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REVIEW

Presurgical Prognostic Factors for Chronic Postsurgical Pain Across Developmental Stages in Children, Adolescents, and Young Adults: A Systematic Review and Meta-Analysis

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Background: Chronic postsurgical pain (CPSP) is a significant postoperative complication in children and adolescents, with long-term consequences that may extend into young adulthood. Although numerous presurgical factors have been proposed as potential predictors of CPSP, findings remain inconsistent, and no prior review has examined prognostic factors across a developmental window spanning childhood to young adulthood. The aim of this study was to identify presurgical prognostic factors for CPSP at 3, 6, 12, and 24 months after surgery in children, adolescents, and young adults.

Methods: A systematic search of five electronic databases was conducted from inception to September 2025, following PRISMA guidelines. Eligible studies included participants aged 8–22 years undergoing major surgery and reporting presurgical variables associated with CPSP. Two reviewers independently screened studies, extracted data, and assessed risk of bias. When possible, meta-analyses were performed using standardized mean differences or odds ratios.

Results: Thirty articles were included. Although 44 presurgical factors were identified, only eight could be meta-analyzed due to inconsistent reporting, limiting the strength of quantitative conclusions. Baseline pain intensity was the most consistent predictor of CPSP, showing significant associations at 3 months and 6 months. Older age showed a small association with CPSP at 3 months but not thereafter. Sex, child pain catastrophizing, pain anxiety, anxiety sensitivity, parent pain catastrophizing, and parent pain anxiety were not significantly associated with CPSP. The evidence base was limited by heterogeneity, small samples, and underreporting of effect sizes.

Conclusion: Across childhood, adolescence, and young adulthood, baseline presurgical pain intensity is the most robust predictor of CPSP. Psychological and sociodemographic factors showed limited prognostic value. However, quantitative conclusions are limited by inconsistent reporting across studies.

Plain Language Summary:

Why was the study done?

Some children, adolescents, and young adults continue to experience pain long after surgery. This long-lasting pain is called chronic postsurgical pain and can affect daily life, school, work, and well-being. Most previous studies have focused on adults or only on short age ranges. Because of this, we still do not clearly understand which factors increase the risk of developing chronic pain after surgery across different stages of development, from childhood to young adulthood. Identifying these factors matters because some of them may be changed, helping to prevent long-term pain.

What did the researchers do?

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By carefully selecting high-quality studies, the researchers explored which biological, psychological, and social factors were linked to a higher risk of long-term pain after surgery at different ages.

What did the researchers find?

The study found that several factors were linked to a higher risk of chronic postsurgical pain. These included older age within the pediatric-to-young-adult range, higher pain levels before or shortly after surgery, and psychological factors such as anxiety or fear related to pain. Similar factors appeared to play a role across ages, although their influence may change with development.

What do these results mean?

These findings show that chronic postsurgical pain develops through a combination of physical, psychological, and social factors. Importantly, some of these factors can be addressed early. This suggests that age-appropriate screening and tailored support before and after surgery may help reduce long-term pain in young people.

Keywords: chronic postsurgical pain, prognostic factors, childhood, adolescence, young adulthood

Introduction

Chronic postsurgical pain (CPSP), defined as persistent pain 3 months after surgery,[1] represents a significant global health concern with substantial physical, psychological, and social consequences across the lifespan.[2–4] In pediatric populations, CPSP is increasingly recognized as a common postoperative complication. Recent studies show that a substantial number of children and adolescents develop CPSP at 3 months after surgery,[2] highlighting the magnitude of the problem and its potential to interfere with recovery, functioning, and quality of life.[2,3,5]

Although similar concerns extend into adulthood, most research has examined CPSP within narrowly defined age groups, typically focusing on "childhood" or "adulthood" in isolation.[6] Consequently, little is known about which presurgical risk factors consistently predict CPSP across the developmental span from childhood to young adulthood.[7] Young adulthood is increasingly understood as a distinct developmental stage marked by accelerated neurobiological maturation, evolving social contexts, and expanding psychosocial and functional demands.[8] Ongoing maturation of prefrontal–limbic circuits involved in pain modulation and emotion regulation, together with increased exposure to psychosocial stressors and autonomy-related demands, increase vulnerability to pain chronification during this period.[9–11] These transitions may heighten vulnerability to persistent postsurgical pain, yet this group remains markedly under-represented in CPSP research. Recent findings on non-surgical chronic pain indicate that a substantial proportion of adolescents continue to experience pain, disability, and psychological distress into early adulthood, underscoring the importance of examining continuity and change across developmental stages.[12] Despite these observations, comparable evidence for CPSP— and particularly evidence examining presurgical predictors across this expanded age range— remains scarce.

A growing body of literature has identified several potentially modifiable predictors of CPSP in children and adolescents, including high levels of acute postoperative pain, psychological symptoms, sleep disturbances, fear of pain, kinesiophobia, and impairments in physical functioning.[2,3,5,13,14] Developmental processes, prior pain experiences, and increased psychosocial vulnerability during adolescence may further contribute to heightened risk.[8] However, existing studies are limited by methodological heterogeneity, narrow age ranges, and a predominant focus on pediatric samples, and inconsistent reporting of effect sizes, resulting in major gaps in our understanding of which presurgical factors demonstrate consistent prognostic value across developmental stages.[2,13]

To date, no review has systematically examined CPSP risk factors across this expanded developmental window. Such integration is essential to clarify which factors consistently predict CPSP from childhood through young adulthood and to identify potential targets for early prevention and intervention.[8,12] Addressing these gaps is crucial given the potential for CPSP to interfere with key developmental milestones and shape long-term trajectories of health, functioning, and wellbeing.

Based on these considerations the primary aim of this study was to identify presurgical predictors of CPSP at 3 months after surgery in children, adolescents, and young adults. Exploratorily, we aimed to identify presurgical predictors of CPSP at 6, 12 and 24 months after surgery in children, adolescents, and young adults.

We hypothesized that older age, higher presurgical pain intensity, greater pain catastrophizing, and higher pain anxiety would be associated with a higher likelihood of developing CPSP at 3 months after surgery. On the other hand, we hypothesized that higher pain intensity and worse quality of life before surgery would be associated with the persistence of CPSP at 6, 12, and 24 months following surgery.

Methods

Study Design

This systematic review was conducted following The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline 2020.[15] The review protocol was registered in PROSPERO (CRD 42020171597).

Protocol Deviations

Three deviations from the registered protocol were made. First, the search period was extended from the originally registered end date (January 1, 2020) to September 2025 to capture newly published studies and ensure the comprehensiveness of the review. Second, although not specified in the original protocol, a meta-analysis was conducted due to the availability of a sufficient number of eligible studies, allowing for a more robust quantitative synthesis of the findings. Third, although the PROSPERO registration initially specified the inclusion of children aged 4 to 18 years undergoing major surgery, a deviation from the protocol was made during the study selection process. When screening eligible studies, the authors identified several relevant investigations in which participant age extended beyond 18 years, typically up to 21 or 22 years. Given the developmental continuity between late adolescence and young adulthood, and the relevance of this transition period for pain chronification, the authors considered it conceptually appropriate to include these studies. This ad hoc decision allowed for a more comprehensive examination of prognostic factors across a broader developmental window, from childhood through young adulthood. Importantly, this deviation was applied consistently, did not alter the predefined outcomes or analytic approach, and was implemented to enhance the interpretability and clinical relevance of the findings.

Search Strategy

Searches in MEDLine, EMBASE, PsycINFO, WOS, Cochrane Database including articles published between January 1st of 2010 to September 30th of 2025, were conducted. The last search in the databases was carried out on August 1st, 2025.

To enhance conceptual consistency and clinical relevance, we restricted inclusion to studies published from 2010 onward. Prior to this period, substantial variability existed in the operational definitions of chronic postsurgical pain, including differences in duration thresholds and exclusion criteria, which could introduce additional heterogeneity in prognostic estimates.[16] Limiting the timeframe allowed us to align with more contemporary and progressively standardized definitions of CPSP,[1] as well as to capture studies conducted under modern perioperative practices[13] and improved methodological and reporting standards.[15,17]

The search strategy for each database was designed following the previous systematic review,[13] and with the directions by a Librarian. The search terms were composed into 5 blocks: 1. Exposure; 2. Population; 3. Clinical condition 4. Type of Study and 5. Risk/Prognostic Factors. The search terms were combined using Boolean. The full search strategy can be found in the Supplementary Document I.

Eligibility Criteria

Study Criteria

Only prospective observational studies in humans were included. Studies were required to include a minimum sample size of 20 participants, consistent with previous systematic reviews.[13]

Participant Criteria

Children, Adolescents and Young Adults

We included studies that analyzed participants aged between 8 and 24 years who had undergone major surgical interventions requiring hospitalization.[18] This age range was selected to capture the developmental continuum from

childhood through young adulthood, a critical period for the transition from acute to chronic pain.[8] Neurobiological maturation, psychosocial development, and pain-related cognitive and emotional processes continue to evolve across adolescence and into young adulthood, influencing vulnerability to pain chronification and long-term functional outcomes.[7,12]

Parents

We include studies that evaluated potential prognostic factors in parents when children and young adults are in pain. These factors could be evaluated by both parents, by one parent, or by the legal guardians.

Postsurgical Chronic Pain Criteria

CPSP was established by the presence of pain in the surgical region by at least 3 months after surgery, in accordance with the most update definition of CPSP.[1] Additionally, studies could incorporate more follow-up time points to explore CPSP at 6, 12 and 24 months after surgery. The articles included in this review evaluated CPSP at least one of these time points.

