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Psychosocial mechanisms underlying the transition from acute to chronic postsurgical pain in youth following spinal fusion: a 6-month longitudinal study

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Abstract

Theoretical and empirical work underscores the role of postsurgical acute pain severity and psychosocial factors in the development of chronic postsurgical pain (CPSP). However, evidence on how psychosocial changes in response to acute pain influence CPSP development in adolescents is limited. In this 6-month longitudinal study, adolescents undergoing spinal fusion were assessed presurgery, monitored for 30 days postsurgery, and re-evaluated at 8 weeks and 6 months. We examined changes in adolescent psychosocial factors (depression, anxiety, and pain catastrophizing) and parental distress from presurgery to 8 weeks postsurgery and tested their mediating effects between postsurgical acute pain intensity and interference, and CPSP development at 6 months. Among 160 adolescents included (10-18 years [M = 14.6, SD = 2.1]; 77% female; 17% Hispanic), 34% developed CPSP (pain intensity ≥3 and quality of life impairment) at 6 months. Adolescents who developed CPSP had higher pain intensity, psychological distress, and parental distress presurgery and 8 weeks postsurgery. Longitudinal causal mediation analyses controlling for sex and presurgery pain and psychosocial factors revealed that changes in adolescent anxiety and pain catastrophizing from presurgery to 8 weeks postsurgery mediated the link between postsurgical acute pain intensity and CPSP, explaining 13.8% and 11.0% of the effect, respectively. In addition, changes in adolescent pain catastrophizing mediated the association between acute pain interference and CPSP, explaining 19.6% of the effect. Significant mediation effects were not observed for changes in adolescent depression or parental distress. Anxiety symptoms and pain catastrophizing are actionable targets both before and after surgery to reduce CPSP development in adolescence.

Keywords: Adolescent, Chronic postsurgical pain, Postsurgical acute pain, Psychosocial mechanisms, Pain catastrophizing

1. Introduction

Pediatric chronic postsurgical pain (CPSP) occurs commonly after invasive pediatric surgeries,[39,66] with 31% of adolescents undergoing spine surgery experiencing persistent pain.[49] CPSP is associated with significant negative consequences on postsurgery outcomes,[12] including functional disability, decreased quality of life,[38] and significant medical expenditures.[13] The strongest risk factor for pediatric CPSP is acute postsurgical pain, with recent research demonstrating that significant acute postsurgical pain measured over several days at home is associated with increased likelihood of persistent postsurgical pain (50.0% vs 9.1% in those with vs without significant acute postsurgical pain).[8,34,38,42,47] Individual psychosocial risk factors in adolescents, including anxiety and depressive symptoms, pain catastrophizing, and family risk factors (e.g., parent distress[33,39,48]), are associated with both higher acute pain and risk of transition to CPSP. These risk factors may be

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intervention targets to mitigate the likelihood and impact of CPSP. However, to date, research has focused predominantly on presurgery psychosocial risk factors and has not examined how postsurgery psychosocial responses relate to the experience of acute pain and development of CPSP in adolescents.

The conceptual model of biopsychosocial mechanisms of acute to chronic postsurgical pain in children and adolescents highlights the specific role of psychosocial factors (e.g., emotions, cognitions, behaviors, and parent/family) in the development of CPSP.[41] The postoperative phase is a key period where some adolescents show marked recovery and others do not. Among the adolescents who struggle to recover, higher levels of acute pain and interference during the critical weeks after surgery may lead to alterations in distress levels and coping, which may in turn be related to the subsequent development of CPSP.[41] In addition, psychosocial risk factors in parents, such as parental distress and their psychological responses to their child's pain,[21,22] may affect their child's pain perception and pain-related functional disability through parent-–child interactions[28,64] and predispose the child to develop CPSP.[33,38] Understanding both individual and family-level psychosocial factors during postoperative recovery, particularly how these factors evolve in response to acute postsurgical pain, has the potential to identify novel avenues for assessment and intervention. Although opportunity for preoperative intervention exists for youth undergoing some elective surgeries, in many cases, postoperative intervention may be more feasible, with the goal of addressing psychosocial responses after surgery to mitigate the risk of transition to chronic pain.

This study examined changes in depression, anxiety, pain catastrophizing, and parental distress from presurgery to postsurgery, and determined whether these changes mediated the relationship between pain intensity and pain interference in the first 30 days after surgery and CPSP 6 months later. We hypothesized that higher pain intensity and pain interference during the 30 days after surgery (i.e., the acute period) would be associated with more adverse changes in psychosocial factors from presurgery to 8 weeks postsurgery, which, in turn, would be associated with a higher rate of developing CPSP at 6 months postsurgery, over and above the contribution of preoperative psychosocial risk (**Fig. 1** for study design).

2. Methods

2.1. Study design

This was a prospective longitudinal cohort study assessing adolescents undergoing spinal fusion, with data collection occurring at four time points over 6 months. Primary data were collected at (T1) presurgery assessment, (T2) first 30 days postsurgery, (T3) 8 weeks postsurgery assessment, and (T4) 6-month follow-up. A total of 160 adolescents were assessed at baseline (T1). Acute postsurgical pain was assessed at (T2), beginning on day 1 after hospital discharge and continuing until 30 days post-hospital discharge. CPSP status was ascertained at 6-months postsurgery (T4). Changes in psychosocial constructs were determined from presurgery (T1) to 8 weeks postsurgery (T3). This is the first outcome report resulting from this study.

2.2. Study procedures

Adolescents 10 to 18 years of age scheduled for elective spinal fusion surgery at two academic pediatric hospitals in Washington and California, and one of their caregivers (referred to as youth–parent dyads) were recruited for participation. Eligible surgery indications included adolescent idiopathic scoliosis, juvenile scoliosis, spondylolisthesis, and kyphosis. Adolescents were excluded if they (1) were currently receiving treatment for chronic pain; (2) had severe systemic disease, (e.g., neuromuscular scoliosis or cancer) requiring daily medication or limiting physical function; (3) had a diagnosed unstable psychiatric condition (e.g., psychiatric admission in past 30 days); (4) had prior major surgery (e.g., prior spine or cardiac surgery); (5) were unable to complete study measures (e.g., learning disability or developmental delay); (6) had no access to the internet; or (7) the parent and/or adolescent could not read or understand English or Spanish.

Potentially eligible participants were identified from medical record surgery reports, screened for eligibility through medical chart review, and sent recruitment materials along with consent/assent forms approximately 2 months before surgery. Potentially eligible participants were contacted through phone, confirmed eligibility criteria, and completed informed consent. Participants who were nonresponsive to phone contact attempts were met at the surgery clinic during a presurgery visit. Youth–parent dyads who agreed to participate in the study completed presurgery (T1, between 8 and 0 weeks before surgery) self-report measures. Adolescent participants completed daily diary assessments of pain intensity and pain interference for 30 days after surgery (T2). At the 8-week (T3) and 6-month (T4) follow-up assessments, both adolescents and parents completed self-report surveys. Adolescents and parents earned gift cards on completion of assessment at each time point. Adolescents and parents earned up to $420 and $150 in gift cards for study participation, respectively. The Seattle Children's Institutional Review Board (Federal Wide Assurance Number FWA00002443) at Seattle Children's Hospital (Study IRB ID: STUDY00001241) and the Children's Hospital Orange County Institutional Review Board #1 - In-House Board (Federal Wide Assurance Number FWA00000255) at Children's Hospital Orange County (Study IRB ID: 181089) approved the study.

