PROCEEDINGS B

royalsocietypublishing.org/journal/rspb

Research

Cite this article: Nitschke JP, Sunahara CS, Carr EW, Winkielman P, Pruessner JC, Bartz JA. 2020 Stressed connections: cortisol levels following acute psychosocial stress disrupt affiliative mimicry in humans. *Proc. R. Soc. B* **287**: 20192941.
http://dx.doi.org/10.1098/rspb.2019.2941

Received: 18 December 2019
Accepted: 19 April 2020

Subject Category:
Neuroscience and cognition

Subject Areas:
behaviour, neuroscience, evolution

Keywords:
stress, mimicry, affiliation, emotion, cortisol, social interaction

Author for correspondence:
Jennifer A. Bartz
e-mail: jennifer.bartz@mcgill.ca

Electronic supplementary material is available online at https://doi.org/10.6084/m9.figshare.c.4955651.

THE ROYAL SOCIETY PUBLISHING

Stressed connections: cortisol levels following acute psychosocial stress disrupt affiliative mimicry in humans

Jonas P. Nitschke[1], Cecile S. Sunahara[1], Evan W. Carr[3], Piotr Winkielman[4,5], Jens C. Pruessner[2,6] and Jennifer A. Bartz[1]

[1]Department of Psychology, and [2]Faculty of Medicine, McGill Centre for Studies in Aging, McGill University, Montreal, Canada
[3]Columbia Business School, Columbia University, New York, USA
[4]Department of Psychology, University of California, San Diego, USA
[5]SWPS University of Social Sciences and Humanities, Warsaw, Poland
[6]Department of Psychology, University of Konstanz, Konstanz, Germany

JPN, 0000-0002-3244-8585; CSS, 0000-0002-1355-5377; PW, 0000-0003-2330-1802; JCP, 0000-0002-8582-2980; JAB, 0000-0001-5043-9458

Mimicry, and especially spontaneous facial mimicry, is a rudimentary element of social–emotional experience that is well-conserved across numerous species. Although such mimicry is thought to be a relatively automatic process, research indicates that contextual factors can influence mimicry, especially in humans. Here, we extend this work by investigating the effect of acute psychosocial stress on spontaneous facial mimicry. Participants performed a spontaneous facial mimicry task with facial electromyography (fEMG) at baseline and approximately one month later, following an acute psychosocial stressor (Trier Social Stress Test). Results show that the magnitude of the endocrine stress response reduced zygomaticus major reactivity, and specifically spontaneous facial mimicry for positive social stimuli (i.e. smiles). Individuals with higher levels of the stress hormone cortisol showed a more blunted fEMG response to smiles, but not to frowns. Conversely, stress had no effect on corrugator supercilii activation (i.e. frowning to frowns). These findings highlight the importance of the biological stress response system in this basic element of social–emotional experience.

1. Introduction

Spontaneous mimicry, that is, the automatic tendency of an observer to match the perceived behaviour of a target, is considered to be a rudimentary element of social–emotional experience. Although mimicry can take various forms (matching postures, gestures, mannerisms and even speech patterns), spontaneous facial mimicry is thought to be a particularly important conduit of social–emotional understanding and shared experience. As de Waal & Preston [1] note, by physically replicating the facial expressions of others in our social world, we can simulate their emotional states to better understand what they are feeling. This affect sharing, in turn, can then facilitate downstream processes like empathy and prosocial action [1,2]. In addition to facilitating social–emotional communication, mimicry (facial and other forms) has been shown to have more general effects on social cohesion, by increasing affiliation, interpersonal rapport, synchrony and liking [3,4]. Indeed, some have argued that mimicry functions as a 'social glue' that supports our fundamental need to belong [5].

Mimicry, and specifically spontaneous facial mimicry, appears to be a conserved mechanism across a variety of species [6]. Indeed, spontaneous facial mimicry has been observed in several species of great apes, including orangutans [7], chimpanzees [8] and lowland gorillas, as well as in some monkey species, including geladas [9] and macaques [10]. There is also evidence for spontaneous facial mimicry in domestic dogs [11]. Like in humans,

this mimicry appears to be a catalyst for social cohesion. For example, mimicry of the 'play face' expression in dogs communicates positive mood during rough-and-tumble play and, in this way, prolongs the play session. Similarly, in primates, mimicry during playful interactions has also been shown to extend the duration of social interactions [12,13].

To our knowledge, no study has yet investigated the effects of acute stress on spontaneous facial mimicry. However, research showing that stress can undermine emotion contagion suggests that stress may affect mimicry. Of course, mimicry is not identical to emotion contagion. Mimicry, by definition, reflects the tendency to behaviourally match others, whereas contagion reflects the tendency to experience the affective states of others, at both the psychological and physiological levels. That being said, some have argued that mimicry and emotion contagion rely on similar neural computational mechanisms [14]. Moreover, it has been argued that mimicry may facilitate emotion contagion, as the enactment of another's emotional expression may bring about the corresponding emotional experience [15,16]. As noted, recent research suggests that stress can disrupt emotion contagion [17]. Recognizing that mice (and humans) are more likely to experience emotion contagion with familiar others [18], Martin *et al*. [17] theorized that it is the stress of interacting with strangers that undermines emotion contagion. Supporting this, in a series of studies involving both mice and humans, they found that interactions with strangers (versus familiar others) were associated with higher levels of the stress hormone cortisol, and that cortisol was negatively associated with emotion contagion. They also showed that blocking cortisol synthesis with the drug metyrapone increased emotion contagion with strangers, essentially making strangers look like friends, thus highlighting the important role of stress in emotion contagion. Buruck *et al*. [19] also observed negative effects of stress on emotion contagion. They showed participants pictures of people in pain and asked them to rate the extent of visible pain. Stressed (versus non-stressed) individuals reported lower ratings of perceived pain in others. Taken together, these findings suggest that the experience of stress—and specifically the presence of the glucocorticoid cortisol—can attenuate emotion contagion (however, see [20]).