To be eligible, studies were required to assess CPSP at least once after surgery, according to the CPSP criteria described above, allowing the determination of whether participants developed CPSP.

Prognostic Factor Criteria

Prognostic factors were required to be assessed during the preoperative period, defined as any assessment conducted prior to the surgical intervention. This assessment could occur once or multiple times before surgery.

Prognostic factors had to be measured using validated instruments, or the methods of assessment had to be clearly described. Prognostic factors could be evaluated in children, adolescents, young adults, and/or their parents or legal guardians.

Study Selection and Data Extraction

Two blinded researchers (G.C.B & E.D.G) selected potential studies after searching the databases. After eliminating duplicates, studies were screened based on titles and abstracts. Discrepancies were resolved by a third researcher (I.L.U. V).[17] The predefined inclusion and exclusion criteria were then applied, and studies not meeting these criteria were excluded. From the final set of included studies, we extracted information on study characteristics, participant characteristics, and surgical characteristics.

Risk of Bias Assessment

A risk of bias analysis was performed using a Quality in Prognosis Studies Tool (QUIPS)[19] for the analysis of the risk of bias in prognostic studies. This instrument assesses the risk of bias in observational studies analyzing prognostic factors through 6 domains: Study participation, Study Attrition, Prognostic Factor Measurement, Outcome Measurement, Study confounding, and Statistical Analysis and Reporting. Each domain was evaluated at three levels: low, medium, and high risk of bias.

Level of Evidence

The degree and quality of the evidence were evaluated using the Grading of Recommendations, Assessment, Development and Evaluations (GRADE),[20] following adaptations for prognostic research proposed by Huguet et al.[21]

Two blinded reviewers (G.C.B. and EG.D.) assessed the quality of evidence for each prognostic factor across the following domains: (1) phase of investigation, (2) study limitations, (3) inconsistency of results, (4) indirectness, (5) imprecision, and (6) publication bias. Based on these criteria, the quality of evidence was classified as high, moderate, low, or very low.[20]

In line with recommendations for prognosis research,[21] the phase of investigation was considered to inform evidence interpretation,[21–23] with prognostic factors examined in six or more independent cohorts classified as supported by Phase 2–3 evidence, and emerging prognostic factors examined in fewer than six cohorts classified as supported primarily by Phase 1–2 evidence, consistent with previous reviews.[14,24–26]

Data Synthesis

Studies included in the meta-analysis were selected following criteria consistent with previous meta-analyses of CPSP prognostic factors:[27]

(a) the prognostic factor was assessed using a validated instrument for the target population; (b) the prognostic factor was evaluated preoperatively; (c) prognostic factor must be measured by the same instrument within each specific meta-analysis; and (d) the outcome (CPSP) was defined as above.

Studies were excluded from the meta-analysis if: (a) insufficient data were provided (eg., group sizes, means, or standard deviations), or (b) CPSP was defined exclusively through changes in pain intensity (eg., improvement or worsening) or percentage change from baseline. These outcomes were excluded to reduce methodological heterogeneity and ensure consistency across pooled analyses.

To analyze the prognostic factors, the Rev.Manager tool from the Cochrane Library was used.[28] Random-effects meta-analyses were conducted, given the expected clinical and methodological heterogeneity across studies.[29–31] Whenever available, raw data were extracted from the included studies, including event counts and total sample sizes for dichotomous outcomes, and means, standard deviations, and sample sizes for continuous outcomes. These data were entered into Rev.Manager[28] using the standard dichotomous and continuous data formats, and effect sizes were calculated within the software. Standardized Mean differences (SMD) (continuous) and odds ratios (OR) (dichotomic) for the variables were calculated. Effect sizes were interpreted using commonly accepted thresholds. For SMD, values of approximately 0.2 were considered small, around 0.5 moderate, and 0.8 or greater large effects.[32] For OR, values around 1.5 were considered small, approximately 2.5 moderate, and 4.0 or greater large effects, reflecting clinically meaningful increases in the likelihood of developing CPSP.[33,34]

There is no universally established minimum number of studies required for meta-analysis;[29] however, at least two studies are necessary to perform a pooled analysis.[35] Meta-analyses were conducted separately for each assessment point (3, 6, 12, and 24 months) when a minimum of two studies reporting data for that specific time point was available.

Assessment of Heterogeneity into Studies

The presence of heterogeneity among studies was evaluated using Cochran's Q statistic.[36] To further quantify this heterogeneity, the I^2 index was calculated, which measures the percentage of total variation across studies that is due to heterogeneity rather than chance.[31] An I^2 value of 75% or higher indicates substantial heterogeneity, showing significant variability among the study results.[31]

Given the anticipated low number of studies, we also included the Tau and tau-squared statistics to estimate heterogeneity among studies, providing a measure of variability beyond what is expected by chance. Higher Tau and tau-squared values suggest greater heterogeneity, indicating that differences among studies are not solely due to chance but reflect real variations in study effects.[37,38]

Results

Selection of Studies

The initial search yielded 22,300 records after removing duplicates (Figure 1). Following title and abstract screening, 216 articles were assessed in full text. Of these, 30 articles (29 studies)[3,39–65] met the inclusion criteria and were included in the review. Twenty-six articles (25 studies) contributed data to the quantitative synthesis (meta-analysis).[3,39,42–47,49–58,60–67]

Discrepancies during the screening process occurred in only three cases and were resolved through consultation with a third reviewer. Inter-rater agreement was excellent ($\kappa = 0.85$).

Characteristics of the Studies and Participants

The 30 included articles (29 studies) were published between 2011 and 2025, with most appearing after 2015 (Table 1). In total, data from 5,135 children, adolescents, and young adults aged 8–22 years undergoing major surgery were analyzed. Sample sizes ranged widely across studies. Follow-up assessments varied, with most studies evaluating CPSP between 3 and 12 months, and a minority extending follow-up to 24 months. Retention rates were generally high across

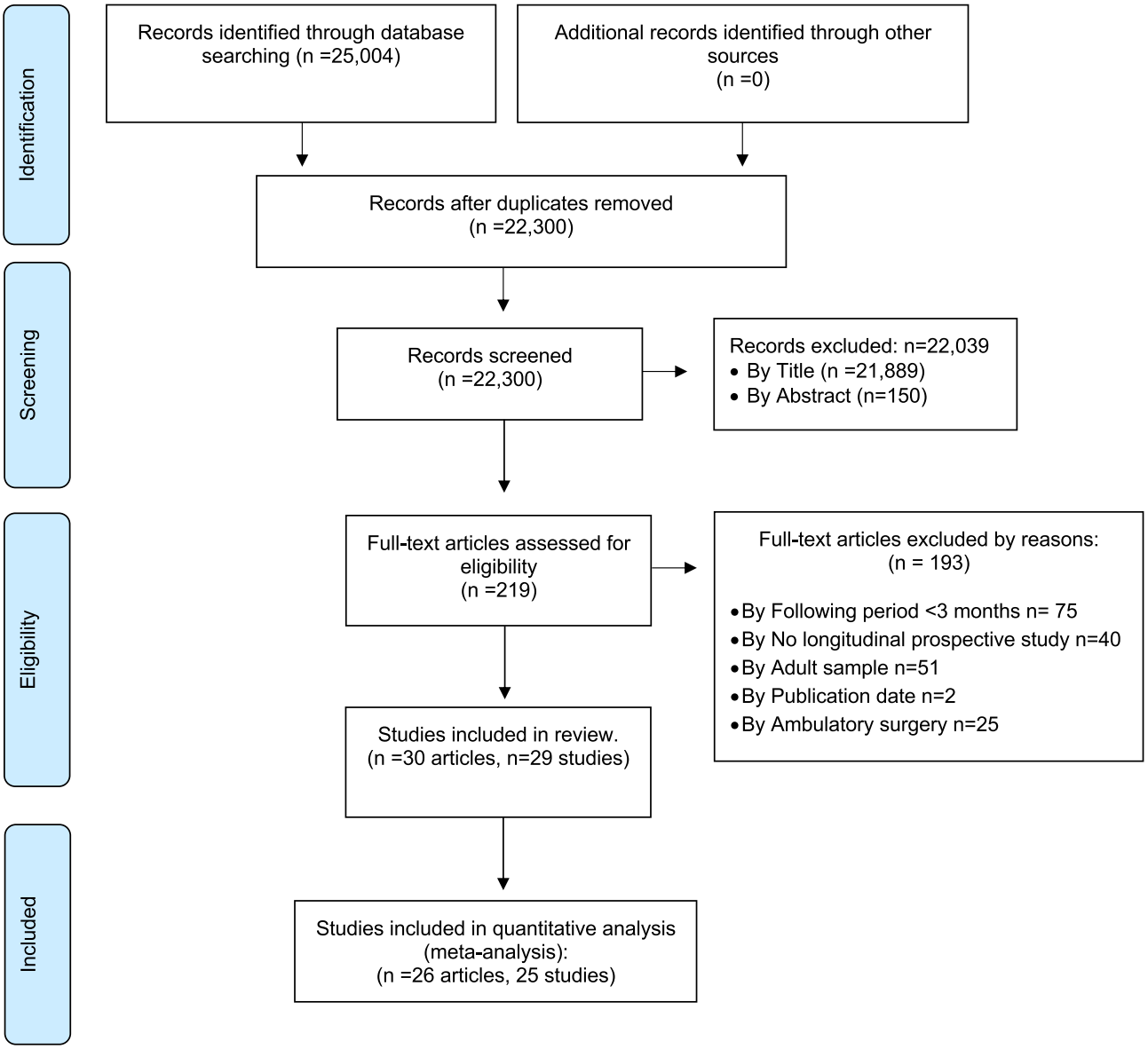

Figure 1 Flow diagram. Flow diagram of the study selection process. A total of 25,004 records were identified through database searching, with no additional records from other sources. After removing duplicates (n = 22,300), records were screened, and 22,039 were excluded based on title and abstract. Full-text articles were assessed for eligibility (n = 219), with 193 excluded for reasons including short follow-up (<3 months), non-longitudinal design, adult samples, publication date, and ambulatory surgery. A total of 30 articles (29 studies) were included in the review, of which 26 articles (25 studies) were included in the quantitative synthesis (meta-analysis).

all time points. A detailed description of sample characteristics, surgical procedures, and follow-up periods is provided in Table 1.