Patients received standard surgical, anesthetic, and analgesic care, which was not altered for this study. Typical intraoperative management included a total intravenous anesthetic to facilitate

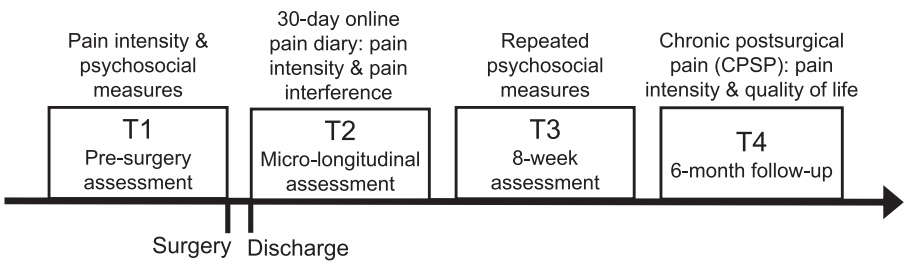

Figure 1. Study design and measurements.

neuromonitoring during surgery and analgesics including ketamine, opioids, and methadone. Postoperative management included expedited nutrition intake, physical therapy, and early ambulation, and multimodal pain management including scheduled nonopioid analgesics (acetaminophen, nonsteroidal anti-inflammatory drugs), scheduled or as needed muscle relaxants, and patient controlled opioid analgesia for 24 hours followed by enteral opioid. Patients were typically discharged on day 2 to 4 with scheduled nonopioid analgesia and a limited course of as needed oxycodone and muscle relaxant. In this study, the mean interval between surgery and hospital discharge was 3.3 days (SD = 0.9, median = 3).

2.3. Measures

2.3.1. Baseline demographic and pain measures

At baseline, parents completed a brief questionnaire self-reporting on demographics including sex, gender, race, ethnicity, parent marital status, parent employment status, and household income. Adolescents self-reported on sex and gender. Baseline age (in years) was calculated using the adolescent's date of birth from the medical record and the date they completed the T1 survey assessment. In addition, adolescents reported on the intensity of their pain during the past 7 days using a 0 to 10 numerical rating scale (0-10 NRS, ranging from "no pain" to "worst pain possible").

2.3.2. Acute postsurgical pain assessment

Beginning on day 1 after hospital discharge, youth completed daily diaries in the evening for a 30-day period, using the Brief Pain Inventory Short Form (BPI-SF) to report on pain intensity and pain interference,[7] consistent with our prior studies.[40] The measure consists of four items assessing pain intensity (worst, least, and average pain in the preceding 24 hours, and current pain) and seven items assessing pain interference on sleep, mood, walking ability, general physical activity, work/school, relationships, and enjoyment of life, each rated on an 11-point numeric rating scale (0-10 NRS). The BPI-SF was originally developed and tested in adult,[7,25] where it demonstrated sensitivity to change.[55] It has been extensively used in adolescents[26,57] and has demonstrated construct validity to assess pain and function in adolescents.[14] This microlongitudinal assessment of acute pain intensity and interference can more reliably quantify the experience of acute pain during the postsurgical recovery period, where dynamic changes are expected. For our analysis, mean daily pain intensity was calculated as the average of worst, least, average, and current pain intensity ratings each day. This daily mean score was then averaged over a 30-day period to represent the overall pain intensity (ranging between 0 and 10) during the acute, postsurgical phase. Similarly, the mean daily pain interference score was calculated by averaging responses to the seven items measuring pain interference. The 30-day mean of this score was used to indicate the overall level of pain interference (ranging between 0 and 10) during the acute, postsurgical phase.

2.3.3. Chronic postsurgical pain assessment

At the 6-month postsurgery assessment, adolescents reported on pain intensity during the past 7 days using a 0 to 10 NRS and completed the Pediatric Quality of Life Inventory (PedsQL). We determined CPSP status at 6 months after surgery using the criteria of a pain intensity score of 3 or higher (NRS $\geq$3) and a quality of life score below 1 SD from the population norm (PedsQL score $<$74.9), as outlined in our previous publication[40,42,49] and consistent with contemporary CPSP definition.[49,65]

2.3.4. Psychosocial measures

Psychosocial factors including adolescent depressive symptoms, anxiety symptoms, pain catastrophizing, as well as parent distress were measured before surgery (T1) and at 8 weeks after surgery (T3).

Adolescent depressive and anxiety symptoms: Adolescents completed the 47-item Revised Child Anxiety and Depression Scale (RCADS) to assess depressive and anxiety symptoms. This measure has five scales for assessing anxiety (social phobia, panic disorder, separation anxiety, generalized anxiety, and obsessive-compulsive) and one scale for assessing depression. For this study, we calculated the T-scores for depressive symptoms and overall anxiety symptoms (based on the sum of individual anxiety subscales), respectively, with 50 being the population mean and 10 being the SD. Higher T-scores indicated higher levels of depressive or anxiety symptoms. A T-score $\geq$70 indicated clinical depression or anxiety, and a T-score $\geq$65 and below 70 indicated borderline clinical depression or anxiety. The RCADS is valid, reliable, and sensitive to change in youth[5,29,36] and has been broadly used in adolescents with pain,[6,23] including in surgical sample.[42]

Adolescent pain catastrophizing: Pain catastrophizing was assessed using the Pain Coping Questionnaire (PCQ) internalizing/catastrophizing subscale, which consists of five self-report items such as "Worry that I will always be in pain" and "Think that the pain will never stop." Each item was rated on a 5-point Likert scale ranging from 1 = "never" to 5 = "very often." The mean score of these items (ranging from 1 to 5) was used to indicate the level of pain catastrophizing, with a higher score indicating a higher level of pain catastrophizing. We used this scale to assess pain catastrophizing in alignment with recent recommendations regarding assessment of internalizing symptoms in children, including limitations of the Pain Catastrophizing Scale for Children (PCS-C).[10] The PCQ internalizing/catastrophizing subscale has been validated in youth with acute and chronic pain and shown sensitivity to change over time.[3,16,44] In this study, the Cronbach alpha for this subscale measure is 0.84, indicating adequate internal consistency reliability.

