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The Effects of Chronic Pain as Continuous Traumatic Stressors

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Abstract

Background: The study aims to provide empirical evidence that Chronic Pain (CP) is an independent continuous traumatic stressor that yields symptoms of Peri-Continuous Traumatic Stress Syndrome (PCTSS) that include symptoms of PTSD, depression, anxiety, pathological dissociation, somatization, executive function deficits, and disability that warrant significant mental health treatment. **Methods:** In a sample of 211 chronic pain adult patients, 75.4% were female, aged 18 to 65. We measured CP, all the components of PCTSS, and cumulative stressors and traumas. We conducted Hierarchical Multiple Regression (HMR) and Structural Equation Modeling (SEM). In the HMR analyses, the dependent variables were the PCTSS components; in each analysis, in the first step, we entered demographics, cumulative stressors, and traumas in the second step and chronic pain in the last step as the independent variables. In the SEM analysis, chronic pain predicted the latent variables of mental health and executive function deficits, as well as the observed variable of disability. **Results:** The results of HMR and SEM found that CP accounted for a significant independent variance for each PCTSS component above and beyond all other cumulative stressors and trauma contributions. **Conclusions:** This is the first study that provides evidence that chronic pain is an independent predictor of PTSD and other PCTSS syndrome components. The conceptual implications of shifting the paradigm we used to frame chronic pain as an independent variable that yields PCTSS syndrome, and the clinical implication for the importance of psychological treatment of chronic pain, especially to reduce the pain drug-overdose suicide epidemic, are discussed.

Keywords

Chronic Pain, Continuous Traumatic Stress, Type III Trauma, PTSD, Overdose

1. What Are Chronic Pain and Its Consequences?

Chronic Pain (CP) is defined as pain that occurs on most days for at least three months (IASP, 1994). Nearly 20% of the global population experiences Chronic Pain (CP) (Goldberg & McGee, 2011). A study conducted by the US Centers for Disease Control and Prevention (CDC) revealed that the lifetime prevalence of CP in the USA ranges from 11% to 40%. The study also noted higher prevalence rates among women, individuals from lower socioeconomic backgrounds, military veterans, and people living in rural areas (Interagency Pain Research Coordinating Committee, 2016). CP can have a profound impact on relationships and self-esteem and is linked to higher rates of divorce, suicide, substance abuse, and reduced life expectancy (Smith et al., 2014). The experience of physical and psychological pain is a common source of stress in our daily lives. Some people even say, "No pain, no gain". However, Chronic Pain (CP) is on a different level altogether, as it involves ongoing traumatic pain stressors. Increased interest in Chronic Pain (CP) as a distinct disease entity and its global burden calls for a plausible operational definition of the phenomenon. It would, therefore, be of immense value to re-conceptualize CP as continuous traumatic stressors. Chronic Pain (CP), as a continuous traumatic stress and its profound impact on health (e.g. disability), somatization, mental health-related suicidal overdose, and executive function deficits, was hardly considered as part of the conditions that warrant a diagnosis of PTSD.

1.1. The Continuous Traumatic Stress Model for Chronic Pain

The continuous traumatic stress (type III trauma) model is an emerging paradigm (Eagle & Kaminer, 2013; Kira et al., 2008, 2013; Kira, 2021, 2022) that may help us better understand the fundamental dynamics of traumatization in natural, ongoing phenomena, such as chronic pain. For instance, low back chronic pain that continues with the patient for long time is one of the most common contributors to disability (Hoy et al., 2014). Chronic pain has been associated with anxiety and depression (Gureje et al., 1998), dependence on opioids, and overdose (Institute of Medicine, 2011).

1.2. Chronic Pain, Overdose Epidemic, Mortality and Disabilities

Rates of overdose due to chronic pain are high (Vowles et al., 2015; López-Martínez et al., 2019). Overdose is responsible for a significant number of deaths globally. Over the past decade, the rise of the opioid epidemic, especially in the United States, Europe, and worldwide, has led to a significant increase in mortality and disability (e.g. Martins et al., 2015). From 1999 to 2019 in the United States, opioid overdose from both regularly prescribed and illegally acquired drugs was the cause of death for nearly 500,000 people (Centers for Disease Control and Prevention, 2024). The use of opioid-based treatments for pain management has been the traditional approach despite research showing their low effectiveness and potential long-term risks for addiction and suicide (Busse et al., 2018; Chou et al., 2022). Furthermore, severe pain is linked to an increased risk of opioid abuse (Novak et

al., 2016) and dependence (Blanco et al., 2016). The escalating misuse and dependence on opioids have been identified as a significant contributor to the high number of drug overdose deaths in the United States, leading to its classification as a public health crisis (Rudd et al., 2016; Saloner et al., 2018). Chronic pain is one significant factor contributing to drug overdose, and its mortal consequences have been grossly misunderstood. The right understanding of CP dynamics and addressing it as continuous traumatic stress should help us find better ways to treat and deal with its negative health, mental health, and cognitive consequences and the overdose epidemic.

1.3. Chronic Pain, Traumatic Stress, PTSD, and Dissociation

According to Kira et al. (2023b), CP fall under the category of type III-e trauma (continuous life-threatening health conditions) despite not being immediately life-threatening. A significant percentage of individuals living with chronic pain contemplate or commit suicide due to various factors, including the severity or intolerability of the pain, which can lead to overdose (e.g. Calati et al., 2015). CP is considered a continuous stressor, which aligns with type III trauma, in contrast to types I (a single past acute stressor) and II (a sequence of past acute stressors).