The aim of the current study was to extend this prior work to investigate the effect of acute stress on mimicry and, specifically, spontaneous facial mimicry, which, as noted, is thought to be an evolutionarily conserved mechanism supporting social emotion experience in human and some non-human animals. Given the aforementioned research on stress and emotion contagion, we predicted that acute psychosocial stress would attenuate spontaneous facial mimicry. Furthermore, we hypothesized that individuals' physiological stress response would moderate the effects of stress on mimicry: following Martin *et al*. [17], we predicted that those individuals who responded to the stressor with higher levels of cortisol would show the most pronounced reduction in facial mimicry following the TSST. Of note, we also measured salivary α-amylase (sAA; a marker of sympathetic nervous system activation); however, we did not have specific predictions about sAA given that the mimicry task was timed to occur during peak levels of cortisol, when sAA levels would be expected to have started to decline [21,22]. Finally, we also tested whether the effect of stress on mimicry depends on mimicry type (reciprocal smiling versus frowning). Research suggests that zygomaticus activation is typically more flexible and reactive in social contexts [3,23,24]; consequently, the effects of stress might be specific to smile mimicry.

2. Material and Methods

(a) Participants

Seventy-three healthy participants (23 men: mean age = 22.8, s.d. ± 3.56; 50 women: mean age = 21.8, s.d. ± 3.11; $F_{1,71} = 1.3$, $p = 0.26$), with no current history of medical or psychiatric illness were recruited from the McGill University campus. Recruitment criteria included: no recreational drug use, consuming fewer than ten alcoholic beverages a week and smoking fewer than seven cigarettes a day (factors that have been shown to influence the hypothalamic–pituitary–adrenal axis; [25]). Women were regularly menstruating and self-reported no chemical contraceptive use (see electronic supplementary material for detailed description of the procedures). All participants provided written informed consent and were compensated 10$ h^{-1}. The study was approved by the McGill University Faculty of Science Institutional Review Board.

(b) Design and procedures

We used a within-subjects design in which participants came to the laboratory on two occasions, on average four weeks apart. Of note, this research was part of a larger programme of research investigating the effects of stress on various aspects of social cognition. During the first visit (Day 1), participants completed self-report questionnaires and an empathic accuracy task ([26,27]; reported on elsewhere), and then a task using facial electromyography (fEMG) to assess spontaneous facial mimicry [24,28]. During the second visit (Day 2), we first induced acute psychosocial stress with the Trier Social Stress Test (TSST [29], see below), after which participants performed the same empathic accuracy and mimicry tasks they performed at baseline. We assessed subjective distress, cortisol and sAA throughout Day 2.

(c) Trier Social Stress Test

The TSST [29] comprises a mock job interview combined with an oral arithmetic task. Specifically, participants are instructed to identify a job they would like to interview for and are then given 10 min to prepare for the 'job interview'. Following this anticipation period, participants perform, in front of a panel of expert judges (i.e. research confederates: one male, one female): (i) a 5 min speech task in which they are instructed to describe why they are qualified for the job and, then, (ii) a 5 min oral arithmetic task in which they must count backwards from 2023 in increments of 17. The TSST has been shown to reliably induce stress across a variety of markers including cortisol, sAA and subjective experience [21,30,31]. Cortisol and sAA were collected via salivary samples (i.e. Salivette; Sarstedt AG & Co, Nümbrecht, Germany), and subjective stress was assessed using a visual analogue scale (i.e. 'how stressed do you feel?'); all measures were taken at seven time-points throughout the Day 2 session, at 10 min intervals starting 20 min before the stress induction, and following stress at +10, +25, +40 and +50 min (hereafter referred to as 'sampling time'; see figure 1 for timeline). Cortisol levels (nmol l^{-1}) were assessed using a time-resolved fluorescence immunoassay [32] and sAA (U ml^{-1}) levels were determined using the enzyme kinetic method [22].

(d) Spontaneous facial mimicry task

This task uses facial electromyography (fEMG) to assess spontaneous facial mimicry [24]. Specifically, participants were presented with 5 s video-clips of individuals going from a neutral expression to either a smile or a frown (two emotions that can

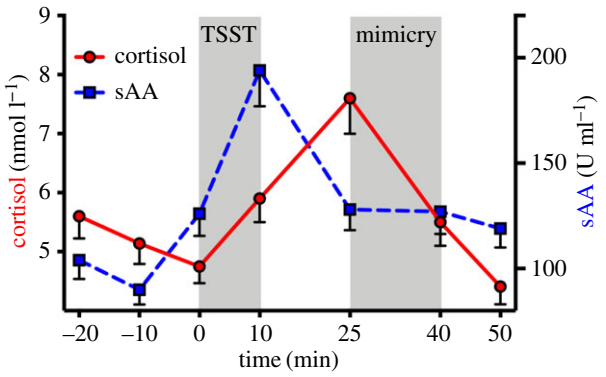

Figure 1. Timeline of the experimental procedure during Day 2 (stress induction day), including cortisol and salivary α-amylase (sAA) responses over time. Sampling times-points are indicated on the *x*-axis with ticks. Participants are introduced to the TSST 10 min before going to the interview room (at 0 min). Twenty-five minutes after the end of the TSST, participants started the facial mimicry task. Note: we did not assess cortisol or sAA measures on Day 1. (Online version in colour.)

reliably be assessed with fEMG; cf. [28]). Participants were instructed to simply observe the stimuli and to press the spacebar every time a face appeared on the monitor. A total of 80 stimuli (40 smiling, 40 frowning faces) were presented in randomized order. During the stimulus presentation, we measured activity in the zygomaticus major muscle (engaged when people smile) and corrugator supercilii (engaged when people frown) to index mimicry [33]. EMG data were obtained with bipolar electrode montage on the left side of the face [34]. Acquisition was controlled by a Biopac MP150 using Acqknowledge software (Biopac Systems). The amplified EMG signals were filtered online with a low pass of 500 Hz and a high pass of 10 Hz, sampled at a rate of 2000 Hz and then integrated and rectified using Mindware EMG software (version 2.52, MindWare Technologies, Ohio, USA).