Parent distress: Parents completed the Brief Symptom Inventory-18 (BSI-18), an 18-item self-reported measure to assess psychological distress and symptom severity in the previous 7 days. Each item was rated on a 5-point Likert scale ranging from 0 ("not at all") to 4 ("extremely"). A total score ranging from 0 to 72 was calculated to indicate the overall level of psychological distress that included somatization, depressive, and anxiety symptoms. A higher total score indicated a higher level of psychological distress. The BSI-18 has been widely used, with its reliability and validity confirmed in adult populations,[9,67] and it has been demonstrated to be sensitive to change over time.[18] We focused on general parent distress rather than specific factors such as parent pain catastrophizing because evidence linking parent pain catastrophizing to adolescent CPSP has been inconsistent,[39] with some larger studies finding no association when more extensive psychosocial factors (e.g., child anxiety and depressive symptoms, child pain catastrophizing) were considered.[2,42]

2.4. Analysis plan

We described the overall sample characteristics using mean (SD), median (first quartile—third quartile), and counts with frequencies. We compared presurgery characteristics of adolescents

who developed CPSP at 6 months after surgery with those who did not, using the Pearson chi-squared test and Fisher exact test for categorical variables and the Wilcoxon rank-sum test for continuous variables. For the psychosocial variables, we compared both T1 and T3 values between those with and without CPSP, as well as the change in scores from T1 to T3 by CPSP status, using the Welch two-sample *t* test.

To address our hypotheses that changes in child and parent psychosocial functioning (from T1 to T3) mediated the associations of acute pain intensity and interference (within 30 days after surgery) with CPSP (at 6 months after surgery), we used causal mediation analysis approaches. Specifically, we used continuous acute pain intensity and acute pain interference as the exposure variables, change scores in the psychosocial variables from T1 to T3 as the mediator variables, and CPSP as the binary outcome variable. We fit separate mediation models for acute pain intensity and each psychosocial mediator and replicated the models using acute pain interference as the exposure. A total of eight causal mediation models (four for acute pain intensity and four for acute pain interference) were fit.

The causal mediation analysis was conducted using the R package "CMAverse",[52] which implements six causal mediation analysis approaches including the regression-based approach,[58,61] the weighting-based approach,[61] the inverse odd-ratio weighting approach,[56] the natural effect model,[62] the marginal structural model,[60] and the g-formula approach.[45] For our analysis, we used the regression-based approach to specify a logistic regression model for the outcome variable CPSP and a linear regression model for the mediator variable of interest (e.g., change in adolescent depressive symptoms from T1 to T3). Both the mediator and outcome models adjusted for adolescent sex assigned at birth, pain intensity at baseline, and the baseline level of the mediator of interest (ie, presurgery psychosocial variable corresponding to the mediator of interest). This conservative adjustment set accounted for the influence of presurgery levels of psychosocial variables on their change over time. We used the direct counterfactual imputation estimation so that point estimate of each causal effect was obtained by imputing counterfactuals directly. The mediated effects were estimated using bootstrapping with 100 iterations and presented with 95% percentile bootstrap confidence intervals. We considered the possibility of exposure–mediator interaction; if interaction effects were nonsignificant, we proceeded with regression models without introducing exposure–mediator interaction.

For each causal mediation analysis, we presented the total effect (i.e., the effect of one unit change in acute pain intensity or pain interference on the odds of CPSP), the indirect effect (i.e., the effect of one unit change in acute pain intensity or pain interference on the odds of CPSP that was mediated through changes in a specific psychosocial variable), the direct effect (i.e., the effect of one unit change in acute pain intensity or pain interference on the odds of CPSP that was independent of changes in the psychosocial variable), and the proportion mediated (i.e., the proportion of total effect that is mediated through changes in the psychosocial variable of interest). We estimated associations using logistic regression models; therefore, the magnitude of the total, indirect, and direct effects are presented as odds ratios (ORs). Two-sided *P*-values <0.05 were deemed statistically significant.

2.5. Sample size and power considerations

Power and sample size calculations were based on precision of CPSP prevalence estimate and associations between potential mediators and CPSP status. Based on preliminary studies, we projected that 25% of the surgery sample would develop CPSP at 6 months. We estimated that a sample of N = 126 would provide a 95% CI with half-width ±7.5% around an incidence of 25%. To account for attrition, we inflated the sample by 20% to arrive at the final enrollment target of N = 160. Because the postsurgical acute recovery patterns were not defined a priori and there lacked any existing mediation effect estimates, we assessed power based on associations between potential mediators and CPSP status, the second segment of the mediational pathway. Assuming 1:3 allocation ratio of the CPSP group and non-CPSP group (25% incidence), total evaluable sample size of N = 126 would provide 80% power to detect a medium effect size Cohen $d = 0.58$ or higher.

3. Results

3.1. Sample characteristics and descriptive statistics

A total of 432 youth–parent dyads were screened, among whom 268 were excluded, including 48 (11.1%) not meeting the inclusion criteria, 170 (39.4%) declining to participate, and 50 unable to be reached (11.6%). Four youth were excluded before the presurgery assessment due to cancellation of surgery (n = 1) or noncompletion of baseline assessment (n = 3). Thus, a total of 160 youth–parent dyads were included and considered our study participants (**Fig. 2** shows the enrollment diagram). Of these 160 youth–parent dyads, CPSP status could be ascertained in 145 adolescents at 6 months (four dropped out of the study by T4, 9 did not complete T4 study data collection, and two had missing data for CPSP). Therefore, the analyses were performed in these 145 youth–parent dyads (deemed as our analytic sample). Comparison of those included in analysis with those not included revealed few differences in demographic characteristics; however, those participants not included in the analysis were more likely to have divorced parents (Supplemental Table 1, http://links.lww.com/PAIN/C281).

Characteristics of the analytic sample are summarized in **Table 1**. Adolescents were aged between 10 and 18 years at presurgery assessment, with a mean age of 14.64 years (SD = 2.06 years). Most were female (78%), White (60%), and non-Hispanic (83%). Compared with adolescents who did not develop CPSP at 6 months (n = 98 [68%]), those who developed CPSP by 6 months (n = 47 [32%]) were more likely to be female (91% vs 71%), had higher presurgery pain intensity (4.70 ± 1.90 vs 3.81 ± 1.96), and were less likely to have married parents (62% vs 82%). All other demographic characteristics were similar among the two groups.

Assessment of postsurgical acute pain indicated that adolescents who developed CPSP had consistently higher levels of daily pain intensity and pain interference during the 30 days after surgery. The average pain intensity during the 30 days was 2.23 (SD = 1.23) for the non-CPSP group, relative to 3.23 (SD = 1.35) for the CPSP group ($P < 0.001$). The average pain interference during the 30 days was 2.92 (SD = 1.57) for the non-CPSP group, relative to 3.90 (SD = 1.44) for the CPSP group ($P < 0.001$). As shown in **Figure 3**, adolescents reporting CPSP had higher pain intensity at baseline (4.70 ± 1.90 vs 3.81 ± 1.96, $P = 0.007$), 8 weeks after surgery (4.22 ± 2.03 vs 2.60 ± 2.01, $P < 0.001$), and 6 months after surgery (4.30 ± 1.44 vs 2.00 ± 1.57, $P < 0.001$).