Research in the pain literature proposed that PTSD affects chronic pain severity. For instance, some studies (e.g. Fishbain et al., 2017; Gasperi et al., 2021; Miró et al., 2008; Siqveland et al., 2017) indicated that PTSD symptoms can increase the perception of pain. As a result, PTSD may be a risk factor for increasing CP severity. Early research generally either did not explore the direction of the relationship between the two variables (CP and PTSD) or focused more on the effects of PTSD on CP severity. Research has observed a high co-occurrence of chronic pain and PTSD without implying a causal link between CP as continuous traumatic stressors and PTSD (e.g. Morasco et al., 2013; Otis et al., 2003; Outcalt et al., 2015). Research has demonstrated that this co-occurrence is associated with increased pain intensity and functional impairment (Åkerblom et al., 2017; Outcalt et al., 2015; Ruiz-Párraga & López-Martínez, 2014). It was suggested that these concurrent and lagged associations may contribute to cyclical symptom exacerbation and may be mutually maintained via several shared pathways, including attentional biases to pain and threat, avoidant styles of emotional coping, and heightened anxiety sensitivity (i.e. fear of anxiety-related sensations; Brennstuhl et al., 2015; Sharp & Harvey, 2001). Post-traumatic stress and chronic pain conditions may influence each other in a way that exacerbates symptoms (Sharp & Harvey, 2001), which may result in a vicious cycle or loop of perpetuating distress and an increased likelihood of abusing opioids to cope with the severity and chronicity of pain and distress (Asmundson & Katz, 2009). Gasperi et al. (2021) noticed that people with chronic pain that does not have Criterion A' trauma displayed PTSD symptoms.

The current literature has not fully acknowledged that chronic pain itself can be a persistent traumatic stressor that predicts not only PTSD but also other men-

tal health conditions, such as depression and anxiety, which could ultimately lead to overdose and suicide. In addition, chronic pain, as a severe traumatic stressor, is not commonly assessed like other traumatic stressors in predicting pathological dissociation. Our current study aims to explore this potential outcome for chronic pain. By investigating the link between pathological dissociation and chronic pain, we aim to provide further evidence supporting the chronic pain model as a continuous traumatic stressor encompassing both ongoing acute and non-acute stressors.

1.4. Chronic Pain and Somatization

Furthermore, the relationship between pain as a sensory phenomenon and somatization is strong. A meta-analytic study (Burke et al., 2015) found that Individuals with CP were most likely to experience psychological problems in physically focused areas—namely, pain anxiety and somatization. CP is highly correlated with somatization and Somatic Symptom Disorder (SSD), which ranges between 0% - 53% (Birket-Smith, 2001). There is a significant correlation between the two, and it may be related to pain level (Fishbain et al., 2009). Somatization is "the tendency to experience and report somatic symptoms due to psychological distress" (Lipowski, 1988: p. 19). It is difficult to differentiate pain and clinical somatization. Similar etiologies have been proposed to underlie both somatization and pain. Neurobiological sensitivity, central sensitization, cognitive-affective mechanisms, social learning, and familial transmission are similar proposed etiologies or contributing pathways for both CP and somatization (e.g. Adams & Turk, 2018; Asmundson et al., 2012; Stone & Wilson, 2016). The co-occurrence of CP and somatization is associated with functional impairments (e.g. Eliasen et al., 2016).

1.5. Chronic Pain, Disabilities and Executive Function Deficits

Disability is an important potential outcome of CP from a clinical and public health perspective. Studies in different countries found that chronic pain intensity was the strongest predictor of pain-related disability (e.g. Raftery et al., 2011; Sirbu et al., 2023). Additionally, several meta-analyses have consistently found that people with chronic pain suffer from mild to moderate impairments in executive function (Berryman et al., 2014; Rathbone et al., 2016). Additionally, research has shown a moderate decline in working and long-term memory performance among chronic pain patients (Mazza et al., 2018). These findings suggest a potential two-way relationship: Pain may disrupt executive functioning, while poor executive functioning could also increase susceptibility to increased pain (Bunk et al., 2019). Impairments in cognitive executive functioning can lead to inadequate regulation of stress, pain, and emotion-related information processing. Conversely, negative emotions, pain, and stress can impair executive functioning. The characteristics of the feedback and feedforward neural connections between the prefrontal cortex and the limbic system play a crucial role in determining adaptive behavior, stress response, and pain experience (Feller et al., 2020).

The current study aims to explore the relationship between CP as a traumatic continuous stressor, PTSD, depression, anxiety, pathological dissociation, somatization, disability and executive functions. The conceptual model we want to test includes CP and predicts PTSD. Depression, anxiety, pathological dissociation, somatization, and executive functions after controlling for the impact of other cumulative stressors and traumas.

1.6. Research Hypotheses

***Hypothesis* 1:** Chronic pain as continuous traumatic stressors independently predicts PTSD, depression, and anxiety. It predicts somatization and pathological dissociation.

***Hypothesis* 2:** Chronic pain as continuous traumatic stressors predicts executive function deficits.

***Hypothesis* 3:** Chronic pain as continuous traumatic stressors predicts disability.

2. Method

2.1. Participants and Procedure

We empirically test the three hypotheses using questionnaire that include in addition to basic demographics, self-report measures that access the variables in the model. The research team administered the questionnaires to patients' participants from May to August of 2023 in Egypt. The team used Google Drive and developed a survey link. Once the participant completed the survey, it was sent anonymously to Gmail and then downloaded to an Excel file. All questionnaires were administered individually to participants. 16.1% of the interviews were conducted face-to-face for those who needed help with internet access, and 83.9% online. Participation was voluntary with built-in informed consent; each patient took approximately 25 min to complete the full questionnaire. The Fayoum University Independent Review Body (IRB) approved the research protocol as part of a cross-cultural study of the effects of chronic pain as continuous traumatic stressors on mental health and cognitive functioning.

We included 11 attention-check questions to ensure the validity of the responses. Seventeen participants (approximately 7.5%) were excluded due to failing 50% or more of the attention-check questions. Consequently, 211 individuals remained in the sample. The study was carried out in Egypt. Among the participants, 75.4% were female. The age of the participants ranged from 18 to 65, with a mean of 29.71 and a standard deviation of 11.32. Table 1 details the demographics of the sample.

2.2. Measurements

***The Patient Health Questionnaire*-15 (*PHQ*-15) (*Somatization*):** The PHQ-15 includes 15 items related to common somatic complaints experienced during the past four weeks (Kroenke et al., 2002). Each item was rated three-point Likert scale

(from 0 = not bothered at all, 1 = bothered somehow, to 2—bothered a lot). The results of the studies (e.g. Zijlema et al., 2013) showed that PHQ-15 is a reliable and valid measure to evaluate somatic complaints. AlHadi et al. (2017) translated the PHQ-15 into Arabic and they reported Cronbach's alpha internal consistency score as .83. The scale has an Omega (another consistency measure) of .88 in current data.

