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Clinical and Evoked Pain, Personality Traits, and Emotional States: Can Familial Confounding Explain the Associations?

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Abstract

Objectives—Pain is a complex phenomenon influenced by context and person-specific factors. Affective dimensions of pain involve both enduring personality traits and fleeting emotional states. We examined how personality traits and emotional states are linked with clinical and evoked pain in a twin sample.

Methods—99 female twin pairs were evaluated for clinical and evoked pain using the McGill Pain Questionnaire (MPQ) and dolorimetry, and completed the 120-item International Personality Item Pool (IPIP), the Positive and Negative Affect Scale (PANAS), and ratings of stress and mood. Using a co-twin control design we examined a) the relationship of personality traits and emotional states with clinical and evoked pain, and b) whether genetics and common environment (i.e. familial factors) may account for the associations.

Results—Neuroticism was associated with the sensory component of the MPQ; this relationship was not confounded by familial factors. None of the emotional state measures was associated with the MPQ. PANAS Negative Affect was associated with lower evoked pressure pain threshold and tolerance; these associations were confounded by familial factors. There were no associations between IPIP traits and evoked pain.

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CONFLICTS OF INTEREST
None of the authors have any conflicts of interest to declare.

Conclusions—A relationship exists between neuroticism and clinical pain that is not confounded by familial factors. There is no similar relationship between negative emotional states and clinical pain. In contrast, the relationship between negative emotional states and evoked pain is strong while the relationship with enduring personality traits is weak. The relationship between negative emotional states and evoked pain appears to be non-causal and due to familial factors.

Keywords

clinical pain; evoked pain; personality; extraversion; neuroticism; emotions; twins

INTRODUCTION

The sensation of physical pain, familiar to all human beings, is strongly influenced by a variety of corresponding and sometimes competing variables. Demographic characteristics, genetic factors (1), and personality dispositions (2) all influence pain responses and individual differences in responses to chronic pain tend to be large (1, 3). However, situational variables, temporary emotional states, and stress also play a role (4, 5). For example, acute negative emotions are often associated with increased pain sensitivity (6, 7).

Evoked pain sensitivity is defined as the acute response to potentially pain-inducing stimuli such as pressure or very cold water. Clinical pain, which can be defined as the subjective perception of spontaneous pain, is often chronic pain. Chronic clinical pain syndromes, such as fibromyalgia, are defined by the presence of both spontaneous and evoked pain, and fibromyalgia patients have been distinguished clinically from other patients with pain by finding evoked pain, that is pain evoked by 4 kg manual pressure, in at least 11 of 18 defined tender points. Fibromyalgia patients have increased sensitivity to several types of evoked pain, including pressure at non-tender-point sites (25) and heat and cold pain (26). Sensitivity to evoked pain via biological and affective mechanisms may thus be a risk factor for chronic clinical pain syndromes.

Typical models of pain recognize interactions between intensity, unpleasantness, and affective dimensions but the complex biological, psychological, and environmental factors are as yet poorly understood (8). The affective dimensions alone are complex, involving both enduring personality traits and fleeting emotional states at the time the pain is experienced. Personality traits may exert their influence by means of cognitive processes related to the ways in which individuals constitute the meanings and implications of pain (9, 10). For example, positive personality traits such as optimism, hope, and self-efficacy, are protective against enhanced pain perception, and this protective effect may be mediated by a lower tendency to pain catastrophizing in these individuals (11). Trait neuroticism—the tendency to experience strong negative emotions—also has been associated with chronic pain (12-14) and with chronic fatigue syndrome, which includes muscle and multi-joint pain in its diagnostic criteria (15-23). In long term prospective twin studies neuroticism has been linked with the likelihood of developing a range of painful physical conditions including chronic fatigue, and these studies identified familial influences suggesting genetic pathways between neuroticism and these pain experiences (22, 24).

There also is a trait component to the tendency to experience certain affective states and those affective states can, in turn, affect pain sensitivity (6, 7). For example, the personality trait of neuroticism is correlated with both trait and state anxiety (25) as well as a number of clinical pain types in a variety of settings (9, 22, 26-29). A profitable way of exploring pain's multiple manifestations, therefore, is to examine both evoked and clinical forms of pain and their associations with affective traits and states. This, however, can be complicated because personality traits, the tendency to experience certain emotional states, and pain responding all have heritable components (i.e. variability in those traits is partially due to genetics) and all may be influenced by developmental experiences (e.g. parental modelling of pain responding during childhood). Twin studies of clinical pain syndromes (30-32) and of experimental pain sensitivity (33-36) have shown that genetics play a moderate to large role in both clinical and evoked pain. There is also evidence that chronic pain symptoms and symptoms of anxiety and depression are mediated by shared genetic factors (37).

Twins are ideal for studying complex phenotypes, such as pain responding, for which appropriate comparison groups are often ill-defined. Monozygotic (MZ) twins who are reared together share 100% of their genes and 100% of their common developmental environment, and dizygotic (DZ) twins share an average of 50% of their genes and 100% of their common developmental environment. Because of this, co-twin control studies can examine whether shared genes and common developmental environment—referred to jointly as “familial factors” in this paper—account for the relationship between two traits. Thus, co-twin studies can help to distinguish between potentially causal relationships among clinical phenotypes, or alternatively, between shared genetic or environmental linkages among phenotypes (39). In cases such as the present study, where randomization to conditions is impossible, co-twin studies provide the best available alternative to understanding the direct, potentially causal, links among variables of interest.

The aim of this study was to examine how both personality traits and emotional states are linked with clinical and evoked pain, and to accomplish this in a twin sample that allowed us to examine confounding by familial factors. We hypothesized that personality traits would show a stronger association with clinical pain than evoked pain; and that conversely, fleeting emotional states would show a stronger association with evoked pain than clinical pain. Based on prior research on personality and chronic fatigue (22) we hypothesized that any association between personality traits and clinical pain would be at least partially confounded by shared familial factors common to both personality traits and clinical pain. We had no a priori hypothesis regarding familial mediation of emotional state and evoked pain.

METHODS

Participants

All twin pairs in this study (n = 99 pairs) were female-female members of the University of Washington Twin Registry (UWTR), a community-based registry of twins identified through the Washington State Department of Licensing. The general characteristics of the UWTR have been reported elsewhere (40, 41). Pairs were recruited between 2006 and 2010 to participate in a twin study of risk factors for chronic pain, investigating the psychological,

behavioral, and physiological risk factors for and correlates of medically unexplained pain in women. Women were targeted because of the greater incidence and burden of medically unexplained pain in women compared to men. Pairs were either discordant for medically unexplained pain or were both pain free (i.e. healthy control pairs). As reported previously (42, 43), in order to participate, both twins were required to be free from severe or chronic medical conditions that could potentially be the source of the unexplained pain (e.g. autoimmune disorders, cancer, etc.), or other medical conditions that could interfere with the study measurements, including cardiopulmonary disorders, uncontrolled endocrine conditions, and uncontrolled allergic or sleep disorders. Potential participants also were excluded if they were current smokers, tested positive for drugs of abuse during the study visit, had body mass index of < 20 or > 30 kg/m^2, were pregnant or planning to become pregnant during the course of the study, had an amputated limb, or were completely blind or deaf.

Potential participants who were either pain-free or had pain not related to a known disease or organic etiology and did not meet the above exclusionary criteria were eligible to participate. Participants were asked to discontinue all medications that affect sleep, hypothalamic-pituitary-adrenal axis function, or autonomic nervous system functioning for two weeks prior to and during the study evaluation. In addition, all pain medications were discontinued for the duration of the study. The study protocol allowed participants to take short-acting over-the-counter pain relievers (e.g., Tylenol) as needed. However, none of the participants needed to take pain relievers during the 2-day laboratory visit. Alcohol (2 drinks per week) and caffeine (1 cup of coffee per day) were restricted for the duration of the study.

Assignment of Zygosity

We employed a commonly-used self-report measure to assign zygosity. This measure contains multiple questions about childhood similarity in twin pairs. Twin pairs whose responses were concordant for the question, “As children, were you and your twin as alike as two peas in a pod, or of only ordinary family resemblance?” were classified as either MZ (both twins agreed on “alike as two peas in a pod”) or DZ (both twins agreed on “ordinary family resemblance”). In cases where responses were discordant, zygosity was assigned by using an algorithm based on several additional questions assessing childhood similarity. The use of this method to assign zygosity has been demonstrated to be 95%-98% accurate compared to biological indicators (44, 45). Pairs with indeterminate zygosity were excluded from the analyses.

General Procedures

Once enrolled, twin pairs completed a 7-day at-home protocol, kept a daily diary of sleep, stress, mood, and pain levels, and completed a comprehensive battery of questionnaires which included the measures of personality and clinical pain. Immediately after the at-home protocol, the twin pairs presented for a 2-day visit at the University of Washington General Clinical Research Center, where they participated in a number of study procedures including the evoked pain testing that is presented here. Twins came to the Research Center together but were evaluated separately at each step. Written informed consent was obtained from all

participants and the study was approved by the Institutional Review Boards at the University of Washington and University of California, San Diego.

Measures

Personality Traits—Personality traits were measured using the IPIP-NEO short form (46) (available at http://www.personal.psu.edu/faculty/j/5/j5j/IPIP/; see also http://ipip.ori.org/). The IPIP-NEO short form is a public-domain 120-item version of the IPIP-NEO that was developed to represent the commercially-available NEO PI-R (47), a widely used and well-validated measure of five factor personality traits. (For a review of reliability and validity estimates of IPIP scales see http://ipip.ori.org/newBroadbandText.htm. The five personality domains of extraversion, neuroticism (emotional instability), agreeableness, conscientiousness, and openness to experience were assessed. (For a review of these domains and their hierarchically structured sub-facets see Digman, 1990 (48).)

Emotional State Measures—A visual analog scale (VAS) rating of current stress and mood was administered each day during the at-home protocol. For this study, we used the VAS ratings completed on the same day as the Short Form McGill Pain Questionnaire (MPQ) (49) which was our measure of clinical pain. The Positive and Negative Affect Scale (PANAS) (50) was administered on the same day as, and in a sequence prior to, the dolorimetry test which was our measure of evoked pain. The PANAS is a well-validated measure of mood states and demonstrated good reliability in the present sample (positive affect $\alpha = .91$; negative affect $\alpha = .87$). The instructions for the PANAS were focused on the way each person was feeling "right now, that is, at the present moment." The VAS and PANAS measures were added after the study began and 12 pairs do not have data.

Clinical Pain—Clinical pain was measured using the Short Form McGill Pain Questionnaire (MPQ) (49). The Questionnaire is a widely used instrument designed to assess multiple dimensions of the pain experience (51). Higher scores indicate greater sensory or affective components to pain severity. This instrument has shown good reliability and validity for a wide variety of acute and chronic pain populations (52). The MPQ was completed approximately one week prior to tests of evoked pain and asked about pain over the past four weeks. In order to identify twins with existing chronic/persistent pain, participants also completed the London Fibromyalgia Study Screening Questionnaire (LFSSQ), a validated screening tool that assesses for continuous pain in muscles, bones, or joints, above and below the waist, and in the head, neck, spine, or back in the past 3 months (53).

Evoked Pain—Evoked pain was measured as pressure pain threshold and tolerance using dolorimetry. A dolorimeter is a calibrated pressure algometer with a 3.14 cm^2 footplate which reads the amount of pressure exerted on an object in kilograms. Using the same sites as assessed for the fibromyalgia manual tender point examination, trained research personnel used the dolorimeter to apply pressure at a rate of 1 kg/second up to 12 kg. Both pain threshold, defined as the point at which the stimulus is experienced as painful, and pain tolerance, the point at which the twin can no longer endure the stimulus, were recorded. For

data analysis, we used the average pressure across all tender points for both threshold and tolerance.