Correlations between the psychosocial variables at presurgery and 8 weeks after surgery, as well as between the change scores in these variables from presurgery to 8 weeks after surgery are presented in **Supplemental Table 2**, http://links.lww.com/PAIN/C281. At presurgery and 8 weeks after surgery, adolescent

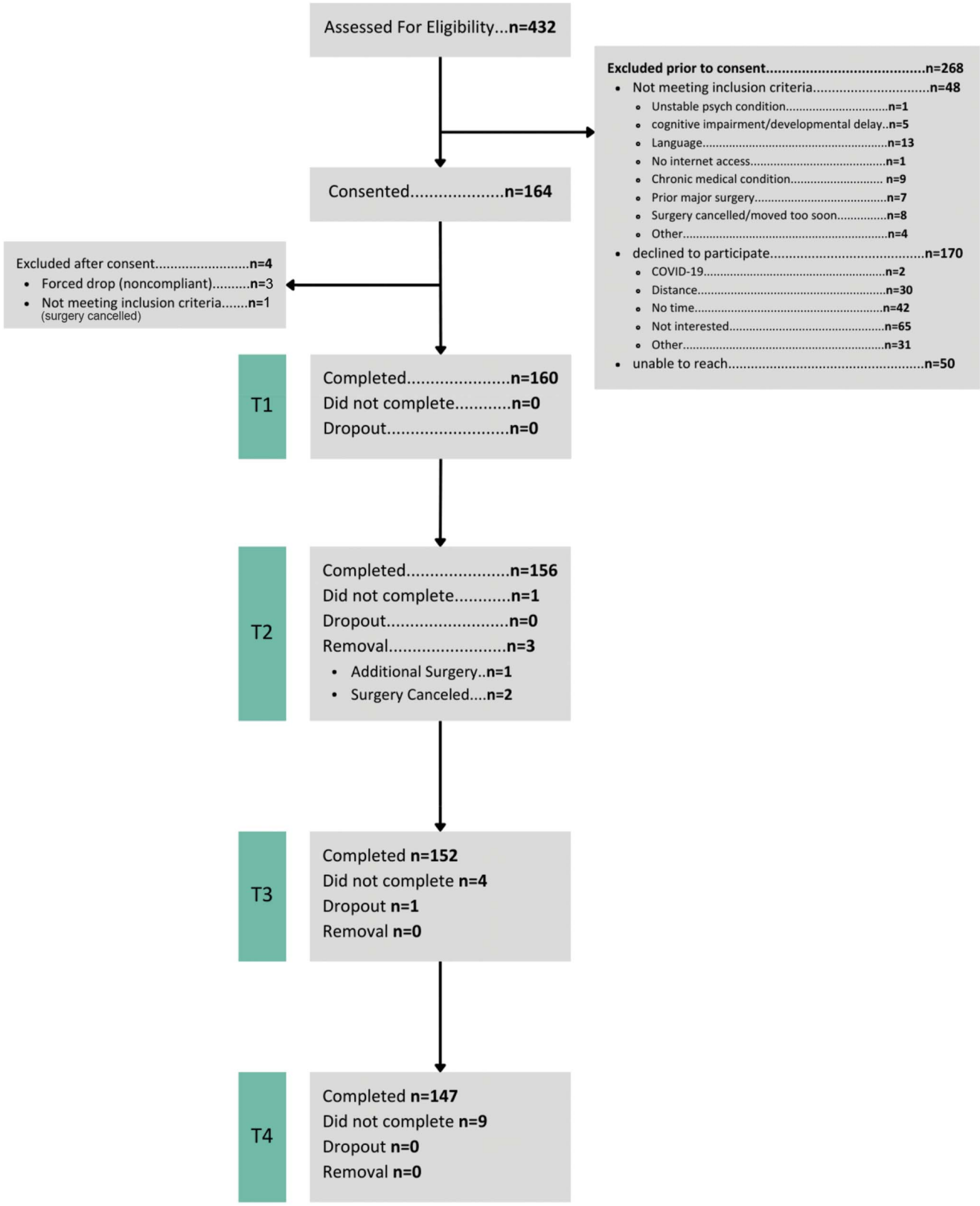

Figure 2. Flowchart of the study participants.

depressive symptoms, anxiety symptoms, and pain catastrophizing were highly correlated with each other (Pearson rs ranged between 0.50 and 0.77). Correlations between parent distress and adolescent psychosocial variables were small (Pearson rs < 0.30). Regarding changes in these psychosocial variables, changes in adolescent depressive and anxiety symptoms from presurgery to 8 weeks after surgery were highly correlated (Pearson $r = 0.62$), with moderate correlations with changes in adolescent pain catastrophizing (Pearson rs < 0.50), and small correlations with changes in parent distress (Pearson rs < 0.30).

Table 1

Baseline characteristics of the study sample, overall and by CPSP status.

Characteristics	Entire sample (N = 145)	CPSP status		P*
		Without CPSP (n = 98)	With CPSP (n = 47)	
Adolescent age: years	14.64 (2.06)	14.64 (2.15)	14.64 (1.88)	0.96
Adolescent sex				**0.006**
Male	32/145 (22%)	28/98 (29%)	4/47 (8.5%)	
Female	113/145 (78%)	70/98 (71%)	43/47 (91%)	
Adolescent gender				**0.01**
Male	32/144 (22%)	28/97 (29%)	4/47 (8.5%)	
Female	108/144 (75%)	66/97 (68%)	42/47 (89%)	
Transgender	1/144 (0.7%)	1/97 (1.0%)	0/47 (0%)	
Nonbinary	2/144 (1.4%)	1/97 (1.0%)	1/47 (2.1%)	
Unsure	1/144 (0.7%)	1/97 (1.0%)	0/47 (0%)	
(Missing)	1	1	0	
Adolescent of Hispanic ethnicity	25/144 (17%)	16/97 (16%)	9/47 (19%)	0.69
(Missing)	1	1	0	
Adolescent race				0.85
American Indian and Alaska Native	4/144 (2.8%)	3/97 (3.1%)	1/47 (2.1%)	
Black	10/144 (6.9%)	8/97 (8.2%)	2/47 (4.3%)	
Chinese	7/144 (4.9%)	4/97 (4.1%)	3/47 (6.4%)	
Japanese	3/144 (2.1%)	2/97 (2.1%)	1/47 (2.1%)	
Korean	3/144 (2.1%)	1/97 (1.0%)	2/47 (4.3%)	
Latin American	18/144 (13%)	10/97 (10%)	8/47 (17%)	
Native Hawaiian and Pacific Islander	1/144 (0.7%)	1/97 (1.0%)	0/47 (0%)	
South Asian	1/144 (0.7%)	1/97 (1.0%)	0/47 (0%)	
South East Asian	6/144 (4.2%)	5/97 (5.2%)	1/47 (2.1%)	
White	87/144 (60%)	58/97 (60%)	29/47 (62%)	
Other	3/144 (2.1%)	3/97 (3.1%)	0/47 (0%)	
Filipino	1/144 (0.7%)	1/97 (1.0%)	0/47 (0%)	
(Missing)	1	1	0	
Had previous surgery	32/139 (23%)	24/95 (25%)	8/44 (18%)	0.36
(Missing)	6	3	3	
Presurgery pain intensity	4.11 (1.98)	3.81 (1.96)	4.70 (1.90)	**0.007**
(Missing)	3	3	0	
Presurgery quality of life	69.95 (18.17)	74.23 (17.39)	60.50 (16.35)	**<0.001**
(Missing)	7	3	4	
Parent relationship to child				0.53
Mother	130/142 (92%)	88/95 (93%)	42/47 (89%)	
Father	12/142 (8.5%)	7/95 (7.4%)	5/47 (11%)	
Other	0/142 (0%)	0/95 (0%)	0/47 (0%)	
(Missing)	3	3	0	
Parent sex				0.76
Male	13/144 (9.0%)	8/97 (8.2%)	5/47 (11%)	
Female	131/144 (91%)	89/97 (92%)	42/47 (89%)	
(Missing)	1	1	0	
Parent marital status				**0.01**
Divorced	22/144 (15%)	9/97 (9.3%)	13/47 (28%)	
Married	109/144 (76%)	80/97 (82%)	29/47 (62%)	
Separated	5/144 (3.5%)	4/97 (4.1%)	1/47 (2.1%)	
Single	6/144 (4.2%)	3/97 (3.1%)	3/47 (6.4%)	
Widowed	1/144 (0.7%)	1/97 (1.0%)	0/47 (0%)	
Other	1/144 (0.7%)	0/97 (0%)	1/47 (2.1%)	
(Missing)	1	1	0	
Parent employment status				0.38
Full-time	77/144 (53%)	48/97 (49%)	29/47 (62%)	
Part-time	23/144 (16%)	17/97 (18%)	6/47 (13%)	
Not working	44/144 (31%)	32/97 (33%)	12/47 (26%)	
(Missing)	1	1	0	