Risk of Bias of the Studies Included

Risk of bias was assessed for all included studies and is summarized in Figure 2. Inter-rater agreement between the two reviewers was good ($\kappa = 0.75$).

Only five studies demonstrated a low risk of bias across all domains.[45,46,52,60,68] The most common source of bias was study attrition, with most studies rated as having moderate or high risk in this domain. Issues were also frequently identified in the statistical analysis and reporting domain. In contrast, the prognostic factor measurement domain showed the lowest concerns, with almost all studies judged to be at low risk. A detailed breakdown of ratings by domain is provided in Supplementary Document II.

Table 1 Characteristics of Included Studies

Study	Country	Race/Ethnicity	Sample Size	Age, Mean (Range)	Female (%)	Surgery Type
Ceniza-Bordallo, 2025 et al[3]	Spain	Race: not reported Ethnicity: not reported	159	12.0 (8–18)	37.1	General surgery
Sieberg et al, 2023[42]	USA	Race: White 87.5%, Black 9.4%, Asian 3.1% Ethnicity: not reported	32	13.9 (10–17)	78.0	Spinal fusion
Ellyson et al, 2022[46]	USA	Race: White 77.8%, Black/African American 4.3%, Asian 3.4%, Pacific Islander 0.8%, American Indian/Alaska Native 0.8%, Multiracial 3.2%, Persian 0.8%, Other/Unknown 9.4%. Ethnicity: Hispanic 8.6%, Not Hispanic 86.3%, Unknown 5.1%	117	14.5 (10–18)	64.1	Major musculoskeletal surgery (spinal fusion, pectus repair, hip/femur osteotomy)
Fraser et al, 2022[41]	USA	Race: not reported Ethnicity: not reported	110	15.0 (range not reported)	86.1	Posterior spinal fixation
Beeckman et al, 2021[40]	Belgium	Race: White 99%, Asian 1% Ethnicity: not reported	100	15.2 (12–18)	77.0	Posterior spinal fusion
Bailey et al, 2021[60]	Canada	Race: not reported Ethnicity: not reported	220	14.6 (10–20)	86.0	Posterior scoliosis correction
Chidambaran et al, 2021[49]	USA	Race: White 79.7% Ethnicity: not reported	74	14.4 (10–18)	85.0	Idiopathic scoliosis or kyphosis undergoing spinal fusion
Rabbitts et al, 2021[45]	USA	Race: White 76.2%, Black/African American 4.6%, Asian 4.6%, Other/Not reported 14.6% Ethnicity: Hispanic/Latino 4.6%, Not Hispanic/Latino 90.0%, Unknown 6.4%	109	14.4 (10–18)	85.0	Major musculoskeletal surgery (spinal fusion, pectus repair, hip/femur osteotomy)
Palabiyik et al, 2021[58]	Turkey	Race: not reported Ethnicity: not reported	158	12.8 (8–18)	38.6	Open appendectomy
Rabbitts et al, 2020[14]	USA	Race: White 78.2%, African American 4.2%, Asian 3.4%, Other/Not reported 14.3% Ethnicity: Hispanic/Latino 8.4%, Not Hispanic/Latino 86.6%, Not reported 5.0%	119	14.9 (10–18)	63.0	Major musculoskeletal surgery
Perry et al, 2020[47]	USA	Race: White 58.3%, Black 11.1%, Asian 5.6%, Other/Missing 25.0% Ethnicity: not reported	36	14.0 (10–17)	75.0	Spinal fusion for idiopathic scoliosis

(*Continued*)

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Table 1 (Continued).

Study	Country	Race/Ethnicity	Sample Size	Age, Mean (Range)	Female (%)	Surgery Type
Ocay et al, 2020[61]	Canada	Race: not reported Ethnicity: not reported	106	15.4 (10–18)	76.4	Spinal fusion for idiopathic scoliosis
Noel et al, 2019[62]	Canada	Race: White 59.5%, African Canadian 5.5%, South Asian 5.1%, East Asian 4.6%, African Caribbean 1.7%, Hispanic 1.7%, Aboriginal 1.3%, Middle Eastern 0.4%, Other 11.0% Ethnicity: not reported	237	14.1 (8–17)	73.8	Orthopedic or general surgery
Rosenbloom et al, 2019[63]	Canada	Race/Ethnicity: Caucasian 65.0%, African Canadian 6.0%, South Asian 5.6%, East Asian 4.4%, African Caribbean 1.7%, Hispanic 1.7%, Aboriginal 1.3%, Other 11.6%	265	14.1 (8–18)	58.5	Orthopedic or general surgery
Chidambaran et al, 2019[50]	USA	Race: White 82.0% Ethnicity: not reported	73	14.4 (10–18)	84.0	Posterior spinal fusion
Li et al, 2019[64]	Canada	Race: not reported Ethnicity: not reported	106	15.4 (10–18)	76.0	Spinal fusion for idiopathic scoliosis
Knudsen et al, 2018[59]	Denmark	Race: not reported Ethnicity: not reported	36	16.0 (13–23)	11.0	Pectus carinatum repair
Voepel-Lewis et al, 2018[68]	USA	Race: not reported Ethnicity: not reported	95	Not reported (10–17)	75.7	Posterior spinal fusion
Chidambaran & Ding et al, 2017[52]	USA	Ethnicity: Caucasian 84.9%	144	14.4 (10–18)	73.4	Spinal fusion for idiopathic scoliosis
Chidambaran & Zhang et al, 2017[51]	USA	Race: not reported Ethnicity: not reported	133	14.5 (10–18)	74.0	Spinal fusion for idiopathic scoliosis
Julien-Marsollier et al, 2017[57]	France	Race: not reported Ethnicity: not reported	36	15.0 (11–17)	86.1	Correction of idiopathic scoliosis
Noel et al, 2017[62]	USA & Canada	Race: White 90.0% Ethnicity: not reported	66	14.7 (10–18)	68.0	Major elective surgery (pectus repair, spinal fusion)
Batoz et al, 2016[67]	France	Race: not reported Ethnicity: not reported	291	12.0 (6–18)	40.0	Orthopedic, thoracic, abdominal, or urologic surgery

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Rabbitts et al, 2015[69]	USA	Race: White 83.4%, African American 3.3%, Asian 3.3%, Other/ Not reported 10.0% Ethnicity: Hispanic 1.7%, Not Hispanic 88.3%, Not reported 10.0%	60	14.7 (10–18)	66.7	Spinal fusion or pectus repair
Sieberg et al, 2013[39]	USA	Race: White 81.7%, Black/African American 8.4%, Asian 3.1%, Hispanic 1.6%, Other 1.6%	260	14.4 (SD 2.2), 14–21	72.3	Idiopathic scoliosis surgery
Bastrom et al, 2013[54]	USA	Race: not reported Ethnicity: not reported	584	14.7 (SD 2.0), 10–21	80.0	Adolescent idiopathic scoliosis surgery
Pagé & Stinson et al, 2013[66]	Canada	Race/Ethnicity: Caucasian 64.0%, Asian 12.0%, African Caribbean/ African Canadian 8.4%, Middle Eastern 4.8%, Hispanic 3.6%, Other 7.2%	83	13.8 (SD 2.4), 8–18	67.5	General surgery
Pagé & Campbell et al, 2013[65]	Canada	Race/Ethnicity: Caucasian 64.0%	83	13.8 (SD 2.4), 8–18	67.5	General or orthopedic surgery
Landman et al, 2011[55]	USA	Race: not reported Ethnicity: not reported	1,433 (295 analyzed)	Not reported (8–22)	80.4	Posterior spinal fusion for idiopathic scoliosis
Fortier et al, 2011[56]	USA	Race: White 89.0%, Asian American 6.0%, African American 3.0%, Other 3.0% Ethnicity: Hispanic/Latino 35.0%, Not Hispanic/Latino 59.0%, Unknown 6.0%	113	12.4 (2–17)	32.0	Orthopedic, urologic, or general surgery

Abbreviations: SD, standard deviation; USA, United States of America.