For each stimulus (i.e. 5 s video-clip), EMG was measured for ten 500 ms windows. Prior to the stimulus presentation, a fixation cross was presented for 3 s, resulting in six pre-stimulus recordings of 500 ms. These six recordings were averaged and used as a baseline measure for each stimulus. For each participant, we *z*-transformed the fEMG data and excluded extreme (± 3 s.d.) data points. This resulted in a loss of 2751 (out of 180 480) data points (1.52% missing values). We used the 'mice' package [35] to impute the excluded data. Next, we corrected each data point according to its respective baseline measure, by subtracting the baseline measure from the value obtained during stimuli presentation (cf. [24]). These mean-rectified values were used for all subsequent analyses. Using the area under the curve (AUC) formula [36], we aggregated the 10 fEMG recordings of the zygomaticus major and corrugator supercilii in response to smiling and frowning faces. This resulted in a total of 160 AUCs for each participant: specifically, 80 zygomaticus (40 responses to smiles; 40 responses to frowns) and 80 corrugator (40 responses to smiles; 40 responses to frowns). Of note, AUCs are not measures of overall muscle activation; rather they quantify the amount of muscle activity in response to a stimulus presentation (i.e. smiles versus frowns) compared with baseline (i.e. the fixation cross) [37].

(e) Statistical analyses

We first conducted manipulation checks to confirm that the TSST elicited a stress response. Specifically, we conducted repeated-measures mixed effects models (rMEMs; [38]) on the measures of cortisol, sAA and subjective stress. Sampling time and participant gender (male = 0, female = 1), and the sampling time × gender interaction were entered as fixed factors. Sampling time was nested within participant as a random factor [39]. Raw cortisol and sAA data were log-transformed. Subsequently, we calculated AUCs for cortisol, sAA and subjective stress using the formula described by Pruessner *et al*. [36].

We then conducted rMEM analyses to test the effects of stress induction on zygomaticus major and, in a separate analysis, corrugator supercilii activity, using the AUCs for each muscle as the dependent variable (note: as we were not interested in comparing zygomatics and corrugator activation, we ran our analyses for these muscles separately, as others have done [24,40,41]; however, results testing one overarching model that includes muscle type as a third factor are comparable to those we report below; see electronic supplementary material). For each analysis, stimulus type (0 = smile; 1 = frown) and day (0 = Day 1/baseline; 1 = Day 2/TSST) were entered as fixed effects, and stimulus presentation order and gender were entered as covariates. In an additional step, we entered a higher-order term for the stimulus type × day interaction. Subject ID was entered as a random effect. Following Barr *et al*. [39,42], we included random slopes for our highest-order combination of within-subject factors subsumed by the stimulus type × day interaction to test for a maximally defined model.

In addition to looking at overall muscle reactivity during stimulus presentation, we adopted a more fine-grained approach looking at muscle activations in each of the 10 EMG recordings to investigate whether stress differentially affects mimicry during earlier versus later phases of the stimulus presentation. To this end, we ran an MEM with congruent muscle activation as the dependent variable and day and time course (i.e. 10 EMG recordings) as independent variables; as in the AUC analysis, stimulus presentation order and gender were entered as covariates. We also included a day × time course interaction term. For a maximally defined model, day × time course was nested within participant as a random factor.

After testing the effect of the stress induction on mimicry, we then probed the effect of the three stress markers (cortisol, sAA and subjective stress); here, we focused our analyses on data from Day 2 (TSST day), as we only assessed these markers on Day 2. Specifically, we ran a series of linear MEMs [43,44], one for each stress marker. Respective AUCs of muscle reactivity were entered as the dependent variable, and stimulus type (0 = smile; 1 = frown) and the stress marker of interest (AUCs) were entered as fixed effects. We also included the interaction between these two fixed effects (i.e. stimulus type × stress marker), to ascertain whether the effect of stress was specific to one kind of stimuli (e.g. smiles). Stimulus presentation order and participant gender were entered as covariates; subject ID was entered as a random effect. Of note, we initially included stimulus type as a random slope (cf. [39]); the model converged for cortisol and sAA, but not for subjective stress. To facilitate comparisons, we reverted to a simple intercept model for all stress marker analyses [45]. This approach did not change the significance of the cortisol effects reported below.

All reported confidence intervals were bootstrapped. All statistical analyses were conducted using R [46] and the lme4-package (1.1–18-1) for the rMEM and MEM analyses [47]. Significant effects from the MEM were decomposed using the formula described by Preacher *et al*. [48].

3. Results

(a) Stress manipulation check

The rMEMs revealed a significant effect of sampling time for cortisol ($F_{5,214.55} = 25.52$, $p < 0.001$), sAA ($F_{5,259.48} = 42.57$, $p < 0.001$) and subjective stress ($F_{5,342.62} = 80.86$, $p < 0.001$), thus indicating successful stress induction (figure 1). Results showed no effects for gender, or the gender × sampling time interaction for cortisol or sAA; all *p*-values greater than 0.1.

However, there was a significant gender effect for subjective stress: women reported higher subjective stress levels than men, $F_{1,70} = 5.8$, $p = 0.02$ (women: mean = 3.69, s.d. = 2.7; men: mean = 2.31, s.d. = 2.25).