(*continued on next page*)

Table 1 (*continued*)

Characteristics	Entire sample (N = 145)	CPSP status		P*
		Without CPSP (n = 98)	With CPSP (n = 47)	
Parent highest education				0.15
High school or less	30/144 (21%)	21/97 (22%)	9/47 (19%)	
Some college	35/144 (24%)	23/97 (24%)	12/47 (26%)	
College	53/144 (37%)	40/97 (41%)	13/47 (28%)	
Graduate	26/144 (18%)	13/97 (13%)	13/47 (28%)	
(Missing)	1	1	0	
Spouse employment status				0.89
Full-time	98/110 (89%)	71/80 (89%)	27/30 (90%)	
Part-time	6/110 (5.5%)	5/80 (6.3%)	1/30 (3.3%)	
Not working	6/110 (5.5%)	4/80 (5.0%)	2/30 (6.7%)	
(Missing)	35	18	17	
Spouse highest education				0.38
High school or less	28/110 (25%)	22/80 (28%)	6/30 (20%)	
Some college	26/110 (24%)	21/80 (26%)	5/30 (17%)	
College	35/110 (32%)	22/80 (28%)	13/30 (43%)	
Graduate	21/110 (19%)	15/80 (19%)	6/30 (20%)	
(Missing)	35	18	17	
Parent Hispanic ethnicity	21/144 (15%)	15/97 (15%)	6/47 (13%)	0.67
(Missing)	1	1	0	
Parent race				0.79
American Indian and Alaska Native	2/144 (1.4%)	1/97 (1.0%)	1/47 (2.1%)	
Black	4/144 (2.8%)	4/97 (4.1%)	0/47 (0%)	
Chinese	7/144 (4.9%)	5/97 (5.2%)	2/47 (4.3%)	
Japanese	4/144 (2.8%)	3/97 (3.1%)	1/47 (2.1%)	
Korean	3/144 (2.1%)	1/97 (1.0%)	2/47 (4.3%)	
Latin American	16/144 (11%)	11/97 (11%)	5/47 (11%)	
Native Hawaiian and Pacific Islander	2/144 (1.4%)	1/97 (1.0%)	1/47 (2.1%)	
South Asian	1/144 (0.7%)	1/97 (1.0%)	0/47 (0%)	
South East Asian	7/144 (4.9%)	6/97 (6.2%)	1/47 (2.1%)	
White	94/144 (65%)	60/97 (62%)	34/47 (72%)	
Other	2/144 (1.4%)	2/97 (2.1%)	0/47 (0%)	
Filipino	2/144 (1.4%)	2/97 (2.1%)	0/47 (0%)	
(Missing)	1	1	0	
Household size	4.05 (1.04)	4.14 (1.05)	3.85 (1.01)	0.12
(Missing)	2	1	1	
Household annual income				**0.02**
<$24,999	9/141 (6.4%)	7/95 (7.4%)	2/46 (4.3%)	
$25,000-$49,999	22/141 (16%)	9/95 (9.5%)	13/46 (28%)	
$50,000-$74,999	20/141 (14%)	17/95 (18%)	3/46 (6.5%)	
$75,000-$99,999	15/141 (11%)	10/95 (11%)	5/46 (11%)	
$100,000-$149,999	24/141 (17%)	16/95 (17%)	8/46 (17%)	
$150,000-$199,999	25/141 (18%)	21/95 (22%)	4/46 (8.7%)	
>=$200,000	26/141 (18%)	15/95 (16%)	11/46 (24%)	
(Missing)	4	3	1	

Bolded values are significant at $P < 0.05$.
* Pearson chi-squared test; Wilcoxon rank-sum test; Fisher exact test.

Table 2 summarizes T scores/mean scores on adolescent and parent psychosocial variables at baseline and 8 weeks after surgery, as well as the changes in these psychosocial variables from baseline to 8 weeks after surgery, by the CPSP status. Adolescent depressive symptoms, anxiety symptoms, pain catastrophizing, and parent distress were consistently higher at both baseline and 8 weeks after surgery in the CPSP group compared with the non-CPSP group (*P*s < 0.05). The prevalence of clinical or elevated depression and anxiety was higher in the CPSP group compared with the non-CPSP group at both time points. Specifically, for the CPSP group, 19% were classified as having clinical depression and 13% with elevated depression at presurgery, compared with 7.2% and 1.0% for the non-CPSP group (*P* for group difference < 0.001), respectively. Similarly, for the CPSP group, 23% were classified as having clinical anxiety and 4.3% with elevated anxiety, compared with 7.2% and 3.1% for the non-CPSP group (P for group difference = 0.017), respectively. The same pattern was seen at 8 weeks after surgery, with 30% having clinical depression and 11% elevated depression in the CPSP group, compared with the 9.4% and 2.1% in the non-CPSP group (*P* for group difference <0.001), respectively. Clinical anxiety was seen in 17% and elevated anxiety 6.4% in the CPSP group, compared with 4.2% and 2.1% in the non-CPSP group (*P* for group difference = 0.011), respectively. Changes in adolescent depressive symptoms from baseline to 8 weeks after surgery differed among the CPSP and non-CPSP groups (difference in *T*-score = 4.73, 95% CI 1.08-8.38, $P = 0.005$); adolescent depressive symptoms increased from baseline to 8 weeks after surgery in the CPSP group, but decreased in the non-CPSP group. Although