Table 1. Characteristics of participants.

Variable	Sample distribution	Frequency	Percent (%)
Gender (n = 211)	Male	52	24.6
	Female	159	75.4
Age (n = 211)	Under 30	135	64
	31 - 50	59	28
	≥51	17	8.1
Religion (n = 211)	Muslim	195	92.4
	Christian	19	7.6
Marital status (n = 211)	Single	38	18
	Married	164	77.7
	Divorced	9	4.3
Socio-economic status (n = 211)	Very low	6	2.8
	Low	3	1.4
	Middle	130	61.1
	High	72	34.1
Work status (n = 211)	Governmental work	38	18
	Private job	52	24.6
	Student	84	39.8
	Unemployed	32	15.2
	Retired	5	2.4

***Posttraumatic Stress Disorder Checklist*-5 (*PCL*-5):** The PCL-5 is a 20-item self-report measure that assesses the severity of PTSD symptoms for the past month (Weathers et al., 2018). The PCL-5 has four dimensions corresponding to each symptom cluster in the DSM-5: avoidance, intrusions, negative alterations in cognitions and mood, and alterations in arousal and reactivity. Initial research suggests that a PCL-5 cutoff score between 31 - 33 is indicative of probable PTSD across samples. Ibrahim et al. (2018) translated the PCL-5 into Arabic and reported good internal consistency in the Arabic form. In this study, the scale has an Omega score of .96.

***Short-Form McGill Pain Questionnaire* (*SF-MPQ*):** The SF-MPQ is a self-report questionnaire developed by Melzack (1985) to assess pain experience. The scale consists of 15 items and 2 dimensions (11 items sensory and 4 items affective

dimensions). Respondents rated intensity on a 4-point Likert-type scale (0 = none, 3 = severe). Terkawi et al. (2017) translated the SF-MPQ into Arabic and they reported good internal consistency (α = .85). In this study, Omega's internal consistency scores calculated were sensory, affective dimensions and total scores as .94, .86, and .95 respectively.

***Cumulative Stress and Trauma Scale (CST-S)* (Kira et al., 2008):** The CST-S was developed to reflect the parameters of the Development-Based Trauma Framework (DBTF) (e.g. Kira, 2001, 2021, 2022). The scale was constructed to measure seven types of stressors/traumas and included three items that measure chronic and main life stressors. The seven types of stressors/traumas span collective identity traumas (e.g. discrimination and oppression) and personal identity trauma (e.g. early childhood adversities such as child abuse and neglect). They also include identity/achievement trauma (e.g. failed business, fired, and dropping out of school). They also include survival trauma (e.g. exposure to car accidents, combat, and different kinds of disasters). Also, they include attachment trauma (e.g. abandonment by parents), secondary trauma (i.e. indirect trauma impact on others), and gender discrimination.

Additionally, the intersected discrimination subscale includes five items that measure gender discrimination by parents and society, social groups-based discrimination, sexual orientation discrimination, and genocidal discrimination. Also, it measures three types of trauma according to severity: Types I, II, and III.

The CST-S assesses cumulative stressors and traumas concerning their occurrence, frequency, type, negative and positive appraisals, and age of happening. However, we used only frequency and occurrence questions in this short survey study. To answer each question on the scale, participants were asked to report their experience with each event (stressor) on a 5-point Likert-type scale (0 = never; 1 = one time, 2 = two times, 3 = three times, 4 = many times).

The CST-S includes two overall cumulative stressors and traumas' dose measures: occurrence and frequency. Investigators can calculate subscales for each of the stressor/trauma types. While most scholars consider traumatic events as independent and internal consistency analysis is not required. It is the judgment of this scale developer that α (consistency between independent events) for cumulative traumas is an index of trauma proliferation in the sample. The CST-S has shown high trauma proliferation index (α = .85), test-retest stability (.95 in 4 weeks), and convergent, discriminant, and predictive validity in different studies (see Kira, 2021 for review). The scale has been validated and translated into different languages, including Arabic, The Omega of cumulative trauma occurrence (or the index of trauma proliferation in this sample) is .95, which indicates high trauma proliferation.

***The Pain Disability Index* (Pollard, 1984):** The Pain Disability Index (PDI) is widely used for measuring disability associated with pain. Pollard defined disability due to pain as "the extent to which chronic pain interferes with a person's abil-

ity to engage in various life activities". The PDI is a seven-item, self-report inventory designed to measure both general and domain-specific disability related to chronic pain as defined by Pollard. The measure had been translated scientifically into Arabic in current study. The measure has an Omega of.92 in the current data.

***Brief Pathological Dissociation Scale* (*PDS-B*) (Kira & Shuwiekh, 2022):** The PDS-B is a nine-item scale used to screen for pathological dissociation, which includes symptoms such as amnesia, derealization, depersonalization, and identity disruption. Both exploratory and confirmatory factor analyses have shown that the scale has a one-factor structure. It has demonstrated good reliability with an Omega of .89, as well as good convergent, discriminant, criterion, and predictive validity. Additionally, it is a strong predictor of conditions such as Complex Post-Traumatic Stress Disorder (CPTSD), Post-Traumatic Stress Disorder (PTSD), and depression. The measure has an Omega of .89 in current data.

***Generalized Anxiety Disorder*-7 (*GAD*-7) (Spitzer et al., 2006):** It is a 7-item scale that measures generalized anxiety. The measure has a cut-off point of 15, translating to severe anxiety. The measure has a specificity of 82% and a sensitivity of 89%. High scores on the scale were found to be highly correlated with functional impairment in multiple domains (Spitzer et al., 2006). Its Arabic version has robust psychometrics (Sawaya et al., 2016). The scale has an Omega of .75 in current data.

***Patient Health Questionnaire*-9 (*PHQ*-9) (Kroenke et al., 2001):** The scale consists of 9 items that measure depression severity. The cut-off score spans from 15 - 19 meaning moderate to severe depression, while a score of 20 and above means severe depression. A study found that the Arabic version of the scale was robust (Sawaya et al., 2016). In the current study, the scale had an Omega of .77.

