Perceived Injustice and Anger in Fibromyalgia With and Without Comorbid Mental Health Conditions

A Hebrew Validation of the Injustice Experience Questionnaire

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Objectives: Perceived injustice (PI), assessed by the Injustice Experience Questionnaire (IEQ), is an important trigger of anger. Both PI and anger are associated with adverse chronic pain outcomes, and with comorbid mental health severity. We aimed examined the roles of PI and anger in mediating pain across Fibromyalgia patients, with and without comorbid anxiety/depression (FM+A/D, FM-A/D, respectively), as well as rheumatoid arthritis (RA), and pain-free controls (PFC). We hypothesized the highest levels of PI, anger, and pain in FM+A/D patients, followed by FM-A/D, RA, and PFC, thus also validating a Hebrew version of the IEQ.

Methods: We translated the IEQ using the forward-backward method and collected data online. Based on self-reported anxiety/depression, the sample comprised 66 FM+A/D patients, 64 FM-A/D, 34 RA, and 32 PFCs. Assessments included the IEQ, state and trait anger, pain intensity, anxiety, depression, and pain catastrophizing. The structure and reliability of the Hebrew IEQ were examined using factor analysis and Cronbach alpha. Bootstrapped-based modeling was used to test the roles of state and trait anger in mediating and moderating the relationship between PI and pain intensity.

Results: We confirmed a one-factor structure of the IEQ, with excellent reliability. FM+A/D patients demonstrated the highest scores in all measures. Within this group, trait anger moderated the mediating effect of state anger in the relationship between PI and pain intensity.

Discussion: Our findings validate a Hebrew IEQ and highlight the importance of PI and state and trait anger in the differential manifestation of mental health comorbidity in FM.

Key Words: injustice experience questionnaire, negative affect, fibromyalgia, chronic pain, mental health

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People with chronic pain face intractable and continuous emotional distress, leading to reductions in day-to-day functioning in all spheres of life, characterized by losses of independence, financial security, social support, and more.[1–3] As a result, a strong sense of injustice and loss can emerge, further compounded by the stigma and misunderstanding associated with chronic pain.[4–6] Perceived injustice (PI) was conceptualized to capture such appraisals of unfair and irreparable suffering, and the Injustice Experience Questionnaire (IEQ) was developed to measure this tendency through elements of blame, loss, and unfairness.[7] Individual differences in IEQ scores were shown to have substantial associations with adverse chronic pain outcomes, including pain intensity, disability, mental health, and quality of life.[8–10] This was demonstrated cross-sectionally[7–13] and longitudinally,[14–18] with the unique role of PI contributing above and beyond other maladaptive cognitive factors, such as pain catastrophizing.[7,12,13,15,19] Although most current evidence is based on cross-sectional studies in which self-reported PI correlates strongly with pain-related outcomes, PI has been shown to mediate the relationship between pain intensity at baseline and quality of life 3 months later,[15] providing some prognostic evidence that PI may predict worse pain-related outcomes.

Anger is a salient experience in the lives of individuals with chronic pain, with a strong theoretical and empirical relationship to PI. Anger is a complex construct associated with strong negative feelings, increased physiological arousal, an approach-oriented motivational stance, and expressive motor reactions.[20,21] Although certain aspects of anger can be considered adaptive—improving communication, detecting offense, and overcoming challenges—anger is a primary precursor for aggression and violence and can have a detrimental impact on one's health and well-being. Indeed, how people experience, express, and regulate their anger can interact with both acute and chronic manifestations of pain.[2,22–29] For example, people with chronic pain demonstrate higher levels of self-reported anger compared with healthy controls, as well as strong associations of anger with chronic pain severity and negative pain-related outcomes.[9,28,30–33] Appraisals of injustice are an important trigger of anger, with psychological studies demonstrating unfair monetary offers to be sufficient in inducing anger.[34] This anger incorporates a sense of unfairness and blame that one has been wronged,[21,35] which may further predispose and perpetuate pain.[24,29,35] In fact, anger was shown to mediate the relationship between PI and pain-related outcomes.[11,15,36–40] The majority of findings were primarily based on measuring state anger, the current experience of

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angry feelings.[32,41] Trait anger, on the other hand—the general proneness to feel angry—was only mildly associated with pain and has thus been suggested as a potential moderator of the relationship between state anger and pain.[22]

The relationship between PI and adverse pain-related outcomes has been shown in a wide range of chronic pain conditions, including fibromyalgia (FM).[7,37,42–45] FM is a common chronic pain condition characterized by neurophysiological abnormalities in peripheral and central mechanisms.[46–48] FM is frequently compared with rheumatoid arthritis (RA), especially due to shared symptomatology of persistent and widespread musculoskeletal pain.[42,49,50] Although RA is clearly recognizable by defined clinical presentation and laboratory tests, the multifaceted nature of FM makes it diagnosable only by extensive clinical evaluation, usually following comprehensive attempts to identify a specific diagnosis for the pain condition first.[47,48] As such, FM is often characterized by higher levels of pain, fatigue, emotional distress (including anger), PI, and maladaptive coping strategies compared with RA.[26,42,51–53] Accordingly, FM is also associated with greater mental health comorbidity than RA,[54] with the prevalence of anxiety and depression reported at over 50% in FM compared with 20% to 35% in RA.[55,56] Such mental health conditions are prominent determinants of worsening pain severity and pain-related outcomes in chronic pain.[31,57,58] For example, in FM, symptom severity of anxiety and depression was associated with physical symptomatology, functional disability, and health-related quality of life.[59–61] PI was also indicated as a factor in mental health comorbidity in chronic pain conditions, moderating the relationship between pain severity and depressive symptoms.[62,63] Moreover, state and trait anger have both been suggested as mediators of the relationship between PI and mental health-related outcomes.[37,39]