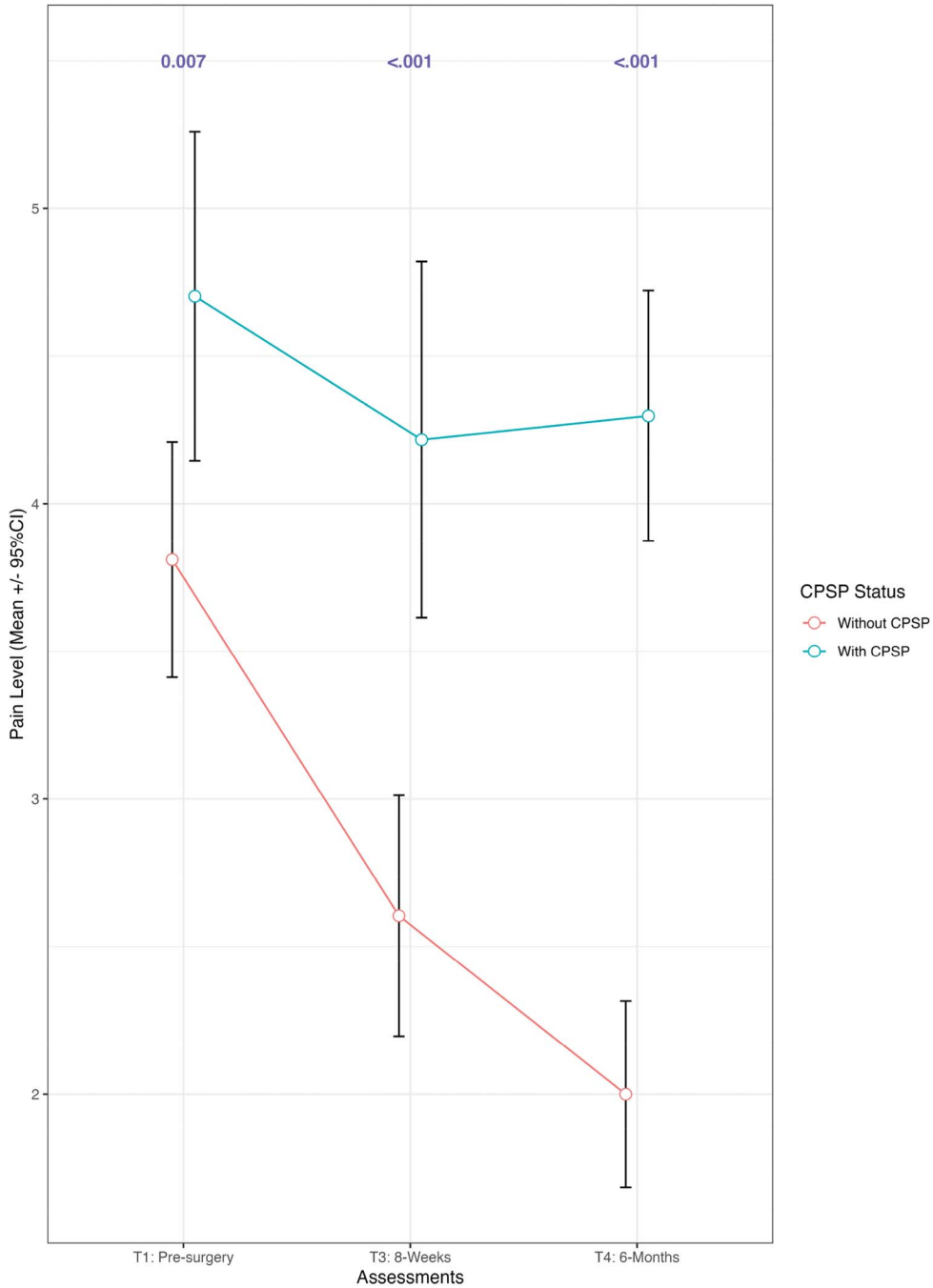

Figure 3. Self-reported pain intensity at baseline (presurgery), 8 weeks, and 6 months after surgery.

adolescent anxiety symptoms decreased in both groups, the decrease was larger in the non-CPSP group (difference in *T*-score = −2.93, 95% CI −6.25 to 0.39, *P* = 0.08). Changes in adolescent pain catastrophizing was not statistically significant, and changes in parent distress were similar among the CPSP and non-CPSP groups.

3.2. Mediation models

3.2.1. Psychosocial mediators for the association between postsurgical acute pain intensity and CPSP

Table 3 summarizes the direct effect of acute pain intensity on CPSP, the indirect effect of acute pain intensity on CPSP mediated by each psychosocial variable, the total effect of acute pain intensity on CPSP, and the proportion mediated for each model. As expected, in all adjusted models, acute pain intensity predicted CPSP status, with statistically significant total and direct effects of acute pain intensity on subsequent CPSP. Regarding psychosocial mediation, changes in adolescent depressive symptoms and parent distress did not mediate the association between postsurgical acute pain intensity and CPSP at 6 months. Notably, changes in adolescent anxiety and pain catastrophizing did mediate this association. Specifically for anxiety, each 1-point increase in the mean postsurgical acute pain intensity was associated with a total of 1.70 odds of developing CPSP (total effect; 95% CI

Table 2

Levels and changes in psychosocial variables by CPSP status.

Psychosocial variables	CPSP status		Difference (95% CI)	*P*
	Without CPSP (n = 98): Mean (SD)	With CPSP (n = 47): Mean (SD)		
Adolescent depressive symptoms (RCADS T-score)				
T1	47.66 (12.33)	57.99 (13.33)	**−10.33 (−14.93 to −5.73)**	**<0.001**
T3	46.17 (13.49)	61.15 (16.74)	**−14.98 (−20.57 to −9.40)**	**<0.001**
Change from T1 to T3	−1.56 (9.08)	3.17 (10.85)	**−4.73 (−8.38 to −1.08)**	**0.01**
Adolescent anxiety symptoms (RCADS T-score)				
T1	46.89 (14.07)	56.97 (13.99)	**−10.08 (−15.03 to −5.14)**	**<0.001**
T3	43.34 (13.04)	56.26 (15.00)	**−12.93 (−18.02 to −7.83)**	**<0.001**
Change from T1 to T3	−3.64 (8.42)	−0.71 (9.79)	−2.93 (−6.25 to 0.39)	0.08
Adolescent pain catastrophizing (PCQ Internalizing/Catastrophizing subscale)				
T1	2.14 (0.88)	2.52 (0.92)	**−0.38 (−0.70 to −0.05)**	**0.02**
T3	1.88 (0.79)	2.51 (0.91)	**−0.63 (−0.94 to −0.31)**	**<0.001**
Change from T1 to T3	−0.26 (0.73)	−0.04 (0.99)	−0.22 (−0.55 to 0.11)	0.18
Parent psychological distress (BSI-18 total score)				
T1	5.05 (6.70)	7.72 (8.22)	−2.67 (−5.42 to 0.07)	0.06
T3	4.33 (5.69)	7.84 (8.30)	**−3.51 (−6.24 to −0.79)**	**0.01**
Change from T1 to T3	−0.72 (6.15)	0.40 (7.12)	−1.12 (−3.58 to 1.34)	0.37

Bolded values are significant at $P < 0.05$.
RCADS, Revised Child Anxiety and Depression Scale; T1, presurgery; T3, 8 weeks after surgery.