***The Adult Executive Functioning Inventory* (*ADEXI*) (Holst & Thorell, 2018):** The scale had been used to assess executive functioning deficits in adults. It is a 14-item that assesses working memory (9 items), and inhibition deficits (5 items). The measure was found to adequately discriminate between adults with ADHD and controls (Holst & Thorell, 2018). The measure was found to have adequate psychometric properties in Arabic populations (Kira et al., 2023a). In the present study, it had an Omega of .85 for the total scale, .80 for the working memory deficits, and .77 for the inhibition deficits.

Shift subscale of the BRIEF-A: The BRIEF-A is a standardized rating scale used to assess executive functions in adults aged 18 to 90. It was adapted to the Arabic culture, and its reliability, validity, and consistency were established by Al-Shuqayrat in 2015 (Al-Shuqayrat, 2015). The Shift (or cognitive flexibility) subscale of the BRIEF-A measures the ability to move freely from one situation, activity, or aspect of a problem to another as the situation demands, thinking flexibly to aid problem-solving. This subscale consists of seven items and has an Omega of .85.

2.3. Data Analysis

To determine the size of the sample required to reveal a medium effect size at

power = .80 for α = .01 for the number of variables in the study, we used criteria developed by Cohen (1992: p. 158) that indicated that sample size of 147 should be adequate for regression analysis. However, the power required for structural equation modeling is controversial (Wolf et al., 2013) and requires at least 200 observations.

We analyzed the data using IBM-SPSS 28 and AMOS 28 software. The participant was asked either to respond to the question or not to continue to the following one and withdraw from the study to ensure minimum missing data. For this reason, there were no significant missing data and the dropouts were rare. We tested the normality assumption through skewness and kurtosis scores. All variables' skewness and kurtosis scores were in acceptable ranges (<±2.0) (George & Mallery, 2010). We used the Mahalanobis distance method to examine multivariate outliers. The linearity assumption was detected through the Variance Inflation Factor (VIF), and no multicollinearity issues were detected (VIF < 5.0) (e.g. Thompson et al., 2017). We computed descriptive statistics for Pearson correlation to explore the relations between the variables. We conducted several hierarchical regression analyses to check our hypotheses that CP predicts PTSD, pathological dissociation, depression, anxiety, executive function deficits, and disability after controlling for demographics and cumulative stressors and traumas. Further, we conducted SEM (path-SEM hybrid) analyses to test the direct and indirect effects of CP on executive function and mental health variables. We reported direct, indirect, and total effects as standardized regression coefficients. We used Byrne's (2012) recommendations for the acceptable fit criteria. The criteria for good model fit were a non-significant Chi-square (χ^2), Chi-square/degrees of freedom (χ^2/d.f. > 5), Comparative Fit Index (CFI) values > 0.90, and Root-Mean-Square Error of Approximation (RMSEA) values < 0.08. We used a bootstrapping procedure with 10,000 bootstrap samples to examine the significance of direct, indirect (mediated effects), and total effects and 95% bias-corrected confidence intervals (95% CI) for each trauma. We trimmed the model by eliminating the non-significant paths, which slightly increased the model fit.

3. Results

Descriptive Results: *CP conditions*, *current treatment modalities*, *pain severity*, *somatization*, *disability*, *trauma exposure*, *dissociation*, *anxiety*, *depression and PTSD*.

87.7% of the participants were treated with drugs, 9.5% underwent surgeries as the main treatment for their pain, and 2.8% used both surgical procedures and pain medication to control their pain. 34.6% reported that their pain was tolerable, 53.1% experienced moderate pain, and 12.3% had severe and unbearable pain. Furthermore, 39.8% had been in pain for three months to less than a year, 32.7% for 1 - 5 years, 27.5% for more than five years 18.5% of the sample suffered from headache syndromes, 32.2% experienced localized pain, 16.1% report rheumatoid-related pain, and 17.5% have pain related to medical conditions. 15.6% have

multiple pain conditions. Pain severity (M = 17.37, SD = 11.03). 31% scored in the high severity of pain level (Physical pain (M = 14.27, SD = 8.73, Psychological pain, M = 3.10, SD = 2.72). Somatization (M = 15.94, SD = 6.70), 67.8% scored on the high levels of somatization. Disability (M = 12.70, SD = 14.07), about 10% scored at the high level of disability. Cumulative trauma frequency was (M = 16.42, SD = 13.98) and cumulative trauma occurrence was (M = 8.37, SD = 5.22). For anxiety (M = 8.71, SD = 5.68), 19.9% scored at or above the cut-off point of 15, which indicates severe anxiety. Depression (M = 11.40, SD = 7.32), 17.1% scored at or above the cut-off point of 20, which indicates severe depression. In pathological dissociation (M = 6.83, SD = 9.24),7.2% scored at or above the cut-off point 22, indicating severe pathological dissociation. PTSD (M = 22.89, SD = 15.36) 29.4% scored at or above the cut-off point of 31, which indicates probable PTSD diagnosis. All variables follow the normal distribution.

Correlation Results: Chronic pain has a large association with somatization, a medium to large association with disability, PTSD, pathological dissociation, depression, working memory deficits, anxiety, inhibition deficits, and a small to medium size association with cognitive flexibility deficits. Table 2 presents the zero-order correlations between these variables.

Table 2. Zero-order correlations between the main variables.

Variables	1	2	3	4	5	6	7	8	9	10
1) Chronic pain	1									
2) Somatization	.62***	1								
3) Disability	.43***	.41***	1							
4) Anxiety	.36***	.54***	.38***	1						
5) Depression	.39***	.59**	.39***	.82***	1					
6) Pathological dissociation	.39***	.50***	.36***	.49***	.63***	1				
7) PTSD	.42***	.67***	.38***	.77***	.82***	.69***	1			
8) Working memory deficits	.38***	.54***	.24***	.51***	.56***	.49***	.60***	1		
9) Inhibition deficits	.33***	.45***	.22***	.55***	.57***	.44***	.60***	.82***	1	
10) Cognitive flexibility deficits	.26***	.47***	.30***	.49***	.52***	.47***	.55***	.63***	.53***	1

***Correlation is significant at the 0.01 level (2-tailed).

Hierarchical Multiple Regression Results:

To test if chronic pain predicts PTSD, depression, anxiety, pathological dissociation, working memory, inhibition, and cognitive flexibility deficits and disability, after controlling for basic demographics and cumulative stressors and traumas, we conducted a series of hierarchical multiple regressions.