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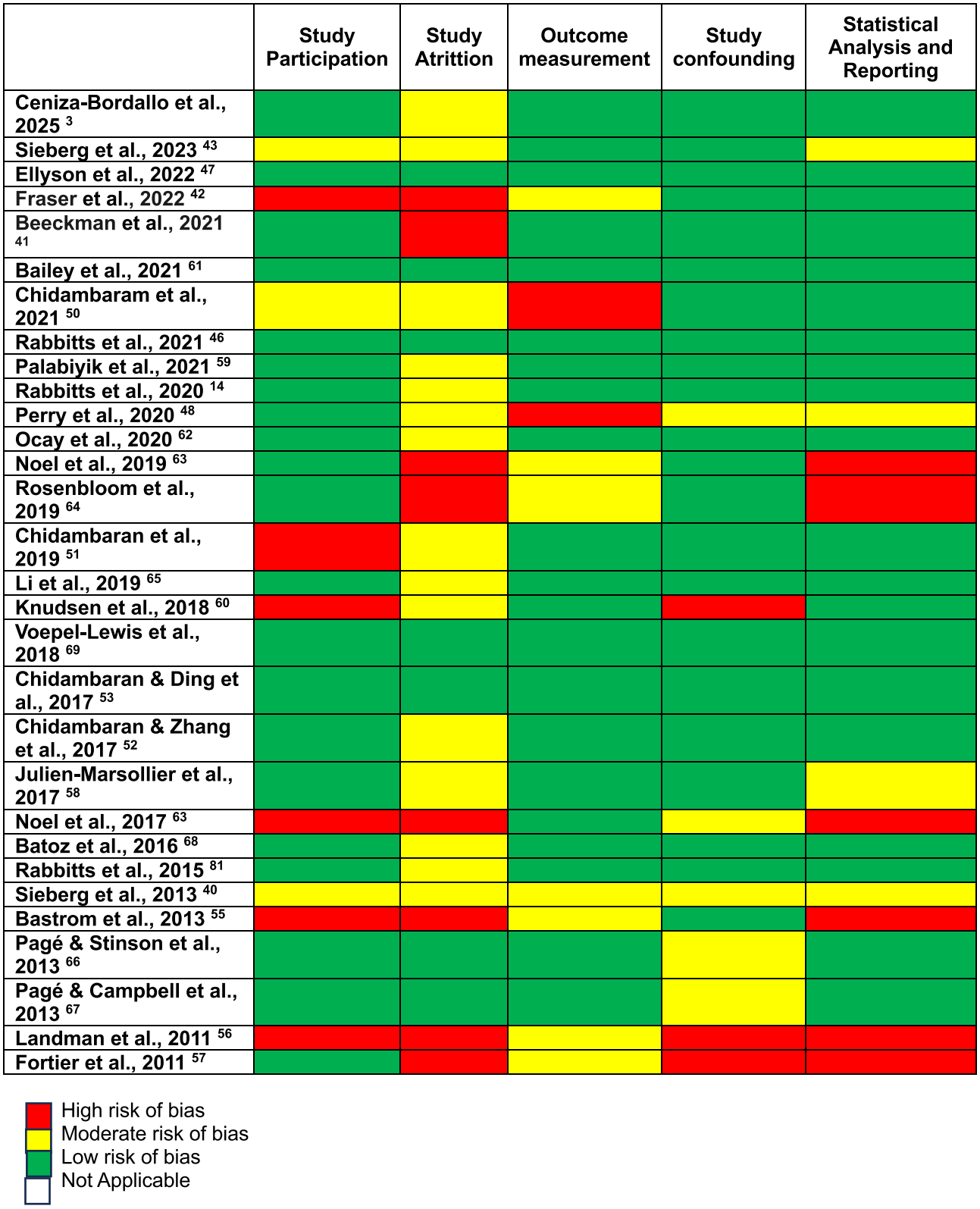

Figure 2 Risk of bias assessment across included studies. Risk of bias evaluation of the included studies across five methodological domains: study participation, study attrition, outcome measurement, study confounding, and statistical analysis and reporting. Each study is represented in rows, and domains are presented in columns. Risk of bias was classified as low (green), moderate (yellow), or high (red), according to predefined criteria. This figure provides an overview of the methodological quality and potential sources of bias across the included studies.

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Prognostics Factors to CPSP

A total of 44 presurgical factors were examined across studies, with follow-up periods ranging from 3 to 24 months. All factors were assessed before surgery—typically within the week prior to the procedure—using a combination of patient-reported outcome measures (PROMs) and objective assessments administered to children, adolescents, and their parents (Table 2).

Most prognostic factors were evaluated in only one or two cohorts (61%), reflecting limited replication across studies (Table 2). Ten factors—including age, sex, race, regional anesthesia, surgical duration, baseline pain intensity, child pain catastrophizing, child anxiety, and parent pain catastrophizing—were assessed in six or more cohorts. Only three factors (age, sex, and baseline pain intensity) were examined by ten or more cohorts, providing sufficient evidence for more robust synthesis.

Table 2 Prognostic Factors Assessment, Statistical Analysis and results by Studies

Study	Population	Baseline Assessment Timing	Baseline Risk Factors Assessed	Statistical Approach	Main Findings
Ceniza-Bordallo et al, 2025[3]	Children and adolescents	The day before surgery	**Child**: Pain intensity (NRS); pain catastrophizing (PCS-C); pain anxiety (CPASS); fear of pain (FOPQ-C); kinesiophobia (TSK-11); quality of life (PedsQL); pain interference (PROMIS-PPI); physical activity (NRS-PA)	Multivariable logistic regression; Group-based trajectory modeling (GBTM)	Higher presurgical pain intensity, pain catastrophizing, and pain anxiety were associated with CPSP at 3 months. Three pain trajectories were identified; presurgical psychological factors differentiated trajectory membership, except physical activity.
Sieberg et al, 2023[42]	Children and adolescents	Not specified	**Child**: Pain intensity (NRS); pain catastrophizing (PCS-C); quantitative sensory testing (pressure pain threshold, mechanical detection and pain thresholds)	One-way ANOVA; regression analyses	Higher presurgical pain sensitivity and pain intensity predicted CPSP. Preoperative helplessness was the most sensitive psychological predictor of CPSP.
Ellyson et al, 2022[46]	Adolescents	Week prior to surgery	**Child**: Age; pain intensity (NRS); anxiety and depressive symptoms (RCADS); sleep quality (ASWS); HRQoL (PedsQL); fatigue. **Parent**: Pain catastrophizing (PCS-P)	Group-based trajectory modeling; logistic and multivariable regression	Two pain trajectories were identified. Higher baseline pain predicted persistent pain; better sleep quality reduced the likelihood of persistent pain.
Bailey et al, 2021[60]	Children and adolescents	Not specified	**Child**: Age; sex; pain catastrophizing (PCS-C); anxiety (STAIC). **Parent**: Pain catastrophizing (PCS-P); anxiety (STAIP-P)	LCGA/LGMM; multinomial logistic regression; MANOVA	Baseline pain and child trait anxiety predicted membership in persistent pain trajectories. Persistent trajectories were associated with poorer functional outcomes.
Chidambaran et al, 2021[49]	Adolescents	Not specified	**Child**: Age; sex; race; weight; pain intensity (NRS); anxiety sensitivity (CASI); surgical duration. **Biological**: genetic and DNA methylation markers	Logistic regression; genetic/ epigenetic association models	CPSP was associated with specific genetic and epigenetic markers; preoperative pain was higher in the CPSP group.

(*Continued*)

Table 2 (Continued).

Study	Population	Baseline Assessment Timing	Baseline Risk Factors Assessed	Statistical Approach	Main Findings
Rabbitts et al, 2021[45]	Adolescents	Week before surgery	**Child**: Age; sex; physical activity (actigraphy); pain catastrophizing (PCS-C)	Multivariable logistic regression	Lower physical activity at 2 weeks post-surgery predicted CPSP at 4 months, independent of baseline psychosocial factors.
Palabiyik et al, 2021[58]	Children and adolescents	Not specified	**Child**: Age; sex; pain intensity (NRS); HRQoL (PedsQL). **Parent**: Parent-proxy HRQoL	Logistic regression	Female sex and scar length were associated with CPSP; children with CPSP reported poorer HRQoL.
Rabbitts et al, 2020[14]	Adolescents	1 week before surgery	**Child**: Pain intensity; pain distribution; anxiety and depressive symptoms (RCADS); sleep quality (ASWS); insomnia (ISI). **Parent**: Pain catastrophizing (PCS-P); family functioning (FAD)	Sequential multivariable logistic regression	Depressive symptoms and poor sleep quality were independently associated with CPSP at 4 months.
Perry et al, 2020[47]	Children and adolescents	Not specified	**Child**: Pain intensity; pain catastrophizing; anxiety; gene expression	Logistic regression; gene expression analysis	Higher baseline pain intensity and helplessness predicted persistent postsurgical pain.
Ocay et al, 2020[61]	Adolescents	7–10 days before surgery	**Child**: Pain catastrophizing (PCS-C); scoliosis-related clinical factors	Growth mixture modeling; GLM	Pain catastrophizing and preoperative pain severity predicted acute pain trajectories, which in turn predicted long-term pain outcomes.
Noel et al, 2019[62]	Children and adolescents	Not specified	**Child**: Pain intensity (rest and movement); anxiety sensitivity; pain catastrophizing; pain anxiety	Hierarchical regression; dominance analysis	Anxiety sensitivity and pain catastrophizing predicted negatively biased pain memories, which were associated with CPSP.
Rosenbloom et al, 2019[63]	Adolescents	Not specified	**Child**: Pain intensity; anxiety; depression; catastrophizing; self-efficacy; fear of movement. **Parent**: Multiple pain-related psychological factors	GMM; logistic regression	Higher baseline functional disability and psychological distress predicted high pain trajectories and later disability.
Chidambaran et al, 2019[50]	Adolescents	Not specified	**Child**: Pain intensity; anxiety sensitivity; perioperative analgesic exposure. **Parent**: Anxiety; catastrophizing	DNA methylation and regression analyses	DNA methylation patterns and anxiety sensitivity were associated with CPSP.
Li et al, 2019[64]	Adolescents	7–10 days before surgery	**Child**: Scoliosis outcomes; pain medication use	Growth mixture modeling	Acute opioid consumption trajectories were not associated with long-term pain outcomes.
Knudsen et al, 2018[59]	Adolescents	1 day before surgery	**Child**: Pain intensity (SF-MPQ); HRQoL; QST	Paired t-tests; chi-square	Sensory disturbances persisted in a subset of adolescents with ongoing pain.

(*Continued*)

Table 2 (Continued).

Study	Population	Baseline Assessment Timing	Baseline Risk Factors Assessed	Statistical Approach	Main Findings
Voepel-Lewis et al, 2018[68]	Adolescents	2 weeks before surgery	**Child**: Pain intensity; widespread pain; fatigue; anxiety; depression; pain interference	Cluster analysis; multivariable regression	A high-symptom cluster predicted worse pain interference and continued analgesic use at 1 year.
Chidambaran et al, 2017[52]	Adolescents	Day of surgery	**Child**: Pain intensity; anxiety; catastrophizing; opioid exposure. **Parent**: Anxiety; catastrophizing	Multivariable regression	Higher perioperative opioid consumption and preoperative pain predicted CPSP.
Julien-Marsollier et al, 2017[57]	Adolescents	During hospital stay	**Child**: Pain intensity; preoperative pain history; morphine consumption	Logistic regression	Preoperative pain and early postoperative opioid use predicted neuropathic CPSP.
Noel et al, 2017[62]	Children and adolescents	Week prior to surgery	**Child**: Pain intensity; pain catastrophizing. **Parent**: Pain catastrophizing	Actor-Partner Interdependence Model	Children's pain memories, but not parents', predicted CPSP.
Batoz et al, 2016[67]	Children and adolescents	1 day before surgery	**Child**: Pre-existing pain; recent pain; surgery characteristics	Logistic regression	Recent preoperative pain was independently associated with CPSP.
Rabbitts et al, 2015[69]	Children and adolescents	Pre-surgery at home	**Child**: Pain intensity; catastrophizing; HRQoL; activity limitations. **Parent**: Pain catastrophizing	GBTM; logistic regression	Parental catastrophizing predicted late pain recovery trajectories.
Sieberg et al, 2013[39]	Adolescents and young adults	1–3 weeks before surgery	**Child/young adult**: Pain intensity; mental health; self-image; school/work absence	Trajectory modeling	Distinct long-term pain trajectories were identified; baseline pain and psychological functioning differentiated groups.
Bastrom et al, 2013[54]	Adolescents	Not specified	**Child**: Sex; surgical approach; HRQoL (SRS-22)	ANOVA	Lower preoperative pain scores increased the likelihood of unexplained postoperative pain.
Pagé & Stinson et al, 2013[66]	Children and adolescents	48–72 hours after surgery	**Child**: Pain intensity; pain unpleasantness; anxiety sensitivity; catastrophizing	GEE; logistic regression	Early postoperative pain intensity and unpleasantness predicted CPSP.
Landman et al, 2011[55]	Adolescents and young adults	Not specified	**Child/young adult**: Pain intensity; anxiety; depressive symptoms; body image	Regression analyses	Higher presurgical pain and poorer body image predicted persistent pain.

Abbreviations: CPSP, chronic postsurgical pain; PPP, persistent postsurgical pain; APSP, acute postsurgical pain; NRS, Numeric Rating Scale; VAS, Visual Analog Scale; PCS-C, Pain Catastrophizing Scale–Child version; PCS-P, Pain Catastrophizing Scale–Parent version; CPASS, Child Pain Anxiety Symptoms Scale; CASI, Childhood Anxiety Sensitivity Index; FOPQ-C, Fear of Pain Questionnaire–Child version; TSK, Tampa Scale of Kinesiophobia; HRQoL, health-related quality of life; PedsQL, Pediatric Quality of Life Inventory; PROMIS, Patient-Reported Outcomes Measurement Information System; QST, quantitative sensory testing; PPT, pressure pain threshold; MDT, mechanical detection threshold; GBTM, group-based trajectory modeling; LCGA, latent class growth analysis; GMM, growth mixture modeling; LGMM, latent growth mixture modeling; GEE, generalized estimating equations; ANOVA, analysis of variance; MANOVA, multivariate analysis of variance; OR, odds ratio; aOR, adjusted odds ratio; CI, confidence interval.

Sociodemographic

Age

Age was the most frequently examined sociodemographic factor, assessed in 19 studies (n = 2,757).[3,39,43–49,51,52,55–58,60,63,67] Most studies (15/19) found no association between age and CPSP,[44,46–49,51,52,55–58,60,63,67] although four[3,39,43,45] reported

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a significant relationship, indicating limited consistency of evidence across studies. Findings varied due to differences in outcome definitions (binary CPSP, pain trajectories, symptom clusters), follow-up periods, and analytical approaches.

When examining analytical approaches separately, among the 13 studies using univariate analyses, 10 reported no association,[47–49,51,52,55–58,67] while 3 found that older age increased CPSP risk.[39,45,50] Five studies used multivariate models,[43,44,46,60,63] and four found no independent association between age and CPSP.[44,46,60,63] One study[43] reported a modest increased risk at 3 months (OR = 1.46, 95% CI: 1.10–1.94).

A meta-analysis including studies with dichotomous CPSP outcomes showed non-significant associations were observed at 3, 6 or 12 months. Heterogeneity was low across all time points (see Figure 3 and Supplementary Document III).

Overall, results across studies were inconsistent, with generally small effect sizes. The certainty of the evidence was rated as moderate due to indirectness and limited consistency (Table 3).

Sex

Sex was examined as a potential prognostic factor in 15 studies (n = 2,626)[43–45,47–51,54–56,60,63,67] and none reported a significant association with CPSP at any follow-up point.

A random-effects meta-analysis including six studies at 3 months showed no increased risk for girls compared with boys (OR = 1.08, 95% CI: 0.69–1.69, p =0.74),[43,45,47,51,52,67] with low heterogeneity (I^2 = 0%) (See Figure 4 and Supplementary Document III).

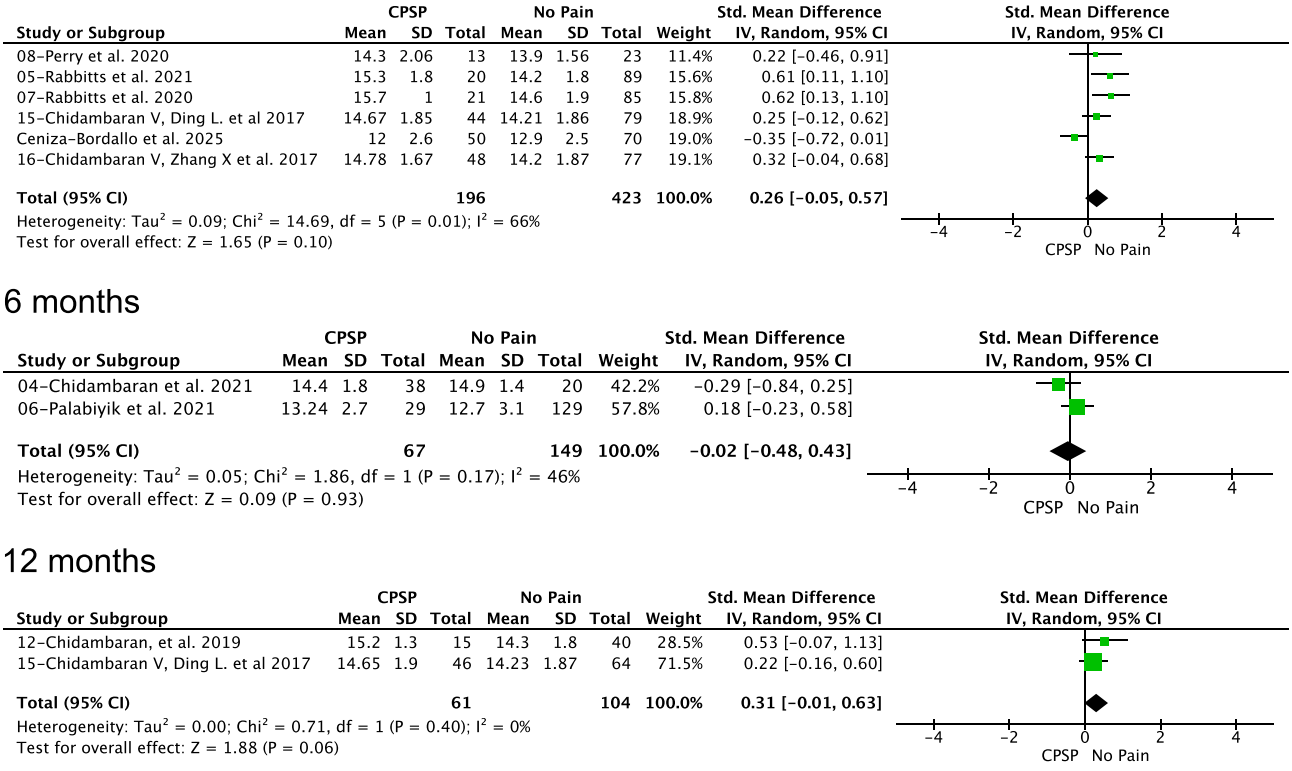

Figure 3 Differences in age between patients who developed chronic postsurgical pain (CPSP) and those without pain at 3, 6, and 12 months after surgery Forest plots showing standardized mean differences (SMD) in age between individuals who developed chronic postsurgical pain (CPSP) and those who did not report pain at 3, 6, and 12 months after surgery. Random-effects meta-analyses are presented separately for each follow-up period. Positive values indicate older age in the CPSP group compared with the no-pain group. Squares represent individual study effect sizes weighted by sample size, horizontal lines indicate 95% confidence intervals, and diamonds represent pooled effect estimates. Between-study heterogeneity is reported using the I^2 statistic.

Table 3 GRADE Assessment in Prognostics Factor to CPSP

					Univariante			Multivariante			GRADE						
Factor type	**Prognostic factor**	**Number of studies**	**Studies**	**Sample**	**+**	**0**	**–**	**+**	**0**	**–**	**Phase**	**Study limitation**	**Inconsistency**	**Indirect**	**Imprecision**	**Publication bias**	**Level of Evidence**
Sociodemographic	**Age**	19	Ceniza-Bordallo, 2025 [3] Ellyson, 2022 [46] Bailey, 2021 [60] Chidambaran, 2021 [49] Rabbitts, 2021 [45] Palabiyik, 2021 [58] Perry, 2020 [47] Rabbitts, 2020 [14] Rosenbloom, 2019 [63] Chidambaran, 2019 [50] Voepel-Lewis, 2018 [68] Chidambaran, Ding, 2017 [52] Chidambaran, Zhang, 2017 [51] Julien-Marsollier, 2017 [57] Batoz, 2016 [67] Rabbitts, 2015 [69] Sieberg, 2013 [39] Landman, 2011 [55] Fortier, 2011 [56]	2757	3	10	0	1	5	0	2	✓	✓	✗	✓	✓	+++

(*Continued*)

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Table 3 (Continued).

					Univariante			Multivariante			GRADE						
Factor type	**Prognostic factor**	**Number of studies**	**Studies**	**Sample**	**+**	**0**	**–**	**+**	**0**	**–**	**Phase**	**Study limitation**	**Inconsistency**	**Indirect**	**Imprecision**	**Publication bias**	**Level of Evidence**
	Sex	15	Ceniza-Bordallo, 2025 [3] Bailey, 2021 [60] Chidambaran, 2021 [49] Rabbitts, 2021 [45] Rabbitts, 2020 [1] Perry, 2020 [47] Rosenbloom, 2019 [63] Chidambaran, 2019 [50] Voepel-Lewis, 2018 [68] Chidambaran, Zhang, 2017 [51] Batoz, 2016 [67] Rabbitts, 2015 [69] Bastrom, 2013 [54] Landman, 2011 [55] Fortier, 2011 [56]	2626	0	10	0	0	5	0	2	✓	✓	✓	✓	✓	++++

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	Race (White vs non-white)	6	Chidambaran, 2021 [49] Perry, 2020 [47] Chidambaran, 2019 [50] Chidambaran, Zhang, 2017 [51] Chidambaran, Ding, 2017 [52] Fortier, 2011 [56]	573	0	5	0	0	1	0	1and2	✓	✓	✗	✓	✓	+++
	Hispanic Ethnicity	1	Fortier, 2011 [56]	113	1	0	0	0	0	0	1	✗	Not applicable	✓	✓	✓	++
Body measurement factors	**Higher Body mass index**	2	Voepel-Lewis, 2018 [68] Landman, 2011[55]	390	0	1	0	0	1	0	1	✓	✓	✗	✓	✓	++
	Weight	5	Chidambaran, 2021 [49] Chidambaran, 2019 [50] Chidambaran, Ding, 2017 [52] Chidambaran, Zhang, 2017 [51] Julien-Marsollier, 2017 [57]	460	0	5	0	0	0	0	1and2	✓	✓	✓	✗	✓	+++

(*Continued*)

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Table 3 (Continued).

					Univariante			Multivariante			GRADE						
Factor type	**Prognostic factor**	**Number of studies**	**Studies**	**Sample**	**+**	**0**	**–**	**+**	**0**	**–**	**Phase**	**Study limitation**	**Inconsistency**	**Indirect**	**Imprecision**	**Publication bias**	**Level of Evidence**
Surgery characteristics	**Regional analgesia in surgery**	6	Ocay, 2020 [61] Rosenbloom, 2019 [63] Voepel-Lewis, 2018 [68] Chidambaran, Ding, 2017 [52] Julien-Marsollier, 2017 [57] Batoz, 2016 [67]	937	0	4	0	0	2	0	1and2	✓	✓	✓	✓	✓	++++
	Surgical duration	6	Chidambaran, 2021 [49] Ocay, 2020 [61] Chidambaran, 2019 [50] Chidambaran, Ding, 2017 [52] Chidambaran, Zhang, 2017 [51] Julien-Marsollier, 2017 [57]	566	0	5	0	1	0	0	1and2	✓	✗	✓	✗	✓	++
	Surgery type	4	Rosenbloom, 2019 [63] Batoz, 2016 [67] Pagé, Stinson et al, 2013 [66] Fortier, 2011 [56]	752	0	2	0	1	1	0	1and2	✓	✓	✗	✓	✓	+++

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Pain characteristics	**Pain intensity**	14 (13 samples)	Ceniza-Bordallo, 2025 [3] Sieberg, 2023 [42] Ellyson, 2022 [46] Chidambaran, 2021 [49] Perry, 2020 [47] Chidambaran, 2019 [50] Voepel-Lewis, 2018 [68] Chidambaran, Ding, 2017 [52] Chidambaran, Zhang, 2017 [51] Noel, 2017 [62] Batoz, 2016 [67] Rabbitts, 2015 [69] Pagé, Stinson et al, 2013 [66] Pagé, Campbell et al, 2013 [65]	1347	2	0	0	9	3	0	2and3	✓	✓	✓	✓	✓	++++
	Quantitative sensory testing	1	Sieberg, 2023 [42]	32	1	0	0	0	0	0	1	✗	Not applicable	✗	✓	✓	+
Psychological factors	**Pain catastrophizing**	9	Ceniza-Bordallo, 2025 [3] Sieberg, 2023 [42] Bailey, 2021 [60] Rabbitts, 2021 [45] Rabbitts, 2020 [14] Perry, 2020 [47] Ocay, 2020 [61] Chidambaran, Ding, 2017 [52] Rabbitts, 2015 [69]	1032	1	2	0	2	4	0	2and3	✓	✗	✓	✓	✓	++++

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Table 3 (Continued).

					Univariante			Multivariante			GRADE						
Factor type	**Prognostic factor**	**Number of studies**	**Studies**	**Sample**	**+**	**0**	**–**	**+**	**0**	**–**	**Phase**	**Study limitation**	**Inconsistency**	**Indirect**	**Imprecision**	**Publication bias**	**Level of Evidence**
	Anxiety	9	Ceniza-Bordallo, 2025 [3] Ellyson, 2022 [46] Bailey, 2021 [60] Rabbitts, 2020 [14] Rosenbloom, 2019 [63] Chidambaran, 2019 [50] Voepel-Lewis, 2018 [68] Chidambaran, Ding, 2017 [52] Chidambaran, Zhang, 2017 [51]	1230	1	4	0	1	3	0	2	✓	✓	✓	✓	✓	++++
	Anxiety sensitivity	4	Chidambaran, 2021 [49] Rosenbloom, 2019 [63] Chidambaran, 2019 [50] Chidambaran, Ding, 2017 [52]	556	0	3	0	1	0	0	2	✓	✓	✗	✓	✗	++
	Depression	1	Rosenbloom, 2019 [63]	237	0	1	0	0	0	0	1	✗	Not applicable	✓	✓	✓	++
	Depressive symptoms	3	Ellyson, 2022 [46] Rabbitts, 2020 [14] Voepel-Lewis, 2018 [68]	331	1	0	0	1	1	0	1and2	✓	✓	✓	✓	✓	+++

	Sleep quality	2	Ellyson, 2022 [46] Rabbitts, 2020 [14]	236	0	0	0	0	0	2	1and2	✓	✓	✓	✓	✓	+++
	Insomnia severity	1	Rabbitts, 2020 [14]	119	0	0	0	0	1	0	1	✗	Not applicable	✓	✓	✓	++
	Fatigue	1	Voepel-Lewis, 2018[68]	95	1	0	0	0	0	0	1	✗	Not applicable	✓	✓	✓	++
	Fear of movement	2	Ceniza-Bordallo, 2025 [3] Rosenbloom, 2019 [63]	394	0	1	0	0	1	0	1	✗	Not applicable	✓	✓	✓	++
	Self-efficacy	1	Rosenbloom, 2019 [63]	237	0	0	0	0	1	0	1	✗	Not applicable	✓	✓	✓	++
	Post-traumatic stress	1	Rosenbloom, 2019 [63]	237	0	0	0	0	1	0	1	✗	Not applicable	✓	✓	✓	++
	Chronic pain acceptance	1	Rosenbloom, 2019 [63]	237	0	1	0	0	0	1	1	✗	Not applicable	✓	✓	✓	++
	Fear of pain	1	Ceniza-Bordallo, 2025 [3]	159	0	0	0	0	1	0	1	✗	Not applicable	✓	✓	✓	++
School functioning	**Absence of school**	1	Sieberg, 2013 [39]	190	0	0	0	1	0	0	1	✗	Not applicable	✓	✓	✓	++
Physical functioning	**Functional disability**	1	Rosenbloom, 2019 [63]	237	0	0	0	1	0	0	1	✗	Not applicable	✓	✓	✓	++
	Health-related quality of life	2	Ceniza-Bordallo, 2025 [3] Palabiyik, 2021 [58]	317	1	0	0	0	1	0	1	✗	Not applicable	✓	✓	✓	++
Pain impact	**Pain Interference**	2	Ceniza-Bordallo, 2025 [3] Voepel-Lewis, 2018 [6]	254	1	0	0	0	1	0	1	✗	Not applicable	✓	✓	✓	++

(*Continued*)

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Table 3 (Continued).

					Univariante			Multivariante			GRADE						
Factor type	**Prognostic factor**	**Number of studies**	**Studies**	**Sample**	**+**	**0**	**–**	**+**	**0**	**–**	**Phase**	**Study limitation**	**Inconsistency**	**Indirect**	**Imprecision**	**Publication bias**	**Level of Evidence**
Gene expression	**Genetic & DNA methylation association**	3	Chidambaran, 2021 [49] Chidambaran, Ding, 2017 [52] Chidambaran, Zhang, 2017 [51]	264	0	0	0	3	0	0	I	✗	✓	✓	✗	✓	++
	Gene Expression	I	Perry, 2020 [47]	36	0	0	0	I	0	0	I	✗	Not applicable	✗	✓	✓	+
Child symptoms reported by parent	**Child health-related quality of life reported by parent**	I	Palabiyik, 2021 [59]	158	I	0	0	0	0	0	I	✗	Not applicable	✓	✓	✓	++
Parent symptoms when children are in pain	**Parent pain catastrophizing**	7	Ellyson, 2022 [46] Bailey, 2021 [60] Rabbitts, 2020 [14] Chidambaran, 2019 [50] Chidambaran, Ding, 2017 [52] Noel, 2017 [62] Rabbitts, 2015 [69]	1119	0	5	0	0	2	0	2and3	✓	✓	✓	✓	✓	++++

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Parent symptoms	**Parent pain anxiety**	4	Rosenbloom, 2019 [63] Chidambaran, 2019 [50] Chidambaran, Ding, 2017 [52] Chidambaran, Zhang, 2017 [51]	587	0	4	0	0	0	0	2and3	✓	✓	✓	✓	✓	++++
	Parent anxiety sensitivity	1	Rosenbloom, 2019 [63]	237	0	1	0	0	0	0	1	✗	Not applicable	✓	✓	✓	++
	Parent depression	1	Rosenbloom, 2019 [63]	237	0	1	0	0	0	0	1	✗	Not applicable	✓	✓	✓	++
Parent medical history	**Parent Pain history**	1	Chidambaran, Ding, 2017 [52]	144	0	0	0	0	1	0	1	✗	Not applicable	✓	✓	✓	++
Family impact due to child pain	**Family functioning**	1	Rabbitts, 2020 [14]	119	0	0	0	0	1	0	1	✗	Not applicable	✓	✓	✓	++

Notes: ✓ = no serious limitations, ✗ = serious limitations, ✗✗ = very serious limitations, For overall quality of evidence: + indicates very low; ++, low; +++, moderate; and ++++, high.

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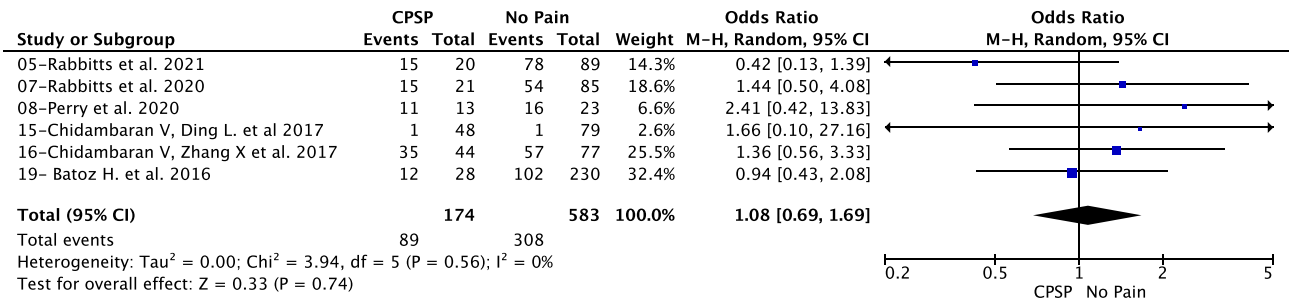


Figure 4 Association between sex and the development of chronic postsurgical pain (CPSP) at 3 months after surgery Forest plot showing odds ratios (OR) for the association between sex and the development of chronic postsurgical pain at 3 months after surgery. Random-effects meta-analysis was conducted using the Mantel–Haenszel method. Odds ratios greater than 1 indicate a higher likelihood of CPSP in females compared with males. Squares represent individual study effect estimates weighted by sample size, horizontal lines indicate 95% confidence intervals, and the diamond represents the pooled effect estimate. Between-study heterogeneity is reported using the I^2 statistic.

Findings from both univariate and multivariate analyses across studies consistently indicated that sex did not predict CPSP, pain trajectories, symptom clusters, functional disability, or health-related quality of life. Given the consistency of results and the robustness of the available data, the certainty of evidence was rated as high (Table 3).

Race

Five studies evaluated race as a potential prognostic factor for CPSP using univariate analyses,[47,49–51,56] and none reported a significant association between White race and CPSP at any follow-up point. One additional study using a multivariate model also found no independent association.[52] These results were derived from individual study analyses because the small number of studies and heterogeneity in outcome definitions precluded meta-analysis. Overall, findings across studies consistently indicated no association between race and CPSP.

However, the available evidence is limited by the marked underrepresentation of non-White participants, with minority groups typically comprising only 2–10% of study samples. This lack of diversity restricts the ability to draw reliable conclusions regarding racial differences. Therefore, the certainty of evidence for race as a prognostic factor was rated as moderate due to indirectness (see Table 3).

Surgery Characteristics

Surgical Duration

Six studies evaluated surgical duration as a potential prognostic factor for CPSP. Five univariate analyses found no association between longer surgical duration and CPSP at any follow-up point.[49–51,57,61] One multivariate model reported a modest increased risk at 2–3 months (OR = 2.16, 95% CI: 1.17–4.00).[52] Interpretation of these findings is limited by substantial heterogeneity in CPSP definitions, follow-up times, and small sample sizes across studies. Accordingly, the certainty of the evidence for surgical duration as a prognostic factor was rated as low (Table 3).

Medical Factors

Pain Intensity in CPSP

Baseline pain intensity was examined in 13 studies.[42,44,46–53,66,67] The meta-analysis showed that individuals who developed CPSP had higher baseline pain intensity at 3 months (SMD = 0.56, 95% CI: 0.23–0.90), with moderate heterogeneity (I^2 = 66%). At 6 months, a significant association was also observed (SMD = 3.32), although heterogeneity was extremely high (I^2 = 98%), limiting interpretability. At 12 months, no significant association was found between baseline pain intensity and CPSP (p= 0.06), and heterogeneity remained high (see Figure 5 and Supplementary Document III).

Overall, baseline pain intensity appears to be a consistent short-term predictor of CPSP, particularly within the first 6 months after surgery. The certainty of evidence was rated as high (Table 3).