(b) Effect of stress induction on zygomaticus major (Day 2 versus Day1)

The rMEMs evaluating zygomaticus major activation revealed a significant effect of stimulus type (smile versus frown), $F_{1,74.0} = 8.66$, $p < 0.01$; consistent with prior mimicry research, across days, participants smiled more to smiling faces (congruent: mean = 514.70, s.e.m. = 42.41) than to frowning faces (incongruent: mean = 207.22, s.e.m. = 40.54). Turning to the effect of stress, our key experimental question, results showed a main effect of day on zygomaticus activity, $F_{1,74.0} = 4.89$, $p = 0.03$, such that participants smiled less on the TSST day (mean = 246.33, s.e.m. = 43.61) relative to baseline (Day 1: mean = 476.35, s.e.m. = 39.26). The reported model (Akaike information criterion, AIC = 213 115) had a better fit than the intercept- only model (AIC = 213 120; $\chi^2_4 = 13.23$, $p = 0.01$). Adding the stimulus type × day interaction did not reveal a significant interaction ($F_{1,73.1} = 0.11$, $p = 0.74$). Thus, our hypotheses about the effects of stress were partially supported: stress did attenuate smiling, but this effect occurred for both smiling and frowning stimuli.

We now turn to the more fine-grained time course data looking at zygomaticus activity during each of the 10 fEMG recordings; here we focus specifically on the mimicry response—that is, zygomaticus activity in response to smiling faces (readers interested in zygomaticus activity in response to frowns are referred to electronic supplementary material, figure S1). Results from the MLM analysis showed a significant day × time course interaction on zygomaticus activity in response to smiling faces, with higher activation on Day 1, compared with Day 2, at 2000 ms ($b = -0.097$ (s.e. ± 0.040; 95% CI [−0.173, −0.018]), $t_{928.9} = 2.45$, $p = 0.014$) and 2500 ms ($b = -0.103$ (s.e. ± 0.044; 95% CI [−0.185, −0.017]), $t_{427.8} = 2.35$, $p = 0.019$). Thus, it appears that stress resulted in a delayed onset of smiling to smiling faces. (For details, see electronic supplementary material, figure S2, panel A). The reported model had a better fit (AIC = 143 671) compared with the intercept model (AIC = 143 729; $\chi^2_{21} = 100$, $p < 0.001$).

(c) Effects of stress markers on zygomaticus major (Day 2)

Given that we observed an overall effect of the stress induction, we conducted additional analyses to examine the effects of cortisol, sAA and subjective stress, respectively, on zygomaticus major activity on Day 2 (the only day we assessed the stress markers). Results from the MEM using cortisol as a predictor revealed a significant stimulus type × cortisol interaction, $b = -14.37$ (s.e. ± 5.20; 95% CI [−24.52, −4.73]), $t_{5588.62} = -2.76$, $p < 0.01$. The reported model had a better fit (AIC = 107 583) than the intercept model (AIC = 107 601; $\chi^2_5 = 27.4$, $p < 0.001$).

Post-hoc testing revealed a significant negative association between cortisol and zygomaticus activation for smiling stimuli ($z = -2.76$, $p < 0.01$), but not for frowning stimuli ($z = -0.379$, $p = 0.41$). As depicted in figure 2, as cortisol levels increased, smiling to smiling faces decreased, but cortisol increases had no effect on smiling to frowning faces. These findings indicate

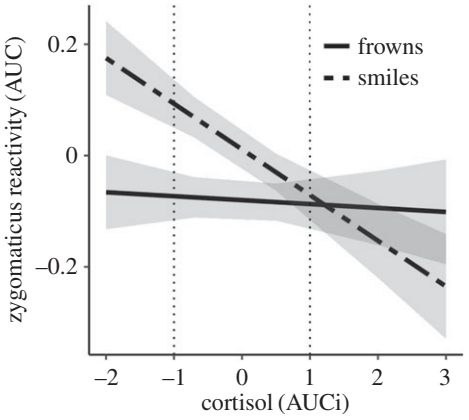

Figure 2. Results of the mixed effects model predicting zygomaticus major reactivity with stimulus type (frowning versus smiling faces) and cortisol levels (low: −1 s.d.; high: +1 s.d.). The shaded areas reflect the standard error of the mean. Results showed a significant association between cortisol and zygomaticus major activation in response to smiling faces, with higher levels of cortisol leading to a reduction in zygomaticus activity. Conversely, there was no effect of cortisol on zygomaticus activation in response to frowns. In addition, simple effect analyses showed significantly higher zygomaticus activation in response to smiles versus frowns for low-cortisol responders, whereas there was no difference in zygomaticus activation in response to smiles versus frowns for high-cortisol responders. In essence, low-cortisol responders had an intact mimicry response that was similar to the baseline/no stress testing day, whereas high-cortisol responders did not show elevated zygomaticus activation in response to smiles (versus frowns), indicating a blunted mimicry response. All data converted to *z*-scores for illustrative purposes.

that the biological stress response, as measured by cortisol, specifically attenuated mimicry, consistent with Martin *et al.* [17] and supporting our hypothesis about the effects of stress on mimicry. In further support of this, analyses showed that participants who did not show a strong cortisol response (−1 s.d.) to the TSST showed significantly higher zygomaticus activation to smiles than to frowns on the TSST day ($z = 4.30$, $p < 0.005$); this suggests that these participants had an intact mimicry response that was similar to the baseline/no stress testing day. By contrast, participants who showed a strong cortisol response (+1 s.d.) did not show higher zygomaticus activation to smiles than to frowns on the TSST day ($z = 0.393$, $p > 0.70$); again indicating that cortisol blunted mimicry (see figures 2 and 3). The MEM with the stimulus type × cortisol interaction term (AIC = 107 589) was significantly better than the non-interaction model (AIC = 107 583; $\chi^2_1 = 7.63$, $p = 0.006$).

In contrast with cortisol, results from the MEM analyses using sAA and subjective stress as predictors revealed no significant effect for either variable on zygomaticus major activation (all *p*-values > 0.05). The non-interaction models had significantly better fits than the interaction model, all *p*-values > 0.2.