For PTSD, in the first step (entering demographics) age was a negative predictive, while unemployment was a positive predictor of PTSD. The first step explained

significant variance (R^2 = .211). In the second step adding cumulative traumas led to a significant increase in the explained variance (change in R^2 = .200; Beta = .40, p < .000). In the third step adding chronic pain led to a significant increase in variance predicting PTSD (change in R^2 = .061; Beta for chronic pain = .27, p < .000). VIF values indicated there is no multicollinearity. The model accounted for.422 of the variance in PTSD. Table 3 presents the details of these results. Similar results were found for depression (Table A1 in **Appendix**), generalized anxiety (Table A2 in **Appendix**), pathological dissociation (Table A3 in **Appendix**), working memory deficits (Table A4 in **Appendix**), inhibition deficits (Table A5 in **Appendix**), and cognitive flexibility deficits (Table A6 in **Appendix**). Chronic pain was a significant predictor of each after controlling for basic demographics and cumulative life stressors and traumas.

For disability, in the first step (entering demographics) income was a negative predictive, while gender was a positive predictor of disability. In the second step, after adding cumulative life stressors and traumas, gender lost its significance as a predictor (probably due to gender discrimination being one of the life traumatic stressors). Adding cumulative traumas led to a significant increase in explained variance (change in R^2 = .087; Beta = .30, p < .000). In the third step, adding chronic pain led to a significant increase in variance predicting disability (change in R^2 = .124; Beta for the effects of chronic pain = .38, p < .000); Beta for the effects of cumulative trauma = .21, p < .000). The effects of chronic pain on disability were much higher than cumulative stressors and traumas. VIF values indicated there is no multicollinearity. The model accounted for.276 of the variance in disability. Table 4 presents the details of these results.

Table 3. Hierarchical multiple regression for the effects of chronic pain on PTSD after controlling for basic demographics and cumulative stressors and traumas.

Dependent variable	Posttraumatic Stress Disorder (PTSD)											
Predictor	Step 1				Step 2				Step 3			
	B (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*
Gender	5.78 (2.95)	1.96	.16+	1.79	3.14 (2.58)	1.22	.09	1.82	2.61 (2.45)	1.07	.07	1.82
Age	−.49 (.12)	−3.98	−.36***	2.16	−.43 (.11)	−4.02	−.32***	2.17	−.43 (.10)	−4.24	−.32***	2.17
work	2.78 (.76)	3.65	.25***	1.26	2.56 (.66)	3.88	.23***	1.26	2.02 (.64)	3.16	.18**	1.30
Education	−.57 (1.24)	−.46	−.03	1.11	−.68 (1.08)	−.63	−.04	1.11	.39 (1.05)	.37	.02	1.16
Income	.80 (2.84)	.28	.02	1.02	1.35 (2.46)	.55	.03	1.02	1.30 (2.34)	.56	.03	1.02
Cumulative trauma					1.35 (.16)	8.33	.46***	1.05	1.16 (.16)	7.33	.40***	1.12
Chronic pain									.37 (.08)	4.83	.27***	1.17
R^2	.211				.411				.472			
R^2 change	.211				.200				.061			
F for R^2 change	10.948***				69.363***				23.366***			

Note: *p < .05, **p < .01, ***p < .001 (two-tailed).

SEM Results: The model fitted well (Chi-square = 23.108, d.f. = 16, p = .111, CFI = .993, RMSEA = .046). The observed variables loaded significantly on their latent variables (over .70). *Chronic pain* had direct medium-size effects on executive function deficits (as predicted by working memory deficits, inhibition deficits, and cognitive flexibility deficits), and direct and indirect effects on mental health (as predicted by PTSD, depression, anxiety) and disability. Its total effects on them are medium to high. Its direct effects on mental health accounted for 45% of its total effects. Its direct effects on disability accounted for 70% of its total effects.

Executive function deficits had direct large-size effects on mental health and indirect low to medium-size effects on disability. Mental ill-health had medium-sized direct effects on disability. Table 5 presents the direct, indirect, and total standardized effects and their 95% confidence intervals of each variable in the model. Figure 1 presents the direct standardized paths between variables.

Table 4. Hierarchical multiple regression for the effects of chronic pain on disability after controlling for basic demographics and cumulative stressors and traumas.

Dependent variable	Disability											
Predictor	Step 1				Step 2				Step 3			
	B (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*
Gender	6.05 (2.94)	2.06	.19*	1.79	4.46 (2.83)	1.58	.14	1.82	3.76 (2.63)	1.43	.12	1.82
Age	−.03 (.12)	−.27	−.03	2.16	.00 (.12)	.02	.00	2.17	.00 (.11)	.02	.00	2.17
Work	1.33 (.76)	1.75	.13	1.26	1.20 (.73)	1.65	.12	1.26	.49 (.68)	.71	.05	1.30
Education	.88 (1.24)	.713	.05	1.11	.82 (1.18)	.70	.05	1.11	2.21 (1.12)	1.92	.12	1.16
Income	−6.34 (2.83)	−2.24	−.15*	1.02	−6.01 (2.70)	−2.22	−.15*	1.02	−6.07 (2.51)	−2.42	−.15*	1.02
Cumulative trauma					.82 (.18)	4.58	.30***	1.05	.57 (.17)	3.36	.21***	1.12
Chronic pain									.48 (.08)	5.89	.38***	1.17
R^2	.065				.152				.276			
R^2 change	.065				.087				.124			
F for R^2 change	2.852***				20.997***				34.642***			

Note: *p < .05, **p < .01, ***p < .001 (two-tailed).