1.30-2.62). Changes in adolescent anxiety symptoms mediated the association between postsurgical acute pain intensity and the odds of CPSP development. Higher postsurgical acute pain intensity attenuated the postsurgical anxiety reduction, leading to high odds of CPSP, explaining 13.8% (95% CI 2.14-32.5) of the total effect. Thus, 13.8% of the effect of postsurgical acute pain intensity on CPSP was mediated through changes in adolescent anxiety symptoms. Similarly for pain catastrophizing, higher postsurgical acute pain intensity was associated with less reduction in adolescent pain catastrophizing scores from presurgery to 8 weeks postsurgery. Limited reduction in pain catastrophizing scores was, in turn, associated with increased odds of developing CPSP, explaining 11.0% (95% CI 2.69-33.4) of the total effect. In all the mediation analyses, no statistically significant exposure–mediator interactions were observed.

3.2.2. Psychosocial mediators for the association between postsurgical acute pain interference and CPSP

Controlling for covariates, higher postsurgical acute pain interference was associated with higher odds of CPSP, with significant direct and total effects (**Table 4**). Regarding psychosocial mediation, changes in adolescent depressive symptoms, anxiety, and parent distress did not mediate the association between postsurgical acute pain interference and CPSP at 6 months. Importantly, changes in adolescent pain catastrophizing did mediate this association. Specifically, each 1-point higher

Table 3

Psychosocial mediation for the association between acute pain intensity and CPSP.

Model	OR	SE	95% CI	*P*
Pain intensity → Δ depression → CPSP				
Direct effect	1.64	0.38	(1.24-2.62)	<0.001*
Indirect effect	1.02	0.04	(0.94-1.11)	0.46
Total effect	1.65	0.37	(1.25-2.62)	<0.001*
Overall proportion mediated (%)	6.14	9.78	(−17.5 to 21.1)	
Pain intensity → Δ anxiety → CPSP				
Direct effect	1.59	0.28	(1.21-2.27)	<0.001*
Indirect effect	**1.06**	**0.04**	**(1.01-1.17)**	**0.02***
Total effect	1.70	0.35	(1.30-2.62)	<0.001*
Overall proportion mediated (%)	13.8	8.47	(2.14-32.5)	
Pain intensity → Δ pain catastrophizing → CPSP				
Direct effect	1.61	0.30	(1.20-2.37)	<0.001*
Indirect effect	**1.05**	**0.04**	**(1.01-1.15)**	**0.02***
Total effect	1.71	0.34	(1.28-2.64)	<0.001*
Overall proportion mediated (%)	11.0	8.06	(2.69-33.4)	
Pain intensity → Δ parent distress → CPSP				
Direct effect	1.55	0.32	(1.10-2.30)	<0.001*
Indirect effect	1.05	0.06	(0.99-1.19)	0.24
Total effect	1.64	0.37	(1.18-2.58)	<0.001*
Overall proportion mediated (%)	12.0	12.7	(−5.17 to 39.4)	

* $P < 0.05$; Significant indirect effects were bolded.

Table 4

Psychosocial mediation for the association between acute pain interference and CPSP.

Model	OR	SE	95% CI	*P*
Pain interference → Δ depression → CPSP				
Direct effect	1.30	0.20	(1.03-1.78)	0.02*
Indirect effect	1.06	0.04	(1.00-1.16)	0.06
Total effect	1.37	0.23	(1.10-2.02)	<0.001*
Overall proportion mediated (%)	20.0	9.8	−0.78 to 57.9	
Pain interference → Δ anxiety → CPSP				
Direct effect	1.30	0.20	(1.02-1.76)	0.04*
Indirect effect	1.06	0.05	(1.00-1.20)	0.06
Total effect	1.37	0.23	(1.07-1.98)	<0.001*
Overall proportion mediated	19.2	27.8	(−1.19 to 73.3)	
Pain interference → Δ pain catastrophizing → CPSP				
Direct effect	1.34	0.22	(1.04-1.85)	0.02*
Indirect effect	**1.06**	**0.04**	**(1.04-1.16)**	**0.02***
Total effect	1.43	0.24	(1.15-2.04)	<0.001*
Overall proportion mediated	19.6	17.9	(3.04-70.2)	
Pain interference → Δ parent distress → CPSP				
Direct effect	1.38	0.23	(1.11-1.89)	<0.001*
Indirect effect	1.02	0.04	(0.99-1.13)	0.28
Total effect	1.43	0.23	(1.16-2.01)	<0.001*
Overall proportion mediated	7.53	12.1	(−4.12 to 37.7)	

* $P < 0.05$; Significant indirect effects were bolded.

mean postsurgical acute pain interference was associated with a total of 1.43 odds of developing CPSP (total effect; 95% CI 1.15-2.04). Higher postsurgical acute pain interference was associated with less improvement in adolescent pain catastrophizing, which in turn, was associated with higher odds of CPSP. Changes in adolescent pain catastrophizing explained 19.6% (95% CI 3.04-70.2) of the total effect of acute pain interference on CPSP. In all the mediation analyses, no significant exposure–mediator interactions were observed.

3.2.3. Prediction of CPSP by presurgical psychosocial variables and their changes from presurgery to 8 weeks postsurgery

As summarized in **Supplemental Table 3**, http://links.lww.com/PAIN/C281, both presurgery psychosocial variables and changes in psychosocial variables, including adolescent depressive symptoms, anxiety symptoms, and pain catastrophizing, significantly predicted CPSP at 6 months.

4. Discussion

The results of this study demonstrate the influence of psychosocial changes in response to the experience of surgery and acute pain on the development of CPSP, by linking postsurgical acute pain intensity and interference to the development of CPSP 6 months later. First, compared with adolescents who did not develop CPSP, adolescents who developed CPSP had higher levels of clinical and borderline clinical depressive and anxiety symptoms, pain catastrophizing, and parental distress both before surgery and at 8 weeks postsurgery. Examining changes in symptoms from presurgery to 8 weeks postsurgery illuminated an increase in depressive symptoms from presurgery to 8 weeks for adolescents who developed CPSP (compared with a decrease for those who did not develop CPSP). Furthermore, anxiety symptoms decreased among those with and without CPSP from presurgery to 8 weeks postsurgery, but reductions were smaller for those who developed CPSP. Compared with adolescents who did not develop CPSP, those who developed CPSP had higher mean pain intensity and interference in the 30 days after surgery. The results identified the importance of changes in adolescent anxiety and pain catastrophizing levels after surgery in driving the associations between postsurgical acute pain and CPSP development. Specifically, higher postsurgical acute pain intensity and interference attenuated reductions in adolescent anxiety and pain catastrophizing, increasing the likelihood of CPSP.