In view of the above, the present study aimed to compare PI and anger across 4 diagnostic groups: FM patients with comorbid depression/anxiety (FM+A/D); FM patients without comorbid depression/anxiety (FM-A/D), RA patients, and PFCs. To date, the IEQ has been validated in multiple languages.[19,64–69] We further aimed to translate and validate a Hebrew version of the IEQ, confirming its structure using factor analysis and measuring internal consistency using Cronbach alpha. In line with previous findings, we hypothesized a linear trend such that PI (as measured by the IEQ), state and trait anger, and measures of anxiety, depression, and pain catastrophizing will be greater in FM+A/D compared with FM-A/D, RA, and PFCs, respectively. We further expected PI to positively correlate with all these measures and, within this, that anger would mediate the relationship between PI and pain. Considering the interaction between state and trait anger, we explored whether trait anger would moderate the mediating effect of state anger in the relationship between PI and pain. Finally, we expected PI to associate with clinical measures of pain in FM, namely the Widespread Pain Index (WPI) and Symptom Severity Scale (SSS),[47] above and beyond the effects of pain catastrophizing (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83, Tables S1 to S3, Supplemental Digital Content 2, http://links.lww.com/CJP/B84, Supplemental Digital Content 3, http://links.lww.com/CJP/B85, Supplemental Digital Content 4, http://links.lww.com/CJP/B86). Confirming our hypotheses would support the construct validity for our Hebrew version of the IEQ.

MATERIALS AND METHODS

General Procedure

Data were collected between April 2021 and January 2022 using the online survey platform Qualtrics, and all participants electronically signed an informed consent before beginning the study. After providing demographic information, participants completed a battery of clinical and psychological questionnaires, randomized in order of presentation, which were expected to last about 15 minutes. All measures were given in Hebrew. All procedures were approved by the Tel Aviv Sourasky Medical Center Institutional Review Board (reference number 0494-21-TLV) for clinical patients and by the Tel Aviv-Yaffo Institutional Ethics Committee (2021037) for the healthy control group.

Participants

The sample included 196 individuals (48.83 ± 15.32 years of age, $M \pm SD$; 159 female [81.12%]), who volunteered to participate. This sample size is in line with the common ratio for principal component analysis of 10 to 15 participants per questionnaire item[70] and in line with the sample size of previous IEQ validations.[7,14,16,19,64,65,67,69,71] Patient groups were recruited via the Rheumatology Department at Tel Aviv Sourasky Medical Center, and the PFC group were recruited via social media.

Inclusion criteria for the overall sample required participants to be at least 18 years of age, native Hebrew speakers, currently living in Israel, and not diagnosed with COVID-19. In the PFC group, participants were excluded from the study if they reported having a medical or mental health condition, and if they reported an average pain intensity in the last week > 2 on a 0 to 10 numerical rating scale (NRS).[72] In the RA group, participants were excluded if they reported having a concurrent mental health condition. This resulted in the exclusion of 48 PFCs: 5 due to COVID-19, 19 due to mental health, 12 due to a medical condition, 6 due to pain > 2, and 6 due to not currently living in Israel. From the RA group, 10 were excluded: 4 due to COVID-19 and 6 due to a concurrent mental health condition. From the FM group, 31 were excluded due to COVID-19. Crucially, as approximately half the FM group self-reported as having concurrent depression and/or anxiety, we further divided the FM group based on the presence or absence of these comorbid mental health conditions (A/D). The final groups consisted of 32 PFCs, 34 RA, 64 FM-A/D, and 66 FM+A/D. An additional 46 participants with both FM and RA will be discussed elsewhere.

Measures

Demographic Measures

Demographic measures included age, sex, and years of education.

Clinical Measures

Clinical characteristics included pain duration, pain intensity, and Hebrew Widespread Pain Index (WPI) and Symptom Severity Score (SSS).[47,73,74] Pain duration referred to the number of years since diagnosis of RA or FM condition. Pain intensity referred to the average pain intensity during the last 7 days, assessed using a 0 to 10 NRS, with 0 referring to "no pain" and 10 to "unbearable pain." The WPI is a self-report measure that quantifies the extent of widespread pain throughout the body, assessing the presence of pain or tenderness in the last 7 days in 19 specific body areas, with each affected area receiving one point, resulting

in a score range of 0 to 19. The SSS is a self-report measure that quantifies the symptom severity of 3 items (fatigue, tiredness upon waking, cognitive impairment) during the previous 7 days on a scale of 0 to 3 (no problems; mild; moderately severe; highly severe), adding 1 point each for the presence in the last 6 months of a further 3 items (headaches, lower abdominal pain, and depression), resulting in an overall score range of 0 to 12.

Primary Measures

Perceived Injustice. The IEQ is a psychometric tool to assess PI following a substantial injury and/or in chronic illness, composed of 2 conceptual factors: the severity/irreparability of loss (eg, "My life will never be the same") and blame/unfairness (eg, "It all seems so unfair").[7] It is a 12-item self-report measure, with participants indicating the degree to which they experience each item on a scale of 0 to 4 (never, rarely, sometimes, often, all the time), resulting in a possible score range of 0 to 48.

The instructions and the 12 items of the IEQ were first translated into Hebrew by 3 of the authors independently (O.E., G.G., and V.A.), all fluent speakers of both Hebrew and English and together combining expertise in the relevant scientific literature and medical practice. Having discussed points of divergence and translational challenges arising from their individual translations, these authors then reconciled a single Hebrew IEQ. Next, an independent English-speaking professional translator, naïve to the questionnaire's concepts and original wording, performed a back-translation into English. An adjusted version of the Hebrew version was then confirmed, taking into consideration the translator's comments and confirming that cross-cultural adaptions and meanings remained consistent with the original questionnaire. Notably, Hebrew is a gendered language that inflects parts of speech according to grammatical gender. To avoid self-report biases arising from this,[75] our translation incorporated sex-neutral phrasing. A final Hebrew version of the IEQ was confirmed by the three authors (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83, Appendix 1, Supplemental Digital Content 5, http://links.lww.com/CJP/B87).

To note, instructions of the IEQ were slightly modified; to appropriately address the target sample of patients, "injury" was replaced with "injury or illness," which also broadens the scope of conditions for its potential future. In addition, since we aimed to recruit a group of PFCs, we added instructions asking participants to complete the questionnaire in relation to a recent past injury or illness if they do not currently experience either.

State and Trait Anger. State and trait anger were assessed using a Hebrew State and Trait Anger Expression Inventory (STAXI-2).[41,76] Participants completed the 15-item state anger questionnaire (S-anger), quantifying how well each item described their feelings during the previous 2 weeks on a 1 to 4 scale (not at all; a little; moderately; very much), resulting in a possible score range of 15 to 60. Participants completed the 10-item trait anger questionnaire (T-anger) using the same scale but rating how well each item described how they usually feel, resulting in a possible score range of 10 to 40.

Secondary Measures

Depression. Depression was assessed using the 9-item Patient Health Questionnaire (PHQ-9),[77] quantifying self-reported symptom frequency in the previous 2 weeks on a scale of 0 to 3 (not at all; several days; more than half the days; nearly every day). Responses are summarized, resulting in a possible total score range of 0 to 27.

Anxiety. Anxiety was assessed using the 7-item Generalized Anxiety Disorder scale (GAD-7),[78] quantifying symptom frequency in the previous 2 weeks on a scale of 0 to 3 (not at all; several days; more than half the days; nearly every day). Responses are summarized, resulting in a possible total score range of 0 to 21. The Hebrew PHQ-9 and GAD-7 are freely available online (https://www.phqscreeners.com/).

Pain Catastrophizing. Maladaptive pain-related cognitions were assessed using a 13-item Hebrew Pain Catastrophizing Scale (PCS),[79,80] describing thoughts and feelings about the experience of pain on a 0-4 scale (not at all, to a slight extent, to a moderate extent, to a large extent, to the greatest extent), resulting in a possible score range of 0-52. Items of the PCS relate to the rumination, magnification, and helplessness of pain in chronic pain.

Data Analysis

We first compared demographic and clinical measures across the 4 diagnostic groups using ANOVA and assessed the structural validity and reliability of the IEQ. To test the structural validity, we conducted a factor analysis using Principal Component Analysis (PCA) with oblique (direct oblimin) rotation. Although some validations of the IEQ have employed Confirmatory Factor Analysis,[64,68,69] we opted for PCA in line with the many other IEQ validation studies.[7,19,65–67] Given the varied findings of factor analyses within these papers, we concluded that the more exploratory PCA was preferable to a confirmatory method. Reliability was assessed using Cronbach alpha, providing a measure of the internal consistency of the questionnaire items.

Next, primary and secondary measures were compared across the 4 groups using ANOVA, with post hoc t tests to explore the relationship between specific groups, applying Bonferroni corrections for multiple comparisons. Where the equality of variance could not be assumed, corrected results are reported. Additional covariates were added to examine possible effects of demographic, clinical, and negative-affect–related factors across the groups. Pearson correlations were then used to examine the relationship between the IEQ and all measures; correlations were examined separately between diagnostic groups. An alpha level of 0.05 was used throughout.

On the basis of these correlations, we tested whether state anger mediated the relationship between IEQ and pain intensity, conducting bootstrapped-based mediation using PROCESS.[81] We further explored the role of trait anger in moderating this mediating role of state anger. Bias-corrected 95% CIs were produced, and the total and indirect effects were considered significant if zero was not included in the CI.

In addition, linear regression models were used to assess the association of IEQ with pain-related measures in the patient groups, above and beyond pain catastrophizing. For each clinical pain-related measure (pain intensity, WPI, and SSS), demographic factors and duration of diagnosis were entered into the first step of the model, IEQ to the second step, and PCS to the third. Importantly, since this is a cross-sectional study in nature, none of the analyses denote causality.

RESULTS

Demographic and Clinical Characteristics

Demographic characteristics according to group are presented in Table 1 (top section). There was no significant

difference between the groups in sex ($F_{3,187} = 2.26$, $P = 0.083$) or education ($F_{3,187} = 1.45$, $P = 0.230$), but there was in age ($F_{3,187} = 26.15$, $P < 0.001$). Post hoc analysis (further detailed in Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83) indicated that the H/PFC group was significantly younger than the clinical groups and the RA group was significantly older than all other groups, whereas the age difference between FM-A/D and FM+A/D was not significant after correcting for multiple comparisons.

Clinical characteristics according to group are presented in Table 1 (middle section). Although we found a significant difference in duration of diagnosis between the 3 patient groups ($F_{2,157} = 4.68$, $P = 0.011$), post hoc analysis revealed none of these differences were significant after correcting for multiple comparisons. Pain intensity varied significantly between groups ($F_{3,192} = 82.47$, $P < 0.001$), such that pain intensity was greater in FM groups compared with RA and greater in RA compared with PFC group (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83). We found that WPI also varied significantly between groups ($F_{3,192} = 41.16$, $P < 0.001$), with widespread pain greater in FM groups compared with RA and in RA compared with PFC (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83). In addition, SSS varied significantly between groups ($F_{3,192} = 70.65$, $P < 0.001$), with post hoc analysis indicating greater levels of symptom severity in FM groups compared with RA and PFC (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83). The 2 FM groups did not differ between themselves, yet, as might be expected, they both demonstrated higher levels of pain and pain-related symptoms, as well as greater distribution of pain across the body, compared with the RA and PFC groups.

Structural Validity and Reliability of the IEQ

The Kaiser-Meyer-Olkin (KMO) value was 0.94, exceeding the minimum recommended value of 0.60, and Bartlett test of sphericity was significant ($\chi^2 = 2077.32$, $P < 0.001$), demonstrating the suitability of the data for PCA. One component met Kaiser criterion with eigenvalues > 1; this component had an eigenvalue of 8.04 and explained 66.95% of the variance. For 12 items in a sample of n = 196, parallel analysis based on an estimation of 1000 random matrices with values corresponding to the 95th percentile of random eigenvalues[82,83] provided an eigenvalue criterion of 1.42, which, again, only the first component in the present analysis exceeded. Examination of the scree plot (Fig. 1) indicated a bending point immediately after the first component, suggesting 1 factor was sufficient to explain most of the underlying data.

The component matrix (Table 2) further indicated that all items had good loadings (> 0.60) on this first component. All communality values exceeded 0.38, indicating a good fit of all items among themselves. Cronbach alpha coefficient was 0.95, with no item showing substantial reductions from this value if deleted (Table 2). Results thus support a 1-factor structure including all items, with good internal consistency.

Primary and Secondary Measures

All primary and secondary measures according to diagnostic group are presented in Table 1 (bottom section). We found a significant difference in IEQ across the 4 groups (Table 1; $F_{3,192} = 34.79$, $P < 0.001$), which remained significant after controlling for demographic factors (age, sex, and education; $F_{6,188} = 19.28$, $P < 0.001$), and, additionally, for depression and anxiety ($F_{8,186} = 48.08$, $P < 0.001$). To control for the effect of duration of diagnosis, we assessed IEQ across the 3 patient groups only, with the difference remaining significant after controlling for demographic factors, diagnosis duration, depression, and anxiety ($F_{8,151} = 28.94$, $P < 0.001$). In line with our hypothesis, post hoc analysis indicated a significant difference in IEQ between all groups, such that levels of PI were greater in FM +A/D than FM-A/D, greater in FM-A/D than RA, and greater in RA than PFC (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83). This also supports the construct validity of the Hebrew IEQ.

TABLE 1. Sample Characteristics According to Diagnostic Group

	PFC	RA	FM-A/D	FM+A/D
Group size (n)	32	34	64	66
Sex (female, %)	21 (65.63)	26 (76.47)	53 (82.81)	59 (89.39)
Age (y)	35.28 ± 13.24*	61.91 ± 13.43*	51.22 ± 11.92 $^{HC, RA,}$†	46.35 ± 14.13 $^{HC, RA,}$†
Education (y)	16.80 ± 7.75	15.09 ± 2.57	14.86 ± 3.23	14.36 ± 2.96
Duration of diagnosis (y)	NA	12.16 ± 10.48†	8.67 ± 6.17	7.76 ± 5.00 †
Pain intensity	0.69 ± 0.90*	3.65 ± 3.06*	7.23 ± 2.25 $^{HC, RA}$	7.18 ± 2.17 $^{HC, RA}$
WPI	0.72 ± 0.81*	2.56 ± 2.94*	7.58 ± 4.49 $^{HC, RA}$	8.92 ± 4.91 $^{HC, RA}$
SSS	2.45 ± 2.00$^{FM-A/D, FM+A/D}$	3.03 ± 2.55 $^{FM-A/D, FM+A/D}$	8.02 ± 2.96 $^{HC, RA}$	8.76 ± 2.45 $^{HC, RA}$
IEQ	4.53 ± 5.95*	12.94 ± 10.93*	21.78 ± 12.66*	28.30 ± 12.87*
S-Anger	19.00 ± 5.46 $^{FM-A/D, FM+A/D}$	17.79 ± 3.18 $^{FM-A/D, FM+A/D}$	22.83 ± 8.16 $^{HC, RA}$†	26.30 ± 11.30 $^{HC, RA}$†
T-Anger	16.88 ± 5.62†	16.18 ± 4.78 $^{FM+A/D}$	17.61 ± 6.10	19.64 ± 6.82 ^{RA}†
PHQ-9	4.06 ± 3.66 $^{FM-A/D, FM+A/D}$	5.09 ± 5.34 $^{FM-A/D, FM+A/D}$	12.09 ± 6.16*	16.05 ± 6.60*
GAD-7	4.25 ± 4.52 $^{FM+A/D}$	3.50 ± 4.41 $^{FM+A/D}$†	5.77 ± 5.12 $^{FM+A/D}$†	9.17 ± 6.02*
PCS	7.50 ± 8.11*	15.12 ± 13.56*	24.92 ± 13.87 $^{HC, RA}$†	30.56 ± 13.96 $^{HC, RA}$†

Mean ± SD unless noted otherwise.

*Significantly different to all other groups.

†Significant non-corrected difference between groups with this sign.

FM+A/D indicates fibromyalgia with mental health condition; FM-A/D, fibromyalgia without mental health condition; GAD-7, Generalized Anxiety Disorder scale; IEQ, Injustice Experience Questionnaire; PCS, Pain Catastrophizing Scale; PFC, Pain-free controls; PHQ-9, Patient Health Questionnaire; RA, Rheumatoid Arthritis; S-anger, State Anger Inventory; SSS, Symptom Severity Scale; T-anger, Trait Anger Inventory; WPI, Widespread Pain Index.

$^{HC, RA, FM-A/D, FM+A/D}$ = significantly different only to groups indicated.

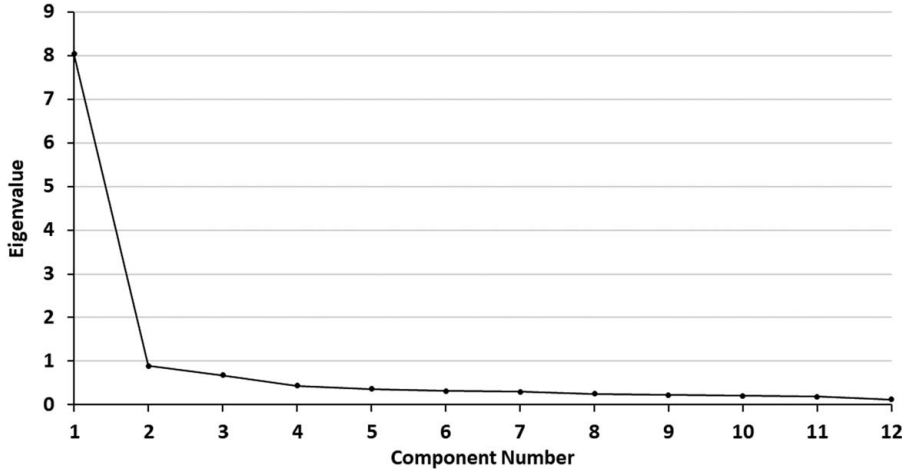

FIGURE 1. Scree plot of the factorial structure of the Hebrew IEQ. The bending point immediately after the first component indicates a one-factor structure.

We found a significant difference in both state and trait anger across the groups (S-anger: $F_{3,192} = 9.80$, $P < 0.001$; T-anger: $F_{3,192} = 3.05$, $P = 0.030$), which remained significant after controlling for demographic factors (S-anger: $F_{6,188} = 5.13$, $P < 0.001$; T-anger: $F_{6,188} = 2.18$, $P = 0.046$) and, additionally, for depression and anxiety (S-anger: $F_{8,186} = 32.04$, $P < 0.001$; T-anger: $F_{8,186} = 10.85$, $P < 0.001$). Across the 3 patient groups, both state and trait anger remained significantly different after controlling for demographic factors, depression and anxiety, and duration of diagnosis (S-anger: $F_{8,151} = 28.47$, $P < 0.001$; T-anger: $F_{8,151} = 11.28$, $P < 0.001$). In line with our hypothesis, post hoc analysis indicated greater levels of state anger in FM groups compared with RA and PFC, but not in FM+A/D compared with FM-A/D, nor in RA compared with PFC. In support of our hypothesis, post hoc analysis indicated trait anger was greater in FM+A/D compared with RA but, after correcting for multiple comparisons, did not differ significantly between any of the other groups (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83).