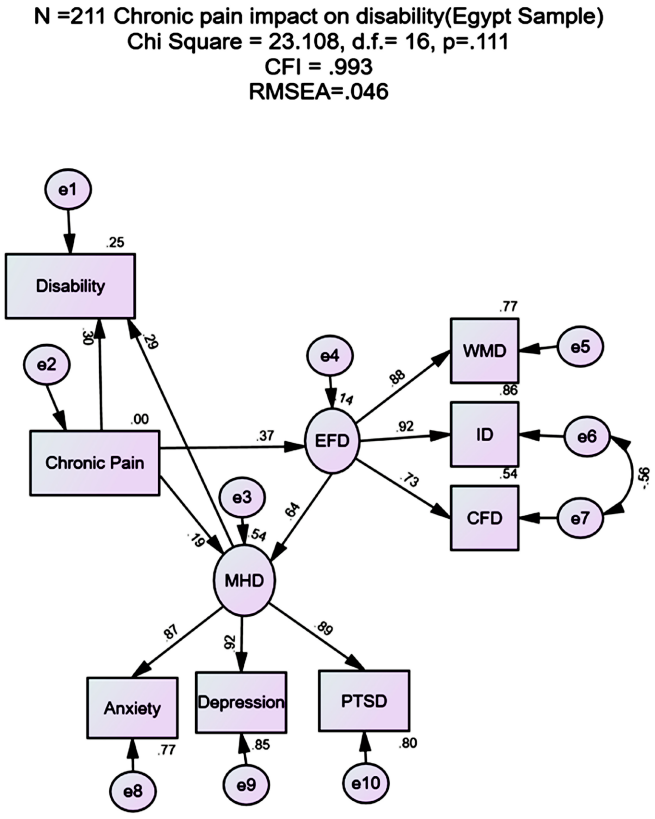
Table 5. The direct, indirect, and total standardized effects and their 95% confidence intervals of each variable in the model.

Causal variables	Endogenous variables								
	EFD	MHD	Anxiety	Depression	PTSD	Cognitive flexibility	Inhibition deficits	WMD	Disability
	Chronic pain								
Direct effects	37** (.24/.51)	.19** (.09/.31)	——	——	——	——	——	——	30** (.15/.39)
Indirect effects	——	.23** (.16/.35)	.38** (.26/.49)	.40** (.28/.53)	.39** (.27/.51)	.27** (.18/.38)	.35** (.23/.47)	.33** (.21/.47)	.13*** (.06/20)
Total effects	.37** (.24/.51)	.42** (.40/.56)	.38** (.26/.49)	.40** (.28/.53)	.39** (.27/.51)	.27** (.18/.38)	.35** (.23/.47)	.33** (.21/.47)	.43** (.29/.56)

Continued

Executive function deficits									
Direct effects	———	.64** (.46/.76)	———	———	———	.74** (.64/.83)	.93*** (.87/.97)	.88* (.81.92)/	———
Indirect effects	———	———	.56** (.51/.66)	.59** (.51/.69)	.57** (.46/.67)	———	———	———	.19** (.10/.29)
Total effects	———	.64** (.46/.76)	.56** (.51/.66)	.59** (.51/.69)	.57** (.46/.67)	.74** (.64/.83)	.93*** (.87/.97)	.88* (.81.92)/	.19** (.10/.29)
Mental health disorders									
Direct effects	———	———	.88** (.82/.92)	.92** (.88/.96)	.89* (.83/.92)	———	———	———	.29** (.15/.46)
Indirect effects	———	———	———	———	———	———	———	———	———
Total effects	———	———	.88** (.82/.92)	.92** (.88/.96)	.89* (.83/.92)	———	———	———	.29** (.15/.46)
Squared R	.140	.540	.765	.848	.800	.540	.856	.773	.254

Note: *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed). Notes: EFD = Executive Function Deficits, MHD = Mental Health Disorders, WMD = Working Memory Deficits, ID = Inhibition Deficits, CFD = Cognitive Flexibility Deficits, PTSD = Posttraumatic Stress Disorder.



Notes: EFD = executive Function Deficits, MHD = Mental Health Disorders, WMD = Working Memory Deficits, ID = Inhibition Deficits, CFD = Cognitive Flexibility Deficits, PTSD = Posttraumatic Stress Disorder.

Figure 1. The standardized direct effects of chronic pain on disability executive function deficits and mental ill health.

4. Conclusion and Discussion

The study validated chronic pain continuous traumatic stress model. CP independently predicted PTSD, pathological dissociation, depression, anxiety, somatization, disability and executive function deficits after controlling for demographics and all cumulative stressors and traumas and explained independent and significant variance in each. The study results shift the paradigm in our understanding of chronic pain and its dynamics as traumatic continuous stressors on their own that predict PTSD and other variables that may contribute to overdose epidemic.

Research suggests that chronic stress, such as in the case of Chronic Pain (CP), includes both acute and non-acute pain and can be just as stressful, or even more so, than a single acute stressor (trauma, e.g. Gold et al., 2005; Katz et al., 1981). The recurrence of chronic stressors, in this case, chronic pain, can have a cumulative impact. The prolonged sequence of repeated acute and non-acute pain can, over time, result in more complex post-trauma or complex peri-trauma symptoms. Exposure to continuous stressors may deplete a person's resources, reducing their stress tolerance and causing severe traumatic reactions with adverse consequences (e.g. Monroe & Simons, 1991; Kira et al., 2020).

The study has important conceptual and clinical implications. Conceptually, the results proposed the existence of post or "peri chronic pain syndrome" that includes somatization, pathological dissociation, disability, PTSD, depression, generalized anxiety and executive function deficits. Such "peri-chronic pain syndrome" can lead to serious behavioral consequences such as suicide and overdose epidemic. Future research can explore further the existence and the structure of this syndrome and the differential impact of its components on behavior.