These findings extend past work, which has primarily examined baseline psychosocial risk factors for CPSP, including anxiety, pain catastrophizing, and depressive symptoms *before surgery*.[33,34,38,42,48] Our new findings provide mechanistic specificity of *how* these psychosocial factors relate to the development of CPSP over time, highlighting mechanistic targets for psychological interventions to reduce the risk of CPSP. Taken together, this body of research supports screening and intervention targeting both presurgery levels and postsurgical changes in psychosocial factors to prevent development of CPSP after major surgery.[33,34,38,42,48]

This study highlights the importance of anxiety, a general psychological factor, and pain catastrophizing, a pain-specific cognitive–emotional factor, in the development of pediatric CPSP. Although only pain catastrophizing was detected as a significant mediator for both acute pain intensity and acute pain interference in relation to CPSP, anxiety and pain catastrophizing showed a similar proportion of mediation (19.2% and 19.6%) for the effect of acute pain interference on the development of CPSP. Both general and pain-specific psychosocial factors have been implicated in the onset and maintenance of chronic pain.[11,27,30] A recent systematic review of psychological and psychosocial predictors of CPSP in adults indicated that state anxiety (or current anxiety) is consistently predictive of CPSP, while trait anxiety (or more stable anxiety) has a weaker association with CPSP.[19] Although numerous studies have examined pain catastrophizing in youth undergoing surgery, studies have been

small, focusing on preoperative or baseline pain catastrophizing only and have yielded conflicting results. A recent meta-analysis found that preoperative anxiety and pain catastrophizing are not consistent predictors of pediatric CPSP.[50] Our results suggest that changes in general and pain-specific anxiety after surgery play a role in the onset of CPSP in adolescents.

Recently, the pediatric diathesis-stress and Interpersonal Fear-Avoidance models of chronic pain (IFAM)[1,53,54,63] were combined to explain transition from acute to chronic pain in youth, positing that psychological responses to pain, including pain catastrophizing, drive pain-related fear, avoidant behaviors, and pain-related disability.[46] This process is largely influenced by youth's own vulnerabilities (e.g., anxiety) and interpersonal and relational factors (e.g., parent vulnerabilities and parent responses to child's pain). Several constructs in the Fear-Avoidance model, most notably pain catastrophizing, have gained extensive empirical support particularly in the context of adult postsurgical pain. For instance, pain catastrophizing, but not kinesiophobia, hypervigilance, or pain sensitivity, was found to mediate the treatment effect of perioperative pain neuroscience education on physical health-related quality of life 1-year postsurgery in individuals undergoing surgery for lumbar radiculopathy.[59] In a small pilot trial, changes in pain catastrophizing, rather than changes in depression or anxiety, were associated with lower pain severity and pain interference at 6 weeks after surgery in those receiving a brief mindfulness-based cognitive behavioral therapy compared with those receiving treatment-as-usual.[35] In a sample of young male patients (13-33 years) undergoing thoracic surgery for pectus excavatum correction, acute postsurgical pain and general psychological variables (e.g., depression and anxiety) did not predict persistent postsurgical pain but pain-related psychological variables (e.g., pain anxiety and pain vigilance) did.[24] Our findings indicate that changes in pain catastrophizing *after* surgery plays a role in the onset of adolescent CPSP.

It is also important to acknowledge discrepancies between significant psychosocial mediators in this study compared with previous research. Previous studies of pain-specific factors, both at the individual and parent-level, found consistent and robust association of catastrophizing with immediate postsurgical acute pain,[15,19,20,37] while findings are mixed about CPSP.[48,50] Some studies suggest that child and parent pain catastrophizing may each be uniquely and differentially related to pain persistence.[2,4] In this study, parent distress was not found to mediate the acute pain–CPSP relationship. However, our study focused on child factors, with limited assessment of parent factors (parent distress only). Future work should examine additional parent psychosocial factors such as parent pain catastrophizing and protective parent behaviors, which may influence their child's CPSP risk. There may be a unique pathway through which parents and caregivers influence adolescents' coping ability after surgery,[1,32] with some work highlighting increased correlation between parent psychosocial factors (e.g., parent catastrophizing immediately after surgery) and child's long-term pain outcomes (e.g., pain intensity at 12 months after surgery), suggesting a mutual learning model in the postsurgical period.[33] Yet these studies did not examine *changes* in individual and parent psychosocial factors. It is also possible that these factors reflect more trait-level factors that do not change. Although our study did not aim to test sociodemographic factors in adolescent CPSP, the lower incidence of CPSP among adolescents with married parents highlights the potential importance of family systems. Married parents may have greater capacity and resources to offer support during the critical postsurgical recovery period, reducing CPSP risk in adolescents. In families of unmarried parents, unique vulnerabilities (e.g., strained relationships and protective parenting)[32] may increase the risk. Further research is needed to explore the influence of parent and family dynamics on adolescent CPSP.

This study has clinically relevant implications for interventions to reduce the onset and impact of CPSP. The results of this study suggest that, although presurgical assessment is an initial step in identifying vulnerabilities for CPSP, postsurgical monitoring of pain intensity, interference, anxiety, and pain catastrophizing may provide opportunities for identification and early intervention for those adolescents at highest risk for CPSP development. Psychosocial interventions targeting both anxiety and pain catastrophizing in the context of pain have robust empirical evidence and could be adapted to meet the postsurgical needs of adolescents.[31,43,51,68] In our sample, only a small proportion of adolescents exhibited clinical or borderline clinical levels of depression and anxiety. This means that mental health screening measures and clinical cut-points may fail to identify most adolescents at risk of CPSP. Thus, additional work is needed to establish optimal screening for CPSP risk taking into account both baseline levels of psychosocial vulnerability, as well as the changes in the psychosocial profiles during the postsurgical recovery phase, to enable comprehensive identification and implementation of targeted interventions for individuals at higher risk of CPSP. In addition, given that the psychosocial mechanisms explained a proportion of the effects of postsurgical acute pain on CPSP, further research is needed to understand additional mechanisms (e.g., surgical, clinical, biological, and social) for the development of pediatric CPSP, which will inform targeted prevention interventions.

This study has several strengths that extend upon prior work. First, we used intensive daily diary assessment of acute pain during the postsurgical period to capture acute pain intensity and interference. We also measured psychosocial functioning at presurgery and 2 months after surgery, thereby offering an opportunity to test the dynamic nature of psychosocial mechanisms in the onset of CPSP. Our definition of CPSP incorporates both pain intensity and quality of life, enhancing the clinical relevance of the outcome. Furthermore, the study included a sizable proportion of Hispanic families and inclusion of Spanish speaking families, increasing diversity and improving the generalizability of the findings. Research is needed to examine sociodemographic disparities and how they affect mechanisms of CPSP.[17]

There are some limitations that warrant comment. First, although our sample included diverse racial and ethnic groups, the small sample sizes within each group (e.g., Hispanic families) limited our ability to conduct the mediation analysis within these subgroups. Similarly, we were constrained in ability to conduct sex-specific mediation analyses. Furthermore, although we examined parent factors, this was limited to their own psychological distress. We examined the individual mediation effect of psychosocial factors; potential complex interrelationships among these factors and their combined mediation effects warrant further investigation.

Overall, this study identified changes in adolescent anxiety and pain catastrophizing after surgery as key factors driving the transition from acute to chronic pain after major surgery. Identifying these psychosocial mechanisms highlights potential postsurgical intervention targets to reduce the impact of the acute postsurgical pain experience and subsequent development of CPSP. Future work should examine outcomes at longer follow-ups (e.g., 12 months) to better understand CPSP trajectories and

persistence and test interventions targeting these psychosocial factors to reduce the risk of CPSP.

Conflict of interest statement

The authors have no conflicts of interest to declare.

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