We found significant difference in depression scores ($F_{3,192} = 43.60$, $P < 0.001$) and anxiety scores ($F_{3,192} = 11.65$, $P < 0.001$) across the groups. In line with our hypothesis, post hoc analysis indicated higher levels of depression in FM+A/D compared with FM-A/D, and in FM-A/D compared with RA, although not in RA compared with PFCs. Further supporting our hypothesis, anxiety was significantly greater

TABLE 2. Principle Component Analysis (PCA) and Cronbach Alpha of the Hebrew IEQ

IEQ item		Component loadings	Commonalities	Cronbach alpha if item deleted
10*	אני חש/ה כאילו נשדד ממני משהו מאוד יקר I feel as if I have been robbed of something very precious	0.886	0.786	0.949
6	אני חש/ה שזה השפיע עליי בצורה בלתי הפיכה I feel that this has affected me in a permanent way	0.875	0.766	0.949
4	אף אחד/ת לא צריכ/ה לחיות בצורה שכזו No-one should have to live this way	0.868	0.753	0.949
5	אני רק רוצה לקבל את חיי בחזרה I just want to have my life back	0.866	0.749	0.949
9*	אין דבר שאי פעם יוכל לפצות אותי על כל מה שעברתי Nothing will ever make up for all that I have gone through	0.847	0.718	0.950
7*	הכל נראה כל כך לא הוגן It all seems so unfair	0.847	0.717	0.950
2	חיי לעולם לא יהיו כפי שהיו My life will never be the same	0.840	0.705	0.950
11*	אני מוטרד/ת מפחדים שלעולם לא אוכל להגשים את חלומותיי I am troubled by fears that I may never achieve my dreams	0.840	0.705	0.950
8	אני מודאג/ת שהמצב שלי לא נלקח ברצינות I worry that my condition is not being taken seriously	0.808	0.652	0.951
12*	אני לא מאמינ/ה שזה קרה לי I can't believe that this happened to me	0.789	0.622	0.952
1	רוב האנשים לא מבינים עד כמה חמור מצבי Most people don't understand how severe my condition is	0.694	0.482	0.955
3*	אני סובל/ת בגלל רשלנות של מישהו/י אחר/ת I am suffering because of someone else's negligence	0.620	0.384	0.957

*Items that comprise the blame/unfairness conceptual subscale of the IEQ. All other items comprise the severity/ irreparability of loss conceptual subscale.

in FM+A/D compared with all other groups (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83).

Finally, we found a significant difference in pain catastrophizing across the groups ($F_{3,192} = 26.68$, $P < 0.001$), with further analysis supporting our hypothesis, indicating significantly greater catastrophizing in FM groups compared with RA and in RA compared with PFC (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83).

Anger as a Mediator Between Perceived Injustice and Pain Intensity

Correlations across all measures and per group are presented in Table 3. In line with our hypothesis, the IEQ demonstrated moderate to strong positive correlations with all related constructs in the 3 patient groups, further supporting its construct validity. In line with previous reports,[12,13,15,19,64,66] we observed high correlations between the IEQ and PCS, particularly in the patient groups, calling into question their conceptual and/or statistical distinctiveness. Our regression analyses examining the association of IEQ with pain measures above and beyond PCS (presented in the Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83, Tables S1 to S3, Supplemental Digital Content 2, http://links.lww.com/CJP/B84, Supplemental Digital Content 3, http://links.lww.com/CJP/B85, Supplemental Digital Content 4, http://links.lww.com/CJP/B86) indicated that in most cases, neither IEQ nor PCS remain significant when the other is included in the model. Nevertheless, the IEQ contributed unique variance above and beyond the PCS in more cases than the reverse. This suggests a separation between PI and pain catastrophizing despite the strong correlation observed.

We observed significant positive correlations between IEQ, state anger and pain intensity in the FM+A/D group, but the correlations between state anger and pain intensity were not significant in any other group. In light of this, we conducted a mediation analysis on the FM+A/D group only. The results of our mediation model indicated a significant positive total effect of IEQ on pain intensity (c: $\beta = 0.10$, $P < 0.001$), a significant positive direct effect within the mediation model (c': $\beta = 0.09$, $P < 0.001$), and a significant positive effect between IEQ and state anger (a: $\beta = 0.55$, $P < 0.001$). However, the positive effect between state anger and pain intensity when controlled for by IEQ was not significant (b: $\beta = 0.02$, $P = 0.428$). Unlike our hypothesis, the indirect mediating effect of IEQ on pain intensity through anger was not significant (a*b: $\beta = 0.01$, BCI = −0.009 to 0.033). In view of the strong relationship between state anger and IEQ, it seems the mediating effect did not explain additional variance in pain intensity.

Exploratory Moderated Mediation Analysis

As indicated in Table 3, we observed a strong correlation between state and trait anger in all 4 groups and a significant correlation between trait anger and IEQ in all 3 patient groups. We therefore conducted an exploratory mediation analysis, assessing the moderating effect of trait anger on state anger[22] as a mediator of the relationship between IEQ and pain intensity (Fig. 2A), in all 3 patient groups.

We found that trait anger moderated the indirect mediation effect in the FM+A/D group, but not in the FM-A/D or RA groups. In each of the RA and FM-A/D groups (Fig. 2B, C), the model indicated significant direct effects of IEQ on pain intensity (FM-A/D: $\beta = 0.07$, $P = 0.008$; RA: $\beta = 0.15$, $P = 0.007$), but the overall indirect effect of mediation by state anger was not significant at any level of trait anger, in either group (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83, Table S4, Supplemental Digital Content 6, http://links.lww.com/CJP/B88). In the FM+A/D group (Fig. 2D), the model indicated a significant direct effect of IEQ on pain intensity ($\beta = 0.10$, $P < 0.001$) and an indirect effect of the mediation by state anger that was significant only when trait anger was high (for the 84th percentile of T-anger: $\beta = 0.05$, BCI = 0.021 to 0.090; 50th percentile: $\beta = 0.01$, BCI = −0.006 to 0.032; 16th percentile: $\beta = 0.002$, BCI = −0.010 to 0.016). Moreover, the FM+A/D group showed a significant effect of T-Anger ($\beta = -0.27$, $P < 0.001$) and of the T-anger*S-anger interaction ($\beta = 0.005$, $P = 0.033$) on pain intensity, whereas these effects were not significant in the FM-A/D or RA groups (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83, Table S4, Supplemental Digital Content 6, http://links.lww.com/CJP/B88). We also found a significant interaction effect of IEQ*T-anger on S-anger in both FM groups (FM+A/D: $\beta = 0.03$, $P < 0.001$; FM-A/D: $\beta = 0.02$, $P = 0.037$), but not in the RA group. That is, the interaction between IEQ and trait anger moderated state anger in FM, but not in RA.

DISCUSSION

Accumulating evidence indicates that perceptions of injustice in chronic pain, as measured by the IEQ, are a crucial risk factor for adverse pain outcomes.[8] In addition, anger, which is a prominent emotional reaction to injustice,[21,22,29] was previously found to mediate the relationship between PI and pain-related outcomes.[15,37] Although both PI and anger have been associated with comorbid mental health severity in people with chronic pain,[37,39,62,63] we here demonstrate that individuals diagnosed with FM, particularly those with self-reported mental health comorbidities, had the most severe scores in all clinical and psychological measures that we assessed. Crucially, the FM+A/D group had higher IEQ and state and trait anger scores compared with FM patients without mental health comorbidities, as well as compared with RA patients and pain-free individuals. These results remained significant when controlling for demographics and several other factors, such as depressive symptoms and duration of diagnosis. Notably, only within the FM+A/D group the current state of feeling angry mediate the relationship between PI and pain, but only for those with a larger propensity to generally feel angry. This highlights that state and trait anger interact to differentially associate with pain severity across chronic pain groups; while the mediation by state anger provides insight into how PI affects pain intensity, the moderation by trait anger informs who might be more susceptible to this manner of pain potentiation. This result thus identifies a potential modifiable target and a potential patient group for specific clinical treatment.[84–86]

While cross-sectional in nature, these findings expand our understanding of the complex interaction between perceived injustice and anger, especially as they relate to differential clinical manifestations of chronic pain and comorbid mental health conditions. In turn, this may advance our theoretical understanding of the affective pathway by which PI seems to impact adverse chronic pain

TABLE 3. Correlation Matrix of IEQ and Related Factors, According to Diagnostic Group

	Pain intensity	WPI	SSS	PHQ-9	GAD-7	PCS	T-anger	S-anger	IEQ
Pain Intensity	1 – PFC	—	—	—	—	—	—	—	—
	1 – RA	—	—	—	—	—	—	—	—
	1 – FM-A/D	—	—	—	—	—	—	—	—
	1 – FM+A/D	—	—	—	—	—	—	—	—
WPI	0.407*	1	—	—	—	—	—	—	—
	0.740**	1	—	—	—	—	—	—	—
	0.304*	1	—	—	—	—	—	—	—
	0.392**	1	—	—	—	—	—	—	—
SSS	0.156	0.441*	1	—	—	—	—	—	—
	0.567**	0.439**	1	—	—	—	—	—	—
	0.459**	0.478**	1	—	—	—	—	—	—
	0.527**	0.484**	1	—	—	—	—	—	—
PHQ-9	−0.102	0.277	0.731**	1	—	—	—	—	—
	0.496**	0.433*	0.808**	1	—	—	—	—	—
	0.445**	0.395**	0.704**	1	—	—	—	—	—
	0.477**	0.494**	0.740**	1	—	—	—	—	—
GAD-7	−0.092	0.257	0.351*	0.602**	1	—	—	—	—
	0.403*	0.333	0.687**	0.814**	1	—	—	—	—
	0.283*	0.188	0.480**	0.717**	1	—	—	—	—
	0.389**	0.410**	0.592**	0.691**	1	—	—	—	—
PCS	0.258	0.056	0.100	0.306	0.408*	1	—	—	—
	0.510**	0.366*	0.666**	0.651**	0.666**	1	—	—	—
	0.411**	0.218	0.464**	0.663**	0.714**	1	—	—	—
	0.603**	0.390**	0.542**	0.639**	0.730**	1	—	—	—
T-anger	0.030	0.515**	0.362*	0.584**	0.426*	0.253	1	—	—
	0.068	0.133	0.456**	0.609**	0.698**	0.407*	1	—	—
	0.208	−0.146	0.277*	0.404**	0.431**	0.420**	1	—	—
	0.110	0.475**	0.296*	0.371**	0.586**	0.458**	1	—	—
S-anger	−0.152	0.429*	0.623**	0.721**	0.572**	0.186	0.651**	1	—
	0.148	0.149	0.505**	0.636**	0.688**	0.451**	0.586**	1	—
	0.164	0.120	0.268*	0.552**	0.784**	0.590**	0.413**	1	—
	0.435**	0.474**	0.462**	0.524**	0.738**	0.606**	0.696**	1	—
IEQ	−0.053	−0.128	0.198	0.516**	0.234	0.569**	0.198	0.201	1
	0.502**	0.550**	0.556**	0.617**	0.585**	0.726**	0.449**	0.527**	1
	0.369**	0.378**	0.465**	0.625**	0.634**	0.819**	0.278*	0.584**	1
	0.598**	0.396**	0.606**	0.725**	0.680**	0.827**	0.452**	0.623**	1

Top to bottom within each cell refers respectively to Pain-free Controls PFC; rheumatoid arthritis (RA); fibromyalgia without anxiety/depression (FM-A/D); fibromyalgia with anxiety/depression (FM+A/D), as indicated in the first cell.

GAD-7 indicates Generalized Anxiety Disorder scale; IEQ, Injustice Experience Questionnaire; PCS, Pain Catastrophizing Scale; PHQ-9, Patient Health Questionnaire; S-anger, State Anger Inventory; SSS, Symptom Severity Scale; T-anger, Trait Anger Inventory; WPI, Widespread Pain Index.