Clinically, research suggests new methods to address chronic pain and overdose through continuous trauma-focused interventions (Kira, 2013; Kira et al., 2015a, 2015b; Murray et al., 2013). Research found that treating PTSD in responsive active-duty veterans who suffer chronic pain using cognitive processing therapy, significantly reduced pain at the same time (Hass et al., 2025). It is important to treat chronic pain as traumatic stress using current evidence-based interventions. Additionally, there is a need for new approaches to tailor current evidence-based interventions to better address the ongoing nature of chronic pain, such as traumatic stress. Urgently, we need to develop new, effective, evidence-based prevention and intervention strategies to prevent the proliferation and accumulation dynamics of chronic pain from reaching intolerable levels. Importantly, it is essential to develop strategies to prevent overdose and suicide resulting from intolerable CP.

The current dominant treatment approach for chronic pain is pharmacological treatment using opioid-based analgesics. Opioids pose a high risk of addiction and over-prescription. They contributed to the current opioid epidemic (Nelson et al., 2015; Rose, 2018)—the psych-social treatment approaches to treating chronic pain, as continuous traumatic stressors include individual and group psychotherapy. The study results call upon policymakers and clinicians to treat CP as they would any psychological trauma, focusing on mitigating its effects through psychotherapeu-

tic interventions rather than through only relying on pharmacological treatments that focus on its physiological etiology. Addressing CP as a traumatic phenomenon may prevent medication addiction, deaths due to overdose, ultimately improving functioning, reducing suffering, suicide and overdose. Meta-analysis of chronic pain treatment (Alldredge et al., 2024) found that group-based treatment produces significantly better outcomes than individual treatment. Group cognitive-behavioral therapy, group acceptance, and commitment therapy, self-management groups, and mindfulness-based stress reduction have shown good evidence. In contrast, group hypnotherapy and group music therapy were promising treatment modalities. There is a need for significant innovations in chronic pain treatment.

The current study has both strengths and limitations. SEM results cannot support causal claims without longitudinal data. The study uses cross-sectional data but implies that CP causes PCTSS symptoms. Given that depression/anxiety could exacerbate pain perception, you cannot rule out reverse causality or bidirectional relationships. Although the study used a random sample of chronic pain patients, it was mostly a convenience sample. The measures used in the study were self-reports, which can be biased by social desirability. For example, the Executive Function (EF) self-report may not represent the same cognitive structures as performance scales. Previous research has shown that self-report and performance scales of EF are related but distinct constructs of cognitive control (Friedman & Gustavson, 2022). Future studies may consider using both performance and self-report measures.

Moreover, the study utilized a cross-sectional design, and future research may benefit from adopting a longitudinal approach. It's essential to emphasize that in Structural Equation Modeling (SEM), direct and indirect effects are expressed as statistical probabilistic stochastic terms, which should not be conflated with causality in deterministic sciences. Additionally, we discussed in detail addiction without measuring it as a variable in the model. Notwithstanding these limitations, the study presented initial empirical evidence supporting the validity of the model of chronic pain as continuous traumatic stress and its severe consequences. Given the study's relatively small size and substantial conceptual and clinical implications, there is a clear need for broader cross-cultural studies and future replications.

5. Highlights

- The study findings provide compelling evidence that chronic pain is an independent traumatic stressor that yields mental health and cognitive syndrome components that include PTSD and are not just correlated with it.
- Chronic pain can lead independently to post- or peri-trauma mental health conditions such as PTSD, depression, dissociation, anxiety, and cognitive deficits.
- CP is continuous and severe in its mental health and cognitive impact. These

findings have the potential to significantly impact our understanding and add a continuous-trauma-focused treatment of chronic pain.

- By prioritizing the treatment of chronic pain as a distinct form of chronic traumatic stress, along with its mental health and cognitive consequences and adopting holistic approach to chronic pain treatment, rather than solely viewing it as a medical condition, we may be able to help mitigate the drug overdose and related suicide epidemic.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethical Approval

All procedures performed in studies involving human participants followed the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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Informed Consent

Informed consent was obtained from all individual participants included in the study.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Appendix

Table A1. Hierarchical multiple regression for the effects of chronic pain on depression after controlling for basic demographics and cumulative stressors and traumas.

Dependent variable	Depression											
Predictor	Step 1				Step 2				Step 3			
	B (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*
Gender	2.52 (1.44)	1.75	.15	1.79	1.33 (1.29)	1.04	.08	1.82	1.11 (1.24)	.89	.07	1.82
Age	−.21 (.06)	−3.42	−.32***	2.16	−.18 (.05)	−3.34	−.28***	2.17	−.18 (.05)	−3.47	−.28***	2.17
Work	1.27 (.37)	3.42	.24***	1.26	1.17 (.33)	3.55	.23***	1.26	.94 (.32)	2.91	.18**	1.30
Education	−.31 (.61)	−.51	−.03	1.11	−.35 (.54)	−.66	−.04	1.11	.10 (.53)	.19	.01	1.16
Income	−1.16 (1.39)	−.89	−.05	1.02	−.91 (1.23)	−.74	−.04	1.02	−.93 (1.18)	−.79	−.04	1.02
Cumulative trauma					.61 (.08)	7.54	.44***	1.05	.53 (.08)	6.59	.38***	1.12
Chronic pain									.16 (.04)	4.09	.24***	1.17
R^2		.173				.353				.402		
R^2 change		.173				.180				.049		
F for R^2 change		8.552***				56.918***				16.096***		

Note: *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed).

Table A2. Hierarchical multiple regression for the effects of chronic pain on anxiety after controlling for basic demographics and cumulative stressors and traumas.

Dependent variable	Anxiety											
Predictor	Step 1				Step 2				Step 3			
	B (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*
Gender	2.79 (1.13)	2.48	.21*	1.79	2.00 (1.04)	1.91	.15	1.82	1.83 (1.01)	1.80	.14	1.82
Age	−.12 (.05)	−2.46	−.23**	2.16	−.10 (.04)	−2.25	−.20*	2.17	−.10 (.04)	−2.32	−.20*	2.17
Work	.97 (.29)	3.34	.24***	1.26	.91 (.27)	3.39	.22***	1.26	.73 (.26)	2.78	.18**	1.30
Education	−.16 (.47)	−.33	−.02	1.11	−.19 (.44)	−.43	−.027	1.11	.15 (.43)	.34	.02	1.16
Income	.10 (1.08)	.92	.06	1.02	1.16 (1.00)	1.17	.069	1.02	1.15 (.97)	1.19	.07	1.02
Cumulative trauma					.41 (.07)	6.23	.38***	1.05	.35 (.07)	5.33	.32***	1.12
Chronic pain									.12 (.03)	3.67	.23***	1.17
R^2		.160				.294				.338		
R^2 change		.160				.134				.044		
F for R^2 change		7.786***				38.809***				13.478***		

Note: *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed).