(d) Effect of stress induction on corrugator supercilii (Day 2 versus Day 1)

Turning to the corrugator supercilii analyses, the rMEM revealed no effect of stimulus type or day; nor was there a significant stimulus type × day interaction (all *p*-values > 0.2): intercept model (AIC = 213 815); reported model (AIC = 213 811; $\chi^2_4 = 12.07$, $p = 0.017$).

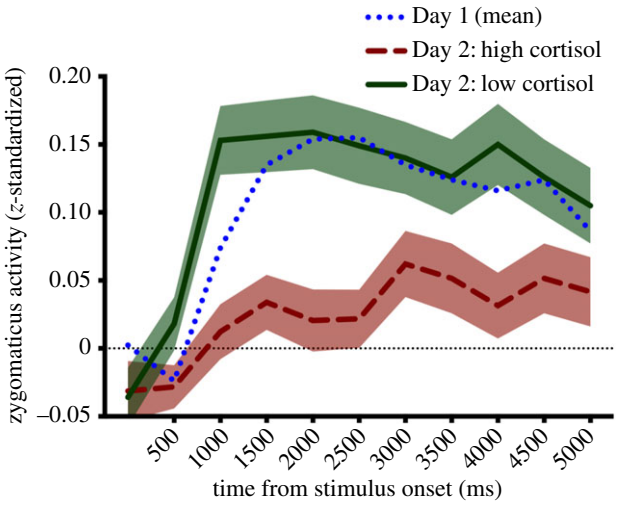

Figure 3. Affiliative mimicry (i.e. zygomaticus major activity in response to smiling faces) as a function of endocrine response. High-cortisol responders are represented by the dashed line and low-cortisol responders are represented by the solid line (median-split for illustrative purposes). Activity during Day 1 (baseline) is depicted by the dotted line for comparison purposes. As can be seen, low-cortisol responders had an intact mimicry response that was similar to the baseline/no stress day response, whereas affiliative mimicry was blunted for high-cortisol responders. (Online version in colour.)

Since none of these effects on the corrugator supercilii was significant, we did not probe for the effects of the different stress markers on frowning mimicry on Day 2.

Thus, stress did not attenuate mimicry to frowns. The lack of effects on the corrugator, however, may be because, as noted, the corrugator is less reactive than the zygomaticus and, typically, also more rare. Indeed, consistent with this hypothesis, as can be seen in electronic supplementary material, figure S2, panel B, the corrugator is less active than the zygomaticus on both days.

4. Discussion

The aim of the current study was to investigate the effects of acute psychosocial stress on spontaneous facial mimicry, a rudimentary and evolutionarily conserved element of social–emotional experience. Our results show that the experience of stress reduced zygomaticus reactivity, and specifically spontaneous facial mimicry for positive social stimuli (i.e. smiles), an effect that was driven by the endocrine stress response. Individuals with higher levels of the stress hormone cortisol showed a blunted fEMG response to smiles, whereas those with lower cortisol levels showed an intact affiliative response that was similar to their baseline response. Our findings extend prior research on stress and emotion contagion (e.g., see [17,19]) by showing that the biological stress response also attenuates spontaneous facial mimicry. These findings may shed light on the observation that humans, and some non-human animals, are less likely to mimic outgroup members [49,50]. Consistent with Martin *et al.*, it may be the stress elicited in such interactions that undermines mimicry (cf. [51]).

Of note, the effects of the endocrine stress response (i.e. cortisol) were specific to reciprocal smiling. There are a few possible explanations for this selective effect. First, the TSST, which is not a pleasant experience, may have primed participants to show negative emotions, in this way overriding the mimicry-attenuating effects of stress on frowns. This explanation, however, seems unlikely given that we did not observe greater corrugator activation on the stress day versus baseline day, nor was the corrugator more active than the zygomaticus on the stress day. A more likely explanation, we think, has to do with the greater flexibility and reactivity of the zygomaticus major muscles [23], a factor that could make them more vulnerable to the influence of stress. Relatedly, frowns are, in general, more rare than smiles [23,52], so the selective effects could be due to a general absence of frowning. In fact, some have argued that mimicry to smiles and mimicry to frowns are not equivalent and support different (albeit sometimes overlapping) goals. According to Hess & Fischer [3], smiling to a smile, or 'affiliative mimicry,' is one key way people communicate their interest because such positive feedback signals enjoyment of the activity/other conspecific, desire for continued interaction, and a strong emotional connection. Such positive mimicry is also a catalyst for social cohesion in some non-human animals [11]. By contrast, anger mimicry (i.e. responding to anger with anger) is often avoided in social situations because it can antagonize a relationship. Future research is needed to better understand the (lack) of an effect of stress on anger mimicry; for example, by examining the effects of stress in situations where anger mimicry is more likely to occur (e.g. competitive situations; [3]), one can ascertain whether stress selectively impairs affiliative mimicry, or whether it also affects anger mimicry under the right circumstances.

What might be the mechanism underlying the effect observed in the current research? It has been argued that stress can lead to social withdrawal in order for the organism to attend to its own affective state (for a review, see [53]). In this regard, there is evidence that the experience of acute stress leads to an increase in self-focus, particularly towards one's own emotional experience [54,55]. Such egocentrism could prevent affiliative mimicry by reducing emotional engagement with the stimulus. Refusing to resonate happiness (i.e. smiling to a smile) might also be a way to solicit social support when one is distressed. Our effects could also be due to reduced attention, given prior research showing that negative emotional states can sometimes undermine both exogenous (stimulus-driven, bottom-up) and endogenous (top-down) attention to emotional faces [56,57], though there are also some attention capture phenomena. Future research using eye-tracking may shed light on this mechanism.

A few aspects of our procedures are worth noting. First, we found no effect of sAA or subjective stress on affiliative mimicry. However, our study design was guided by prior research linking cortisol to reduced emotion contagion (e.g., see [17,19]), and we timed the mimicry task to occur during peak cortisol levels; by this time, sAA levels had begun to decline. Similarly, subjective stress may also have begun to decline 20 min after the TSST. That said, others have also shown a dissociation between subjective stress and the physiological stress response [30,58]. Future research should manipulate task timing to assess the role of other stress markers on facial mimicry. Second, we did not counterbalance the stress manipulation; thus, it is possible that learning or repeated exposure to the mimicry task may have attenuated participants' responses to the stimuli on Day 2. That said, it is not clear why learning or habituation would only occur for participants with higher cortisol levels. Third, we only assessed spontaneous facial mimicry to smiles and frowns (emotions that can reliably be assessed with fEMG; [28]). Of course,

there are numerous other socially relevant emotions: sadness, fear, disgust and guilt, just to name a few. Sadness or distress, in particular, is especially relevant given the significance of mimicry and emotion contagion in empathy; indeed, there is evidence of hormonal stress contagion (i.e. a rise in cortisol) when people observe another in distress [58,59]. It would be interesting to know if pre-existing stress attenuates this emotion contagion effect (cf. [17]).

In closing, the present research indicates that the stress hormone cortisol can attenuate affiliative mimicry (smiling to smiles). Given the importance of mimicry and especially affiliative mimicry to bonding, our findings suggest that stress may undermine bonding (or, perhaps, more precisely, our research sheds light on one mechanism by which stress can undermine bonding). Corroborating this idea, recent research indicates that chronically lonely individuals, who often show alterations in the physiological stress response system [60], show selective impairments in reciprocal smiling [41]. Additionally, to the extent that mimicry serves more sophisticated forms of empathy, our findings suggest that stress could undermine the quality of our social relationships and experience by attenuating emotion sharing and understanding [1,2]. Although we focused on humans in the present research, there is good reason to believe that these effects would extend to at least some non-human animals, given that the phenomenon of spontaneous facial mimicry is conserved across numerous species and the cortisol system is also well-conserved biologically.

Ethics. The study was approved by the McGill University Faculty of Science Institutional Review Board.

Data accessibility. Data available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.kh1893225 [61].

Authors' contributions. J.P.N., J.C.P. and J.A.B. developed the study concept and study design. P.W. and E.W.C. facilitated the study design and implementation of the mimicry task. J.P.N. conducted the data processing and data analyses. J.P.N. and C.S.S. oversaw and assisted with the data collection. J.P.N. drafted the manuscript with the help of J.A.B. and C.S.S.; E.W.C., P.W. and J.C.P. provided critical revisions. All authors approved the final version of the manuscript for submission.

Competing interests. The authors have no financial or other conflicts of interest to declare.

Funding. This study was funded by an NSERC Discovery grant awarded to J.A.B. (grant no. RGPIN-04241). J.P.N. holds a doctoral scholarship from Fonds de Recherche du Québec–Société et Culture (FRQSC).

References

1. de Waal FBM, Preston SD. 2017 Mammalian empathy: behavioural manifestations and neural basis. *Nat. Rev. Neurosci.* **18**, 498–509. (doi:10.1038/nrn.2017.72)
2. Prochazkova E, Kret ME. 2017 Connecting minds and sharing emotions through mimicry: a neurocognitive model of emotional contagion. *Neurosci. Biobehav. Rev.* **80**, 99–114. (doi:10.1016/j.neubiorev.2017.05.013)
3. Hess U, Fischer A. 2013 Emotional mimicry as social regulation. *Pers. Soc. Psychol. Rev.* **17**, 142–157. (doi:10.1177/1088868312472607)
4. Chartrand TL, Lakin JL. 2013 The antecedents and consequences of human behavioral mimicry. *Annu. Rev. Psychol.* **64**, 285–308. (doi:10.1146/annurev-psych-113011-143754)
5. Lakin JL, Jefferis VE, Cheng CM, Chartrand TL. 2003 The chameleon effect as social glue: evidence for the evolutionary significance of nonconscious mimicry. *J. Nonverbal Behav.* **27**, 145–162. (doi:10.1023/A:1025389814290)
6. Hecht EE, Patterson R, Barbey AK. 2012 What can other animals tell us about human social cognition? An evolutionary perspective on reflective and reflexive processing. *Front. Hum. Neurosci.* **6**, 224. (doi:10.3389/fnhum.2012.00224)
7. Ross MD, Menzler S, Zimmermann E. 2008 Rapid facial mimicry in orangutan play. *Biol. Lett.* **4**, 27–30. (doi:10.1098/rsbl.2007.0535)
8. Palagi E, Norscia I, Pressi S, Cordoni G. 2019 Facial mimicry and play: a comparative study in chimpanzees and gorillas. *Emotion* **19**, 665–681. (doi:10.1037/emo0000476)
9. Mancini G, Ferrari PF, Palagi E. 2013 In play we trust. Rapid facial mimicry predicts the duration of playful interactions in geladas. *PLoS ONE* **8**, e66481. (doi:10.1371/journal.pone.0066481)
10. Scopa C, Palagi E. 2016 Mimic me while playing! Social tolerance and rapid facial mimicry in macaques (*Macaca tonkeana* and *Macaca fuscata*). *J. Comp. Psychol.* **130**, 153–161. (doi:10.1037/com0000028)
11. Palagi E, Nicotra V, Cordoni G. 2015 Rapid mimicry and emotional contagion in domestic dogs. *R. Soc. Open Sci.* **2**, 150505. (doi:10.1098/rsos.150505)
12. Mancini G, Ferrari PF, Palagi E. 2013 Rapid facial mimicry in geladas. *Scient. Rep.* **3**, 1527. (doi:10.1038/srep01527)
13. Davila-Ross M, Allcock B, Thomas C, Bard KA. 2011 Aping expressions? Chimpanzees produce distinct laugh types when responding to laughter of others. *Emotion* **11**, 1013–1020. (doi:10.1037/a0022594)
14. Iacoboni M. 2009 Imitation, empathy, and mirror neurons. *Annu. Rev. Psychol.* **60**, 653–670. (doi:10.1146/annurev.psych.60.110707.163604)
15. Winkielman P, Coulson S, Niedenthal P. 2018 Dynamic grounding of emotion concepts. *Phil. Trans. R. Soc. B* **373**, 20170127. (doi:10.1098/rstb.2017.0127)
16. Winkielman P, Niedenthal P, Wielgosz J, Eelen J, Kavanagh LC. 2015 Embodiment of cognition and emotion. In *Attitudes and social cognition. APA handbook of personality and social psychology*, vol. 1, pp. 151–175. Washington, DC: American Psychological Association. (doi:10.1037/14341-004)
17. Martin LJ *et al.* 2015 Reducing social stress elicits emotional contagion of pain in mouse and human strangers. *Curr. Biol.* **25**, 326–332. (doi:10.1016/j.cub.2014.11.028)
18. Langford DJ, Crager SE, Shehzad Z, Smith SB, Sotocinal SG, Levenstadt JS, Chanda ML, Levitin DJ, Mogil JS. 2006 Social modulation of pain as evidence for empathy in mice. *Science* **312**, 1967–1970. (doi:10.1126/science.1128322)
19. Buruck G, Wendsche J, Melzer M, Strobel A, Dörfel D. 2014 Acute psychosocial stress and emotion regulation skills modulate empathic reactions to pain in others. *Front. Psychol.* **5**, 517. (doi:10.3389/fpsyg.2014.00517)
20. Tomova L, Majdandžic J, Hummer A, Windischberger C, Heinrichs M, Lamm C. 2017 Increased neural responses to empathy for pain might explain how acute stress increases prosociality. *Soc. Cogn. Affect. Neurosci.* **12**, 401–408. (doi:10.1093/scan/nsw146)
21. Nater UM, Rohleder N, Gaab J, Berger S, Jud A, Kirschbaum C, Ehlert U. 2005 Human salivary alpha-amylase reactivity in a psychosocial stress paradigm. *Int. J. Psychophysiol.* **55**, 333–342. (doi:10.1016/j.ijpsycho.2004.09.009)
22. Engert V, Vogel S, Efanov SI, Duchesne A, Corbo V, Ali N, Pruessner JC. 2011 Investigation into the cross-correlation of salivary cortisol and alpha-amylase responses to psychological stress. *Psychoneuroendocrinology* **36**, 1294–1302. (doi:10.1016/j.psyneuen.2011.02.018)
23. Niedenthal PM, Mermillod M, Maringer M, Hess U. 2010 The Simulation of Smiles (SIMS) model: embodied simulation and the meaning of facial expression. *Behav. Brain Sci.* **33**, 417–433, 433–480. (doi:10.1017/S0140525X 10000865)
24. Carr EW, Winkielman P, Oveis C. 2014 Transforming the mirror: power fundamentally changes facial responding to emotional expressions. *J. Exp.*