*Within-group significance at the 0.05 level (2-tailed).

**Within-group significance at the 0.01 level (2-tailed).

outcomes,[8,22] and may support the development of more precision-based, personalized interventions.

Unlike our hypothesis, we did not replicate previous findings of anger mediating the relationship between PI and pain intensity consistently across the groups.[11,15,37] This may be a result of the high correlations we observed between PI and state anger in all diagnostic groups, minimizing the statistical ability of state anger to explain variability in pain intensity beyond PI. Moreover, it should be noted that previous mediation results were reported in samples of general musculoskeletal pain[37] and postinjury spinal cord pain,[39] and/or in studies that failed to systematically differentiate between dimensions of anger (state, trait, expression, and regulation[22]), whereas our study focused specifically on state and trait anger in FM and RA. In addition, the present lack of anger-mediation may be due to the lack of correlation of anger with pain intensity, as well as WPI and SSS in the RA and FM-A/D groups. This suggests that in these patient groups, the role of anger in explaining pain severity is minimal, particularly compared with the roles of depression and anxiety. However, in the FM+A/D group, we observed no significant differences between anger, depression, and anxiety in the strength of their correlations with pain intensity, WPI, or SSS, further highlighting the complexity of anger in the context of pain. Indeed, in FM +A/D, anger seems to uncover an intricate relationship with pain severity that, while being on par with that of other negative affect–related factors (such as depression and anxiety), may reveal unique opportunities for understanding how pain is maintained and chronified. This furthers similar findings,[2,22,87,88] demonstrating the complex interactions between anger, pain, and other negative affect-related factors.

Consistent with our findings, a recent meta-analysis indicated a stronger correlation of pain with state, compared with trait anger,[22] which authors speculated to reflect a synchronization of state-level anger and pain-related symptom fluctuation. Indeed, considering this variability in state anger to be largely associated with underlying trait anger,[32,89] and given the strong positive correlations we observed between state and trait anger, our exploratory analysis indicated an interaction between the 2. We found

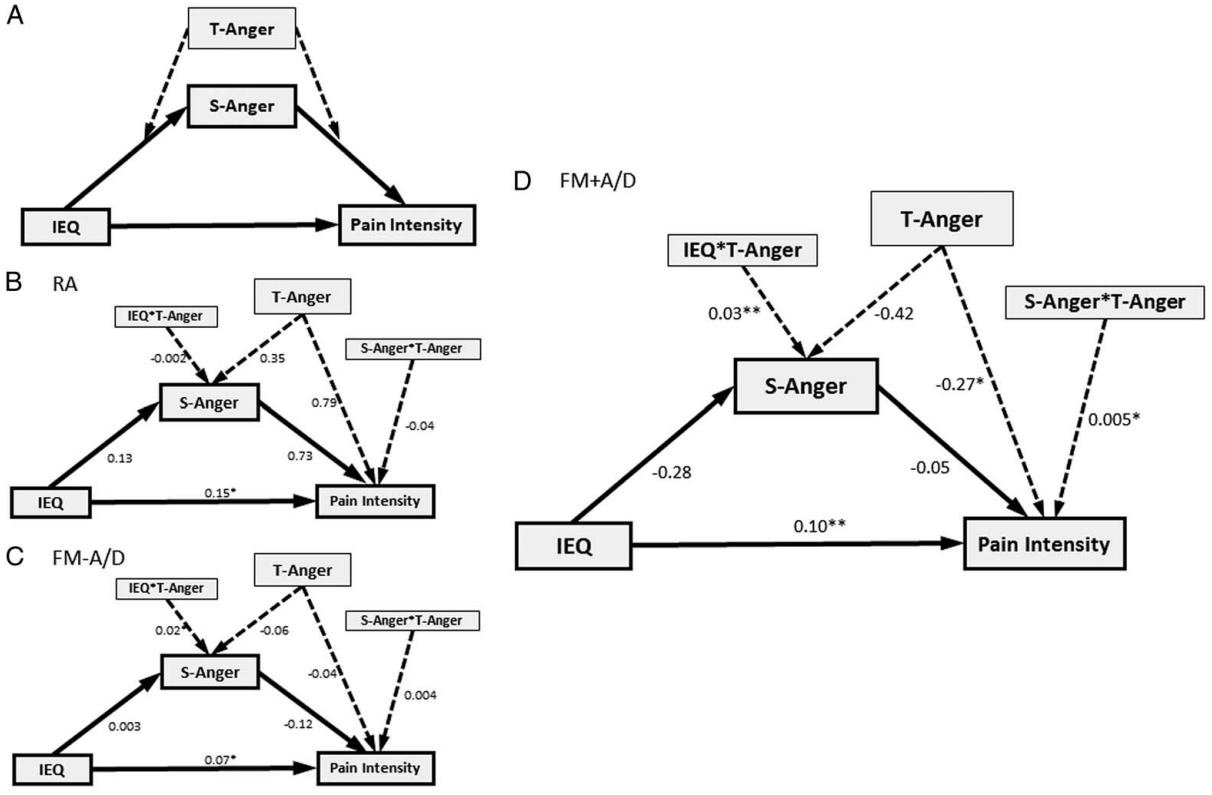

FIGURE 2. Moderated mediation model illustrating the moderating effect of trait anger on the mediating effect of state anger in the relationship between perceived injustice and pain intensity. A, Conceptual illustration of the model. B, Statistical model for the rheumatoid arthritis (RA) group. C, Statistical model for the fibromyalgia without anxiety/depression (FM-A/D) group. D, Statistical model for the fibromyalgia with anxiety/depression (FM+A/D) group. Path coefficients are shown. Solid arrows indicate mediation pathways; broken lines indicate moderation pathways. IEQ indicates Injustice Experience Questionnaire; S-anger, State Anger Inventory; T-Anger, Trait Anger Inventory. *significance at $P < 0.05$ **significance at $P < 0.001$.

trait anger to be a moderator in the FM+A/D group only, such that when trait anger was high, state anger mediated the relationship between PI and pain intensity. Interestingly, we also found that PI and trait anger had an interaction effect on state anger in both FM groups but not in the RA group, suggesting that the differential effects of anger could depend additionally on the specific diagnosis of chronic pain. Taken together, these findings highlight the importance of different dimensions of anger and of acknowledging their complexity in relation to differing clinical manifestations of chronic pain, taking into account potential mental health comorbidities.[24–30,32] Future work could helpfully extend these findings, systematically exploring the differential effects of anger and PI across more chronic pain and mental health conditions, particularly in experimental and longitudinal studies.