Table A3. Hierarchical multiple regression for the effects of chronic pain on pathological dissociation after controlling for basic demographics and cumulative stressors and traumas.

Dependent variable	Pathological dissociation											
Predictor	Step 1				Step 2				Step 3			
	B (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*
Gender	−.09 (1.89)	−.05	−.00	1.78	−1.74 (1.65)	−1.06	−.08	1.81	−2.06 (1.58)	−1.30	−.10	1.81

Continued

Age	−.22 (.08)	−2.83	−.28**	2.14	−.19 (.07)	−2.72	−.23**	2.15	−.19 (.07)	−2.84	−.23**	2.15
Work	.22 (.49)	.45	.03	1.25	.07 (.42)	.17	.01	1.25	−.24 (.41)	−.59	−.04	1.29
Education	−1.48 (.80)	−1.85	−.13	1.11	−1.55 (.69)	−2.25	−.14*	1.11	−.92 (.68)	−1.36	−.08	1.16
Income	−1.65 (1.85)	−.89	−.06	1.02	−1.42 (1.65)	−.89	−.05	1.02	−1.49 (1.53)	−.97	−.05	1.02
Cumulative trauma					.87 (.07)	8.29	.49***	1.05	.75 (.10)	7.30	.43***	1.12
Chronic pain									.22 (.05)	4.38	.26***	1.170
R^2		.107				.335				.393		
R^2 change		.107				.228				.058		
F for R^2 change		4.855***				68.777***				19.186***		

Note: *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed).

Table A4. Hierarchical multiple regression for the effects of chronic pain on working memory deficits after controlling for basic demographics and cumulative stressors and traumas.

Dependent variable	Working memory											
Predictor	Step 1				Step 2				Step 3			
	B (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*
Gender	1.57 (1.43)	1.10	.10	1.79	.63 (1.34)	.47	.04	1.82	.38 (1.29)	.30	.02	1.82
Age	−.14 (.06)	−2.32	−.22*	2.16	−.12 (.06)	−2.10	−.19*	2.17	−.12 (.05)	−2.19	−.19*	2.17
Work	.44 (.37)	1.18	.09	1.26	.36 (.35)	1.04	.07	1.26	.10 (.34)	.31	.02	1.30
Education	−1.21 (.60)	−2.00	−.14*	1.11	−1.24 (.56)	−2.21	−.14*	1.11	−.75 (.55)	−1.36	−.09	1.16
Income	.61 (1.38)	.44	.03	1.02	.81 (1.28)	.63	.04	1.02	.79 (1.23)	.64	.04	1.02
Cumulative trauma					.48 (.09)	5.73	.36***	1.05	.40 (.08)	4.74	.29***	1.12
Chronic pain									.17 (.04)	4.26	.27***	1.17
R^2		.118				241				.303		
R^2 change		.118				.122				.062		
F for R^2 change		5.509***				32.810***				18.168***		

Note: *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed).

Table A5. Hierarchical multiple regression for the effects of chronic pain on inhibition deficits after controlling for basic demographics and cumulative stressors and traumas.

Dependent variable	Inhibition deficits											
Predictor	Step 1				Step 2				Step 3			
	B (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*
Gender	1.02 (.87)	1.17	.10	1.79	.50 (.83)	.60	.05	1.82	.37 (.81)	.46	.04	1.82
Age	−.09 (.04)	−2.51	−.24*	2.16	−.08 (.03)	−2.31	−.21*	2.17	−.08 (.03)	−2.37	−.21*	2.17
Work	.42 (.22)	1.86	.14	1.26	.37 (.21)	1.77	.12	1.26	.25 (.21)	1.18	.08	1.30
Education	−.52 (.37)	−1.41	−.10	1.11	−.54 (.35)	−1.55	−.10	1.11	−.29 (.34)	−.84	−.06	1.16
Income	.35 (.84)	.42	.03	1.02	.46 (.79)	.58	.04	1.02	.45 (.77)	.58	.04	1.02

Continued

Cumulative Trauma		.27 (.05)	5.15	.33***	1.05	.22 (.05)	4.29	.27***	1.12
Chronic Pain						.09 (.03)	3.41	.22***	1.17
R^2	114	.216				.258			
R^2 change	114	112				.042			
F for R^2 change	5.255***	26.520***				11.621***			

Note: *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed).

Table A6. Hierarchical multiple regression for the effects of chronic pain on cognitive flexibility after controlling for basic demographics and cumulative stressors and traumas.

Dependent variable	Cognitive flexibility											
Predictor	Step 1				Step 2				Step 3			
	B (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*	*B* (*SE*)	*t*	β	*VIF*
Gender	−.09 (.71)	−.12	−.01	1.79	−.42 (.69)	−.61	−.05	1.82	−.50 (.69)	−.73	−.06	1.82
Age	−.11 (.03)	−3.78	−.36***	2.16	−.11 (.03)	−3.64	−.34***	2.17	−.11 (.03)	−3.68	−.34***	2.17
Work	.44 (.18)	2.38	.17*	1.26	.41 (.18)	2.30	.16*	1.26	.33 (.18)	1.86	.13	1.30
Education	−.37 (.30)	−1.22	−.08	1.11	−.38 (.29)	−1.31	−.09	1.11	−.23 (.29)	−.79	−.05	1.16
Income	.20 (.68)	.29	.02	1.02	.27 (.66)	.40	.03	1.02	.26 (.65)	.40	.03	1.02
Cumulative trauma					.17 (.04)	3.95	.27***	1.05	.15 (.04)	3.29	.22***	1.112
Chronic pain									.05 (.01)	2.40	.16*	1.17
R^2	.126				.128				.210			
R^2 change	.126				.026				.023			
F for R^2 change	5.898***				15.616***				5.897**			

Note: *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed).