the higher the grades of all classmates. This pattern suggests the possibility that stress reappraisal contagion could manifest in larger teams and signals the potential of testing the contagious effects of stress reappraisal in teams larger than two and in organizations. The present work dovetails with recent theorizing on the role of affect in altering interpersonal dynamics within groups (Algoe, Dwyer, Younge, & Oveis, in press) and enables a consideration of how stress reappraisal could facilitate contagion within online social networks (see Brady, Wills, Jost, Tucker, & Van Bavel, 2017).

Understanding how emotion regulation processes unfold between teammates has the potential to improve health and performance outcomes within organizations (see Côté, 2005; Grandey, 2000). In considering the potential contagious benefits of stress reappraisal interventions, future research should consider whom to manipulate. Humans regulate their physiology through social interaction and proximity to others, and are thus constantly attentive to other people in the social environment (Atzil, Gao, Fradkin, & Barrett, 2018; Beckes & Coan, 2011). However, certain people, namely those with higher power, tend to receive increased attention (e.g., Magee & Smith, 2013). Previous research has shown that leaders' emotional displays may exert greater contagious influence because they draw disproportionate attention from others (Sy, Côté, & Saavedra, 2005). Thus, leaders who engage in stress reappraisal may exert an even greater contagious effect on the stress responses of others at the group level.

Limitations

Limitations should be considered when interpreting the research presented here. First, it is important to note that the present results were embedded within a particular, targeted context: team and individual performance. Previous research shows that the functional value of regulation strategies is moderated by myriad contextual factors (Greenaway, Kalokerinos, & Williams, 2018; McRae, 2016; Troy, Shallcross, & Mauss, 2013), as is affect contagion. For example, in work groups that possess strong mood-regulation norms, work group members are more likely to converge in their moods because they attend more closely to one another (Bartel & Saavedra, 2000). Thus, there are likely team, work, and/or cultural contexts (Ford & Mauss, 2015; Soto, Perez, Kim, Lee, & Minnick, 2011) in which reappraisal would not positively impact self or teammate. We also note that the team-

mates in the present study were strangers, and that expectations of behavior based on previous interactions could modify the observed effects. The present work is generative in opening the door to considering how intrapersonal emotion regulation may incidentally influence social partners across contexts.

A similar point should be made regarding the preliminary evidence observed for a potential contagion mechanism, affective displays: It is possible that stress reappraisal and other stress regulation approaches could facilitate contagion via different mechanisms (or combinations of mechanisms) in different contexts. Future work investigating how outcomes of stress reappraisal transmit across teammates could be informed by research on emotion coregulation and physiological linkage in close relationships (Butler & Randall, 2013; Helm, Sbarra, & Ferrer, 2014; Waters, West, Karnilowicz, & Mendes, 2017). For instance, research from the coregulation literature indicates that threat-type stress responses can impair individuals' capacity to be responsive to their interaction partners, and this lack of responsiveness can result in threat responses in the partner (Peters, Reis, & Jamieson, 2018). In addition, although participants did not have any physical contact in the present study, research from the physiological linkage literature indicates that physical touch may help transmit stress from mothers to infants (Waters et al., 2017). Thus, future research should remain open to other potential contagion mechanisms derived from channels other than visual cues or subjective perceptions.

Even within the context of the present study, limitations should be noted about the conclusiveness of the mechanism data. First, we tested two potential pathways by which reappraisers' affective displays could serve as mechanisms of contagion, and found evidence supporting one pathway (when present, stress reappraisers' affective displays had more of a relationship with teammates' physiological stress responses) but not the other (stress reappraisal would be associated with more PA and less NA, thus facilitating contagion). Second, although reappraisers' PA predicted their nonmanipulated teammates' improved CO, no such association was observed for TPR. Similarly, although manipulated reappraisers' NA (marginally significantly) predicted their nonmanipulated teammates' worsened TPR, no such association was observed for CO. Finally, we cannot definitively conclude that the other mechanisms tested (social connection and perceived warmth/competence) but not supported did not facilitate contagion in this context. For example, social connection was assessed via a single-item measure (the IOS scale) that taps only one aspect of connection: self-other overlap. However, social connection itself is a multifaceted construct, and more fully measuring this construct may improve predictive utility. Taken together, additional research on mechanisms of stress regulation contagion is needed to better inform the interpersonal dynamics of these processes: Contagion is complex, can occur via multiple mechanisms simultaneously, and is bound and shaped by context.

We also note that we experimentally manipulated emotion regulation in the present research. In future research, it will be important to consider how similar social effects might emerge from dispositional differences in emotion regulation (e.g., Ford, Lam, John, & Mauss, 2018; Gross & John, 2003; McRae, Jacobs, et al., 2012). We speculate that—just as teaching one member of a team to engage in stress reappraisal produced team-level benefits

in this research—adding a person to a team who tends to engage in stress reappraisal may produce team-level benefits, as well.

Another limitation of the present research is that it does not lend itself to drawing conclusions about whether stress reappraisal contagiously impacts task performance. The current paradigm was not designed to index task performance: The paradigm was designed to be extremely demanding and to ensure extensive coordination during the collaborative part of the task; there was not a clear rubric for what constituted successful performance; and the sample size was determined based on expected physiological responses to Trier-like tasks, which are often larger than behavioral outcomes (e.g., Beltzer et al., 2014; Goodman, Janson, & Wolf, 2017; Hangen et al., 2019; Jamieson et al., 2012; Jamieson et al., 2013). In exploratory fashion, we coded performance on the individual performance task (see the [online supplemental material](#)); whereas the pattern of means approximated what was found in the physiological response data, no significant differences by condition were observed. Future research will be necessary to determine if stress reappraisal improves performance via contagion.

Finally, we note that the conclusions that can be drawn from suppression-control comparisons in the present paper are limited. The performance task was modeled after the Trier Social Stress Test, which is one of the most widely used and reliable evaluative threat paradigms to elicit threat-type physiological responses (e.g., Goodman et al., 2017). And, as noted above, our methodological decisions produced extremely demanding tasks. Participants assigned to the control condition were expected to exhibit threat responses, which enabled us to test the hypothesis that stress reappraisal would improve stress responses relative to a control condition and another emotion regulation condition. It is true that, relative to baseline, manipulated suppressors and their nonmanipulated teammates often displayed threat-patterned physiological stress responses (i.e., increased TPR, decreased CO). However, because controls were already threatened, suppression was unlikely to have an additive effect (and did not). Thus, any comparisons between the suppression and control conditions are limited. Future research will be necessary to examine whether intrapersonal expressive suppression produces contagion of stress responses.

Conclusion

Much evidence exists documenting the intrapersonal consequences of emotion regulation, but research considering interpersonal consequences of emotion regulation is in its nascent stage. Adding to this growing corpus of research, the data presented here suggest that one person's emotion regulation can reverberate to influence others in a team, classroom, organization, or social network. Given the prevalence of emotion regulation in everyday life (English & John, 2013), this research direction should prove fruitful.

Context of the Research

The development of the stress reappraisal regulatory approach was informed by theories from psychological and affective science, notably the BPS model of challenge and threat, extended process model of emotion regulation (Gross, 2015), and theory of constructed emotion (Barrett, 2017). In the context of these mod-

els, cognitive processes—and appraisals in particular—play prominent roles in constructing stress responses. Thus, stress responses can be regulated by modifying those upstream processes. Stress reappraisal involves highlighting the adaptive benefits of stress such that bodily signs of stress (e.g., a racing heart) are conceptualized as coping resources. This message runs counter to lay beliefs, namely that stress responses are negative states to be unilaterally avoided. By reappraising stress as functional and adaptive, appraisals of coping resources are increased, which in turn, can help improve outcomes. The “first wave” of research on stress reappraisal and related regulation approaches, such as stress mindsets (e.g., Crum et al., 2013), focused on elucidating main effects on intraindividual cognitive, physiological, behavioral, and performance outcomes. As this literature matures, we anticipate expansions into moderators that elucidate for whom and in what contexts reappraising stress is most effective (e.g., Hangen et al., 2019), unpacking interpersonal and temporal dynamics of reappraisal (the focus of this research), and boundary conditions of these types of regulatory techniques. Stress reappraisal research also interfaces with other approaches focused on optimizing stress responses, such as implicit theories and mindsets (e.g., Yeager et al., 2016, for a review see Jamieson et al., 2018).

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Call for Nominations

The Publications and Communications (P&C) Board of the American Psychological Association has opened nominations for the editorships of *Developmental Psychology*, *Journal of Consulting and Clinical Psychology*, and *Journal of Experimental Psychology: General*. Eric Dubow, PhD, Joanne Davila, PhD, and Nelson Cowan, PhD are the incumbent editors.

Candidates should be members of APA and should be available to start receiving manuscripts in early 2022 to prepare for issues published in 2023. The APA Journals program values equity, diversity, and inclusion and encourages the application of members of all groups, including women, people of color, LGBTQ psychologists, and those with disabilities, as well as candidates across all stages of their careers. Self-nominations are also encouraged.

Search chairs have been appointed as follows:

- *Developmental Psychology*, Chair: Pamela Reid, PhD
- *Journal of Consulting and Clinical Psychology*, Chair: Danny Wedding, PhD
- *Journal of Experimental Psychology: General*, Co-Chairs: Richard Petty, PhD and Michael Roberts, PhD

Nominate candidates through APA's Editor Search website (<https://editorsearch.apa.org>).

Prepared statements of one page or less in support of a nominee can also be submitted by e-mail to Jen Chase, Journal Services Associate (jchase@apa.org).

Deadline for accepting nominations is Monday, January 11, 2021, after which phase one vetting will begin.