Statistical Analyses

Descriptive statistics were computed as means and standard deviations for continuous measures and percentages for categorical measures. We also calculated basic correlation coefficients to show the associations among the clinical and evoked measures of pain. Prior to conducting regression analyses, bivariate associations of each personality and emotional state measure with each of the pain indices were estimated by using generalized estimating equations (GEE) analyses which correct for interdependency within twin pairs. Only those trait and state measures that were associated with the pain indices at a probability level of ≤ 0.15 (corrected for interdependency among observations) were included in the subsequent linear regression models (54). We then used linear regression to estimate the associations of those personality and emotional state indices that were identified through bivariate associations, with clinical and evoked pain. The regression models were fit using GEE with robust variance estimation to control for the correlation within twin pairs (55). In total, four separate regression models were estimated using each of the four pain measures as outcomes. To estimate the most parsimonious models, a sequential backward elimination approach using a more liberal 0.15 probability level was used to eliminate personality and emotional state predictors not contributing significantly to the prediction equations for these models.

Our analytic strategy was to first estimate the overall association between personality traits and emotional state with each pain outcome in all twins as described above, to determine if a phenotypic association existed. Then, we used GEE regression to estimate the within-twin pair effects of personality and emotional state on pain. These within-pair analyses adjust for familial influences by estimating the extent to which differences in personality or emotional states within twin pairs predict differences in pain outcomes. Only those predictors that remained in the overall models were included in the subsequent within-pair analyses. Because twin pairs share a similar family environment and a portion of their genes (100% in monozygotic and on average 50% in dizygotic pairs), within-twin pair estimates are adjusted for familial and some genetic influences. If within-twin pair associations are attenuated and rendered non-significant compared to the overall effect obtained in our initial regression analyses, we can conclude these associations were confounded by familial factors (i.e. familial factors contribute to these associations). Alternately, a within-twin pair association that remains robust compared to the overall effect provides evidence that familial factors do not play a prime role in the association between personality and pain, and instead a causal effect of exposure on the outcome is possible given that other conditions of causation are met (39). Models were adjusted for age and chronic pain status as measured by the LFSSQ. Significance level was set at $p < 0.05$. Data were analyzed using Predictive Analytics Software (PASW v. 18.0, SPSS Inc.).

RESULTS

Twin Participants

Of the 198 twins who participated in the study, 4 participants (2%) were excluded due to an indeterminate zygosity. The participants were all female, and 75% were identified as monozygotic. The average age of the sample was 29 years (*SD* = 10); 89% identified themselves as White and 92% had some college education or greater. In terms of pairwise concordance on the LFSSQ, 52 pairs were concordant for no pain, 11 had one member with no pain and one member with only transient pain (i.e. lasting for less than 1 week during the prior three months), 22 had one member with no pain and one member with persistent pain (i.e. lasting for more than one week), and the remaining 14 pairs had members with widespread persistent pain.

Bivariate Associations

Table 1 displays the bivariate correlations between all of the clinical (MPQ) and evoked pain (dolorimetry) measures. As expected, clinical and evoked pain measures were significantly correlated (*r*'s ranging from an absolute value of 0.15 - 0.84). Greater sensory and affective clinical pain ratings were significantly associated with lower evoked pressure pain threshold and tolerance.

Table 2 provides the bivariate associations of personality trait and emotional state measures with clinical and evoked pain measures. Neuroticism was the personality trait most strongly correlated with sensory and affective clinical pain indices, with higher neuroticism related to greater sensory and affective clinical pain. As expected, neither of the positive and negative PANAS sub-scales was significantly correlated with either of the clinical pain measures because the PANAS data were not collected on the same day as the MPQ.

However, the VAS stress and mood ratings, which were collected on the same day as the MPQ, were significantly correlated with the clinical pain indices, with higher VAS ratings related to greater sensory and affective clinical pain. There were no significant associations between the personality traits and evoked pressure pain threshold, but the personality traits of agreeableness and conscientiousness were either significant or met our 0.15 probability cut-off to be included in the overall multivariate models for evoked pain tolerance. In addition, the PANAS negative affect sub-scale was strongly negatively correlated with both evoked pain measures.

Adjusted Associations

Clinical Pain—The overall multivariate regression analyses for both clinical pain indices included neuroticism, and VAS stress and mood ratings as predictors. The top portion of Table 3 displays the overall and within-twin pair regression estimates for both indices of clinical pain. After adjusting for age and chronic pain status in the overall model, only neuroticism remained as a significant predictor for sensory pain. After adjusting for familial factors in the within-twin pair analyses, the association between neuroticism and sensory pain ratings remained significant and the magnitude of the association was the same as in the overall association. The regression estimates for the associations of neuroticism and VAS

stress ratings with affective pain ratings were not significant in the overall model and were not significant in the within-twin pair analyses.

Evoked Pain—The overall multivariate regression analyses for evoked pain threshold included the PANAS negative affect sub-scale as a predictor; PANAS negative affect, agreeableness, and conscientiousness were predictors in the model for evoked pain tolerance. The bottom portion of Table 3 displays the overall and within-twin pair regression estimates for both indices of evoked pain. After adjusting for age and chronic pain status in the overall models, only PANAS negative affect remained as a significant predictor for both evoked pain threshold and tolerance. Unlike the clinical pain measures, the relationships between negative affect and both evoked pain measures were attenuated and rendered non-significant after adjusting for familial factors in the within-twin pair analyses.

DISCUSSION

To our knowledge, this is the first study to examine the differential relationship of personality and emotional states with clinical and evoked pain in the same sample. We found that while measures of clinical and evoked pain were related to each other in the expected directions, the pattern of associations with personality trait and emotional state differed between clinical and evoked pain. In multivariate analyses, we found a significant positive association of neuroticism with the sensory component but not the affective component of pain. In contrast, negative emotional state as measured by the PANAS was negatively associated with both evoked pressure pain threshold and tolerance. Further, the link between the trait of neuroticism and sensory pain was not confounded by familial factors, whereas the associations between negative affect and evoked pain threshold and tolerance were confounded by familial factors, indicating that the links between emotional state and evoked pain may be due to the shared familial and genetic mechanisms common to both negative affect and evoked pain.

In their broadest outline, our results suggest that the relationship between the enduring *trait* of neuroticism and clinical pain is stronger than the relationship between transient *states* of negative affect (stress and mood) and clinical pain; and that the relationship of the trait of neuroticism to clinical pain is not due to the influences of familial factors including shared genetics and common environment. That is, our results suggest that the more emotionally unstable an individual is temperamentally, the more likely that individual is to complain of pain and that this relationship between temperament and pain is robust and relatively uninfluenced by transient mood and stress states. These results provide evidence that neuroticism as a temperamental trait is involved in the expression of clinical pain. In contrast, for evoked pain, the relationship with negative emotional states is strong while the relationship with enduring personality traits is weak or nonexistent. Further, we found that the association between emotional states and evoked pain appears to be confounded by familial factors. Therefore, negative emotional states and evoked pain may share a common familial origin. Because this was observed in a model controlling for neuroticism, the trait aspects of neuroticism do not appear to be involved in this potential mediation by familial factors.