The systematic differences we observed in IEQ scores across the diagnostic groups support previous reports of increased PI in FM compared with RA[42] and in chronic pain with, compared with without, comorbid depression,[62] further validating our Hebrew version of the IEQ. In line with previous suggestions, the elevated PI in FM compared with RA could in part be due to the higher levels of pain reported by FM patients, as well as a potential result of the extended diagnostic process and relative lack of understanding associated with FM.[42,50,51] In addition, FM has been associated with elevated levels of abuse and victimization[90,91] and, as such, the increased IEQ and anger we observed in FM compared with RA could represent a legitimate and at least partially adaptive response to experienced trauma and injustice earlier in life.[45] We extend these findings through our specific comparison of the FM +A/D and FM-A/D groups, highlighting the need for a broader understanding of mental health comorbidity in FM. The increased depression, anxiety, and anger scores observed in the FM+A/D group both confirm the use of our self-report measure to distinguish the FM groups and support the high prevalence of emotion-related mental health comorbidity in FM more generally.[54]

Although some level of PI and anger in FM might be adaptive,[92] our findings indicate that, particularly within the FM+A/D group, a substantial part of this anger seems maladaptive, as it is associated with elevated pain severity and mediates the relationship between PI and pain intensity. This highlights the potential clinical distinction between FM-A/D and FM+A/D groups, emphasizing the importance of better understanding how we think about, diagnose, and treat chronic pain-related mental health comorbidities.[2,56,57,61,93] For example, considering recommendations of PI as a target for therapeutic intervention,[43,94,95] the successive pattern of elevated PI we observed in RA, FM-A/D, and FM+A/D suggests that PI may represent a more beneficial target for intervention in FM compared with RA, and in the FM+A/D group specifically. As such, our findings represent an important advance toward personalizing patient-oriented treatments for chronic pain.

In validating our Hebrew version of the IEQ, we confirmed a 1-factor structure for all 12 items with excellent reliability. Previous validations and translations of the IEQ have yielded mixed results regarding a 2-factor[64–66,69] versus 2-factor[19,67,68] structure, although the latter consistently reports very high overlap between the components. As such, our findings support Sullivan et al's[7] original conception that the IEQ might be best construed as a complex but unitary construct, with the theoretical subscales not deriving statistically separate components. In either case, it is important to consider that the items comprising each conceptual subscale may not be consistent across translations, as noted in the Spanish and Danish versions.[19,65] Our Hebrew IEQ further demonstrated strong relationships with all clinical and psychological measures assessed, across all patient groups, indicating strong construct validity. Together, our findings thus support the psychometric properties of the Hebrew IEQ, indicating its suitability to be used as a measure of PI across varying chronic pain diagnoses, with and without mental health comorbidities.

In the patient groups, the strong correlations between the IEQ and PCS replicate findings both in the IEQ's initial development[7] and more recently (ranging from 0.65 to 0.75).[12,13,15,19,64,66] At the same time, these studies indicated a tendency of the IEQ to explain additional variability above and beyond PCS. In the current study (Supplemental Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B83, Tables S1–S3, Supplemental Digital Content 2, http://links.lww.com/CJP/B84, Supplemental Digital Content 3, http://links.lww.com/CJP/B85, Supplemental Digital Content 4, http://links.lww.com/CJP/B86), IEQ scores explained additional pain variability above and beyond PCS only within specific patient groups and pain measures, whereas PCS explained additional variability above and beyond IEQ in only one such case. Previous studies report differential longitudinal outcomes between the IEQ and PCS, with PI, for example, found to mediate the relationship of pain intensity with the quality of life and emotional functioning at a 3-month follow-up, whereas pain catastrophizing mediated the relationship between pain intensity and 3-month social functioning.[15] Together, PI and pain catastrophizing likely represent cognitively similar, and generally maladaptive coping responses to chronic pain, yet there remains a distinction in their specific associations with pain severity measures and in their contributions to long-term clinical and psychosocial outcomes.

To note, a validation study of another Hebrew version of the IEQ was published after we completed data collection.[71] Although that study was conducted on a traumatic injury-related sample in a physical therapy setting, our study focused on chronic pain and provided broader construct validity, both by comparing across clinical diagnostic groups and by associating the IEQ with various related clinical and psychological constructs. In this regard, we observed high correlations between the IEQ and measures of anxiety, depression, anger, and pain catastrophizing, which may represent a wider negative-affectivity bias across self-report measures, at least within the 3 chronic pain groups. In light of this, it is important to note that the phrasing of the IEQ instructs participants to mark how often they experience each item "when thinking about their injury or illness," and so we maintain the conceptualization of PI as injustice related to the experience of conditions like RA and FM, rather than part of an overarching negativity bias. Other limitations to note are that, due to the cross-sectional design of our study, conclusions as to the directionality of the relationships explored should be drawn with caution. Although our correlations are strong and consistent, future longitudinal or intervention-based research could helpfully target PI to explore its effects on pain intensity, as well as inducing state anger[76] to explore its mediating effects between the 2. Moreover, our validation of the Hebrew IEQ is based largely on convergent validity, whereas future validation studies could beneficially consider its discriminant validity in relation to associated constructs. Regarding limitations on generalizability, it should be noted that our patients were recruited from a large clinical center in the center of Israel, but still just the one and healthy participants were recruited through social media. In addition, our sample consisted mostly of females (80%). The perception of FM as almost exclusively affecting women has recently been challenged.[48] Thus, although our findings remained significant when controlling for sex, future studies should aim for the inclusion of a more equal sex distribution.

Overall, the present study offers a novel understanding of the interaction between state and trait anger in the context of PI in chronic pain, particularly for individuals experiencing comorbid mental health conditions. This may have important clinical implications, highlighting the potential for therapeutic interventions that target state anger in improving pain intensity associated with higher levels of PI. Although such interventions may be inapplicable to more general chronic pain cohorts, our findings suggest their efficacy in a more tailored approach, specifically to FM patients with mental health comorbidity who have high baseline trait anger.

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