Psychol. Gen. **143**, 997–1003. (doi:10.1037/a0034972)
25. Kudielka BM, Hellhammer DH, Wüst S. 2009 Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology* **34**, 2–18. (doi:10.1016/j.psyneuen.2008.10.004)
26. Zaki J, Bolger N, Ochsner K. 2008 It takes two: the interpersonal nature of empathic accuracy. *Psychol. Sci.* **19**, 399–404. (doi:10.1111/j.1467-9280.2008.02099.x)
27. Bartz JA, Zaki J, Bolger N, Hollander E, Ludwig NN, Kolevzon A, Ochsner KN. 2010 Oxytocin selectively improves empathic accuracy. *Psychol. Sci.* **21**, 1426–1428. (doi:10.1177/0956797610383439)
28. McIntosh DN. 2006 Spontaneous facial mimicry, liking and emotional contagion. *Pol. Psychol. Bull.* **37**, 31.
29. Kirschbaum C, Pirke KM, Hellhammer DH. 1993 The 'Trier Social Stress Test'–a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology* **28**, 76–81. (doi:10.1159/000119004)
30. Ali N, Nitschke JP, Cooperman C, Pruessner JC. 2017 Suppressing the endocrine and autonomic stress systems does not impact the emotional stress experience after psychosocial stress. *Psychoneuroendocrinology* **78**, 125–130. (doi:10.1016/j.psyneuen.2017.01.015)
31. Goodman WK, Janson J, Wolf JM. 2017 Meta-analytical assessment of the effects of protocol variations on cortisol responses to the Trier Social Stress Test. *Psychoneuroendocrinology* **80**, 26–35. (doi:10.1016/j.psyneuen.2017.02.030)
32. Dressendörfer RA, Kirschbaum C, Rohde W, Stahl F, Strasburger CJ. 1992 Synthesis of a cortisol-biotin conjugate and evaluation as a tracer in an immunoassay for salivary cortisol measurement. *J. Steroid Biochem. Mol. Biol.* **43**, 683–692. (doi:10.1016/0960-0760(92)90294-S)
33. Ekman P, Friesen WV. 1982 Felt, false, and miserable smiles. *J. Nonverbal Behav.* **6**, 238–252. (doi:10.1007/BF00987191)
34. Fridlund AJ, Cacioppo JT. 1986 Guidelines for human electromyographic research. *Psychophysiology* **23**, 567–589. (doi:10.1111/j.1469-8986.1986.tb00676.x)
35. van Buuren S, Groothuis-Oudshoorn K. 2010 mice: Multivariate imputation by chained equations in R. *J. Stat. Softw.* **45**, 1–68. (doi:10.18637/jss.v045.i03)
36. Pruessner JC, Kirschbaum C, Meinlschmid G, Hellhammer DH. 2003 Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology* **28**, 916–931. (doi:10.1016/S0306-4530(02)00108-7)
37. Fekedulegn DB, Andrew ME, Burchfiel CM, Violanti JM, Hartley TA, Charles LE, Miller DB. 2007 Area under the curve and other summary indicators of repeated waking cortisol measurements. *Psychosom. Med.* **69**, 651–659. (doi:10.1097/PSY.0b013e3 1814c405c)
38. Gueorguieva R, Krystal JH. 2004 Move over ANOVA: progress in analyzing repeated-measures data and its reflection in papers published in the Archives of General Psychiatry. *Arch. Gen. Psychiatry* **61**, 310–317. (doi:10.1001/archpsyc.61.3.310)
39. Barr DJ, Levy R, Scheepers C, Tily HJ. 2013 Random effects structure for confirmatory hypothesis testing: keep it maximal. *J. Mem. Lang.* **68**, 255–278. (doi:10.1016/j.jml.2012.11.001)
40. Korb S, Malsert J, Strathearn L, Vuilleumier P, Niedenthal P. 2016 Sniff and mimic—intranasal oxytocin increases facial mimicry in a sample of men. *Horm. Behav.* **84**, 64–74. (doi:10.1016/j.yhbeh.2016.06.003)
41. Arnold A, Winkielman P. 2019 Smile (but only deliberately) though your heart is aching: loneliness is associated with impaired spontaneous smile mimicry. (doi:10.31234/osf.io/jfdgy)
42. Barr DJ. 2013 Random effects structure for testing interactions in linear mixed-effects models. *Front. Psychol.* **4**, 328. (doi:10.3389/fpsyg.2013.00328)
43. Baguley T. 2012 *Serious stats: a guide to advanced statistics for the behavioral sciences*. Houndmills, UK: Palgrave Macmillan.
44. Holmes Finch W, Bolin JE, Kelley K. 2014 *Multilevel modeling using R*. Boca Raton, FL: CRC Press.
45. Brauer M, Curtin JJ. 2018 Linear mixed-effects models and the analysis of nonindependent data: a unified framework to analyze categorical and continuous independent variables that vary within-subjects and/or within-items. *Psychol. Methods* **23**, 389–411. (doi:10.1037/met0000159)
46. R Core Team. 2018 *R: a language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing. See http://www.R-project.org/.
47. Bates D, Mächler M, Bolker B, Walker S. 2015 Fitting linear mixed-effects models using lme4. *J. Stat. Softw. Articles* **67**, 1–48. (doi:10.18637/jss.v067.i01)
48. Preacher KJ, Curran PJ, Bauer DJ. 2006 Computational tools for probing interactions in multiple linear regression, multilevel modeling, and latent curve analysis. *J. Educ. Behav. Stat.* **31**, 437–448. (doi:10.3102/10769986031004437)
49. Bourgeois P, Hess U. 2008 The impact of social context on mimicry. *Biol. Psychol.* **77**, 343–352. (doi:10.1016/j.biopsycho.2007.11.008)
50. Campbell MW, de Waal FBM. 2011 Ingroup-outgroup bias in contagious yawning by chimpanzees supports link to empathy. *PLoS ONE* **6**, e18283. (doi:10.1371/journal.pone.0018283)
51. Gray HM, Mendes WB, Denny-Brown C. 2008 An in-group advantage in detecting intergroup anxiety. *Psychol. Sci.* **19**, 1233–1237. (doi:10.1111/j.1467-9280.2008.02230.x)
52. Carr EW, Winkielman P. 2014 When mirroring is both simple and 'smart': how mimicry can be embodied, adaptive, and non-representational. *Front. Hum. Neurosci.* **8**, 505. (doi:10.3389/fnhum.2014.00505)
53. Sandi C, Haller J. 2015 Stress and the social brain: behavioural effects and neurobiological mechanisms. *Nat. Rev. Neurosci.* **16**, 290–304. (doi:10.1038/nrn3918)
54. Starcke K, Polzer C, Wolf OT, Brand M. 2011 Does stress alter everyday moral decision-making? *Psychoneuroendocrinology* **36**, 210–219. (doi:10.1016/j.psyneuen.2010.07.010)
55. Tomova L, von Dawans B, Heinrichs M, Silani G, Lamm C. 2014 Is stress affecting our ability to tune into others? Evidence for gender differences in the effects of stress on self-other distinction. *Psychoneuroendocrinology* **43**, 95–104. (doi:10.1016/j.psyneuen.2014.02.006)
56. Yiend J. 2010 The effects of emotion on attention: a review of attentional processing of emotional information. *Cogn. Emot.* **24**, 3–47. (doi:10.1080/02699930903205698)
57. Mansell W, Clark DM, Ehlers A, Chen Y-P. 1999 Social anxiety and attention away from emotional faces. *Cogn. Emot.* **13**, 673–690. (doi:10.1080/026999399379032)
58. Buchanan TW, Bagley SL, Stansfield RB, Preston SD. 2012 The empathic, physiological resonance of stress. *Soc. Neurosci.* **7**, 191–201. (doi:10.1080/17470919.2011.588723)
59. Engert V, Plessow F, Miller R, Kirschbaum C, Singer T. 2014 Cortisol increase in empathic stress is modulated by emotional closeness and observation modality. *Psychoneuroendocrinology* **45**, 192–201. (doi:10.1016/j.psyneuen.2014.04.005)
60. Hawkley LC, Cacioppo JT. 2003 Loneliness and pathways to disease. *Brain Behav. Immun.* **17**(Suppl. 1), S98–S105. (doi:10.1016/S0889-1591(02)00073-9)
61. Nitschke JP, Sunahara CS, Carr EW, Winkielman P, Pruessner JC, Bartz JA. 2020 Data from: Stressed connections: cortisol levels following acute psychosocial stress disrupt affiliative mimicry in humans. Dryad Digital Repository. (doi:10.5061/dryad.kh1893225)