It is possible that the potentially shared genetic factors that underlie the negative emotional states and lower evoked pain threshold and tolerance are a product of underlying neural circuitry, perhaps a broader “neural alarm system” involved in responding to noxious stimuli or threats (56). The anterior cingulate cortex has long been implicated in regulating the felt unpleasantness and distressing affective components of physical pain. Individuals who are dispositionally pain-sensitive show more anterior cingulate cortex activity and report greater levels of perceived unpleasantness to painful stimulation (56-58). Patients who have undergone cingulotomy for intractable pain have reported that they are able to still feel the pain but that it no longer distresses them (56). Regions involved in pain response networks include the dorsal subdivision of the anterior cingulate cortex and its links to the insula, somatosensory cortex, thalamus, and periaqueductal gray area (58). There is mounting evidence from the animal lesion and human neuroimaging studies that both physical and social pain overlap in their underlying neural circuitry and computational processes, and that the anterior cingulate cortex plays a key role in this overlap (56). It may be that shared genetic factors underlying evoked pain sensitivity and emotional state intensity influence the feedback loops within this neural alarm and response system.

Neural circuits are obviously influenced by genetics but social learning through the common family environment may also play a role in the development of neural alarms and responses. Several studies support that observational learning processes play an important role in acquisition of pain-related information and response trajectories (59, 60). Thus individuals appear to gain input from their environment regarding the potential danger of specific pain stimuli and appropriate coping responses. Additionally, the impact of social modeling is supported by investigations of parents and children as well as healthy college students (60-62).

Our differing results for clinical versus evoked pain with respect to the influence of personality traits and affective states have several clinical implications. The finding of a positive and potentially direct association between the trait of neuroticism and the sensory component of clinical pain—that is, an association that is not influenced by familial factors—provides further evidence for the hypothesis that clinical pain and its associated syndromes in general, whatever their precise etiology from case to case, are inherently multifactorial in the way they arise and are sustained, and involve important psychological trait factors in addition to physiological factors. This hypothesis can be extended to specific syndromes involving pain complaints such as chronic widespread pain or fibromyalgia. The case for the multifactorial nature of these syndromes, and the necessity of employing both psychosocial and neurobiological explanations to understand them, has been made by Wessely and others (63-67). Interventions that can address enduring personality trait and behavioral difficulties, such as cognitive behavioral therapy and graded exercise therapy may be of significant benefit in managing chronic pain syndromes (68-70). Alternately, the weak relationship we found between enduring traits and evoked pain and the strong relationship between transient negative emotional states and evoked pain suggest that a patient's personality characteristics might contribute less to the variation in how patients will experience pain stimuli in a medical setting, for example when undergoing a painful procedure. Stress reduction and interventions to improve mood may be of considerable benefit in managing pain in those settings.

This study has several limitations. First, the sample was all female, predominantly White, and well educated. Although this matches the demographics of both the Puget Sound region and people who present to physicians with chronic pain complaints, the generalizability of the findings is limited and additional research should be conducted on males and more heterogeneous samples. Second, the sample consisted primarily of individuals who reported minimal long-lasting chronic pain. Therefore, these findings should be replicated in samples of patients with substantial amounts of clinical pain. Third, the evoked pain assessment focused on pressure pain which may not be representative of other pain modalities. Additionally, our measure of evoked pain is commonly employed to diagnose clinical pain in fibromyalgia and does not have established validity as a measure of acute pain. Future research should further validate our findings and use a variety of evoked pain protocols to examine the potential differential association of personality traits and mood states with clinical and evoked pain. Fourth, because of the limited sample of DZ pairs, we were not able to separately examine the confounding effects of shared genetics versus common environmental influences. Finally, despite the power of twin studies to disentangle familial confounding in bivariate associations, a longitudinal study—particularly one in which personality and evoked pain are measured in a large inception cohort prior to onset of chronic pain—could provide additional evidence regarding personality traits, clinical pain, and evoked pain sensitivity.

In conclusion, we found that the trait of neuroticism, but not negative emotional state, was associated with clinical pain. This association was not influenced by familial factors, suggesting that high neuroticism may be directly linked to clinical pain as a risk factor. In contrast, negative emotional state but not the trait of neuroticism, was associated with evoked pain indices. These associations were confounded by shared familial mechanisms suggesting shared genetic and/or common environmental vulnerabilities to both negative emotions and evoked pain. This study highlights the need to investigate cognitive or coping styles as potential risk factors for increased clinical pain and the potential for strategies to change cognitive and emotional coping that reduce the impact on clinical pain. The link between emotional states and evoked pain especially at the level of neural circuits also warrants further investigation.

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HIGHLIGHTS

- We examined how personality traits and emotional states link with clinical and evoked pain in twins
- Personality traits were associated with clinical pain controlling for familial factors
- Emotional states were not associated with clinical pain
- Emotional states were associated with evoked pain; this was confounded by familial factors
- Personality traits were not associated with evoked pain

Table 1

Correlations for relationships between clinical and evoked pain indices (n = 192 - 194).

	McGill Sensory	McGill Affective	Dolorimetry Threshold
McGill Affective	0.56***		
Dolorimetry Threshold	−0.20**	−0.19**	
Dolorimetry Tolerance	−0.15*	−0.15*	0.84***

Note. n = number of twins.

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$.

Table 2

Bivariate associations between personality trait or emotional state measures and pain indices.

	IPIP-NEO Personality Trait					Emotional State			
						PANAS		VAS	
Pain Indices	**N**	**E**	**O**	**A**	**C**	**Positive**	**Negative**	**Stress**	**Mood**
Clinical Pain									
McGill Sensory	0.23* (0.005)	0.04 (0.59)	0.002 (0.98)	−0.06 (0.42)	−0.01 (0.92)	−0.02 (0.86)	0.02 (0.70)	0.19* (0.04)	0.20* (0.03)
McGill Affective	0.28* (0.01)	−0.12 (0.06)	−0.07 (0.18)	−0.16* (0.05)	−0.13* (0.09)	0.02 (0.73)	−0.03 (0.61)	0.27* (0.02)	0.23* (0.04)
Evoked Pain									
Dolorimetry Threshold	−0.04 (0.77)	0.05 (0.56)	−0.06 (0.40)	0.06 (0.34)	0.10 (0.16)	−0.03 (0.68)	−0.14* (0.07)	*NA*	*NA*
Dolorimetry Tolerance	−0.09 (0.39)	0.08 (0.33)	0.03 (0.71)	0.17* (0.01)	0.12* (0.14)	0.04 (0.59)	−0.64* (0.002)	*NA*	*NA*

Note. N = neuroticism, E = extraversion, O = openness, A = agreeableness, C = conscientiousness, Positive = PANAS positive affect, Negative = PANAS negative affect, Stress = VAS stress rating at time of McGill completion, Mood = VAS mood rating at time of McGill completion, NA = not applicable. All *p* values in parentheses are corrected for interdependency among observations using GEE analyses.

*All bivariate relationships with a *p* value ≤ 0.15 were included in subsequent adjusted overall individual-level GEE analyses.

Table 3

Adjusted associations of personality and emotional state with clinical and evoked pain.

		Overall			Within-Pair*	
Outcome	**Predictor**	***n***	**B**	***p***	**B**	***p***
Clinical Pain						
McGill Sensory	**N**	192	0.048	0.002	0.047	0.04
	Stress			*NS*	--	--
	Mood			*NS*	--	--
McGill Affective	**N**	174	0.01	0.09	0.02	0.07
	Stress		0.01	0.06	0.01	0.22
	Mood			*NS*	--	--
Evoked Pain						
Threshold	**Negative**	174	−0.03	0.02	−0.003	0.89
Tolerance	**Negative**	174	−0.05	0.003	−0.03	0.16
	A			*NS*	--	--
	C			*NS*	--	--

Note.

N = neuroticism, Stress = VAS stress rating at time of McGill completion, Mood = VAS mood rating at time of McGill completion, Negative = PANAS negative affect, A = agreeableness, C = conscientiousness. All analyses were adjusted for age, zygosity, and pain status. Sample size = 174 when the VAS or PANAS measures were included in the models.

*Adjusted for familial contributions.