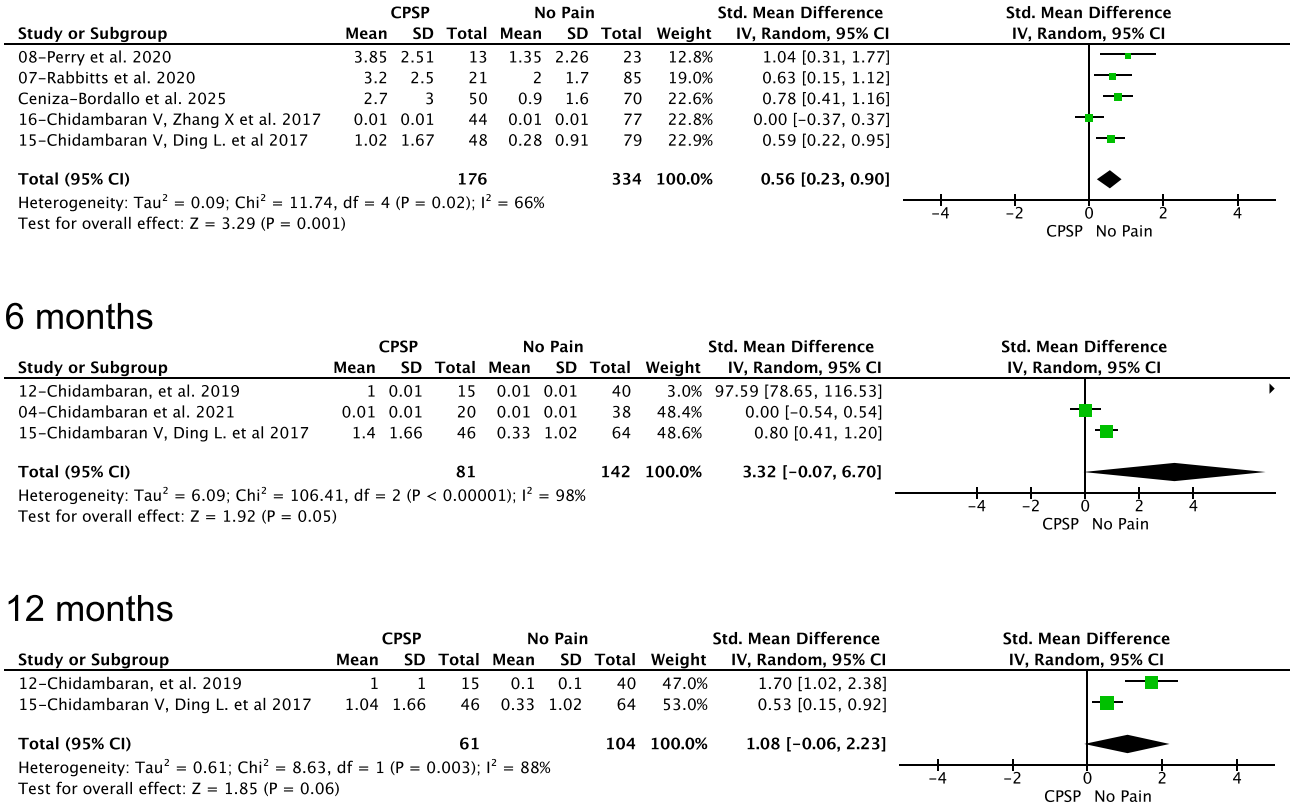


Figure 5 Differences in pain intensity between patients who developed chronic postsurgical pain and those without pain at 3, 6, and 12 months after surgery Forest plots showing standardized mean differences (SMD) in pain intensity between children, adolescents and young adults who developed chronic postsurgical pain and those who did not report pain at 3, 6, and 12 months after surgery. Random-effects meta-analyses are presented separately for each follow-up period. Positive values indicate higher pain intensity in the CPSP group compared with the no-pain group. Squares represent individual study effect sizes weighted by sample size, horizontal lines indicate 95% confidence intervals, and diamonds represent pooled effect estimates. Between-study heterogeneity is reported using the I^2 statistic.

Psychological Factors

Child Pain Catastrophizing in CPSP

Nine studies (n = 1,032) evaluated child pain catastrophizing as a potential prognostic factor for CPSP.[3,42–45,47,52,60,61] Three studies reported a significant association,[3,42,61] whereas six found no relationship, including all four multivariate analyses.[43–45,47,52,60]

Noticing this inconsistency in the literature, we conducted a subgroup analysis. Due to the heterogeneity of follow-ups, CPSP criteria, and sample sizes, we could only perform a random effects meta-analysis of SMD comparing child pain catastrophizing between children and adolescents who developed CPSP and those who did not. We used CPSP as a binary variable and only had data to conduct the analysis at the 3-month follow-up. The results of the analysis, based on data from 5 studies[3,43,45,47,52] (n= 537 participants) revealed a non-significative SMD between participants who develop CPSP and who not develop CPSP at 3 months (CI 0.17–1.57) (see Figure 6 and Supplementary Document III). Heterogeneity among the studies, was high (Tau^2 =0.56, Chi^2= 42.29, p<*0.001*, I^2=91%).

Overall, findings across studies were inconsistent, and no clear evidence supports child pain catastrophizing as an independent predictor of CPSP. The certainty of the evidence was rated as moderate due to inconsistency (Table 3).

Child Sensitivity to Anxiety in CPSP

Four studies (n = 556) evaluated child sensitivity to anxiety as a potential prognostic factor for CPSP.[49,50,52,63] Three studies,[49,50,63] all using univariate analyses, found no association between higher anxiety sensitivity and CPSP at follow-

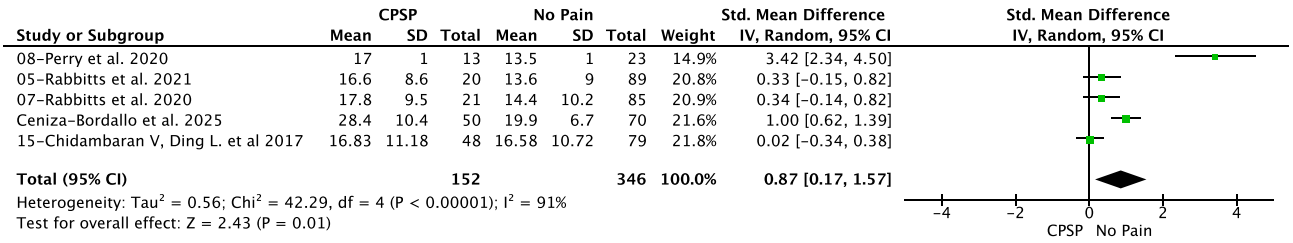


Figure 6 Differences in child pain catastrophizing between patients who developed chronic postsurgical pain (CPSP) and those without pain at 3 months after surgery Forest plot showing standardized mean differences (SMD) in child pain catastrophizing between children, adolescents and young adults who developed chronic postsurgical pain and those who did not report pain at 3 months after surgery. A random-effects meta-analysis was conducted. Positive values indicate higher levels of pain catastrophizing in the CPSP group compared with the no-pain group. Squares represent individual study effect sizes weighted by sample size, horizontal lines indicate 95% confidence intervals, and the diamond represents the pooled effect estimate. Between-study heterogeneity is reported using the I^2 statistic.

up periods ranging from 4 to 12 months. One study reported that greater anxiety sensitivity increased the risk of CPSP at 12 months (aOR = 1.24, 95% CI: 1.09–1.42).[52]

Because findings were inconsistent, we conducted a random-effects meta-analysis including only studies that assessed anxiety sensitivity using the Child and Adolescent Symptom Inventory (CASI). At 3 months, pooled data from two studies (n = 248)[51,52] showed no significant difference between children who developed CPSP and those who did not (p =0.42), with low heterogeneity (I^2 = 0%). At 12 months, data from three studies (n = 235)[49,50,52] indicated a significant difference in anxiety sensitivity between groups (p <0.01), with moderate heterogeneity (I^2 = 55%) (see Figure 7 and Supplementary Document III).

Overall, the evidence for child anxiety sensitivity as a prognostic factor for CPSP remains limited and inconsistent. The certainty of the evidence was rated as low due to indirectness and variability across studies (Table 3).

Child Anxiety in CPSP

Nine studies (n = 1,230) evaluated child pain anxiety as a potential prognostic factor for CPSP.[3,43,46,48,50–52,60,63] Two studies[3,48] reported higher presurgical pain anxiety among children who later developed CPSP, whereas seven studies—including all multivariate analyses—found no association.

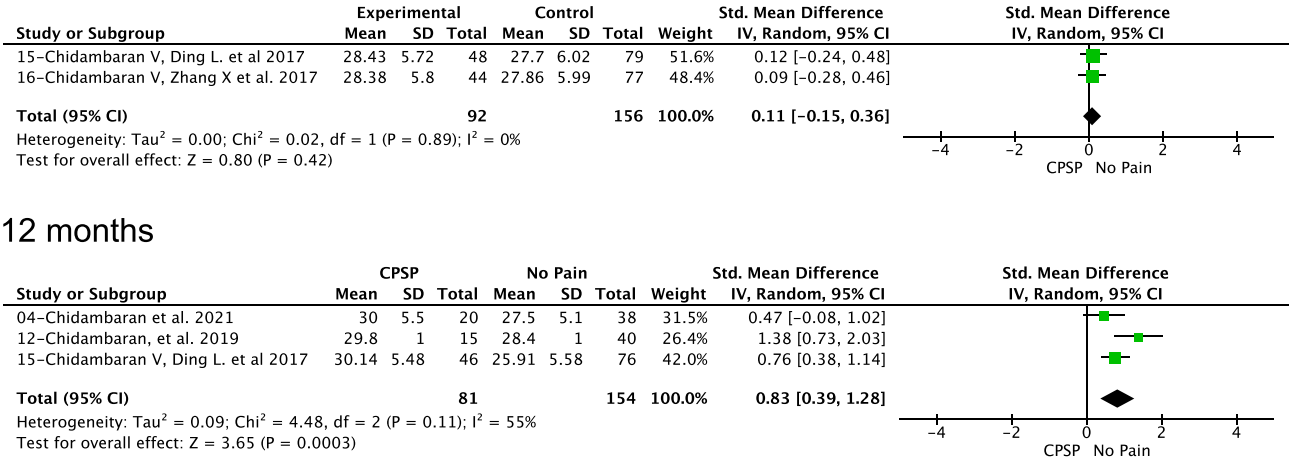


Figure 7 Differences in child anxiety sensitivity between patients who developed chronic postsurgical pain and those without pain at 3 and 12 months after surgery Forest plot showing standardized mean differences (SMD) in child pain catastrophizing between children, adolescents and young adults who developed chronic postsurgical pain and those who did not report pain at 3 months after surgery. A random-effects meta-analysis was conducted. Positive values indicate higher levels of pain catastrophizing in the CPSP group compared with the no-pain group. Squares represent individual study effect sizes weighted by sample size, horizontal lines indicate 95% confidence intervals, and the diamond represents the pooled effect estimate. Between-study heterogeneity is reported using the I^2 statistic.

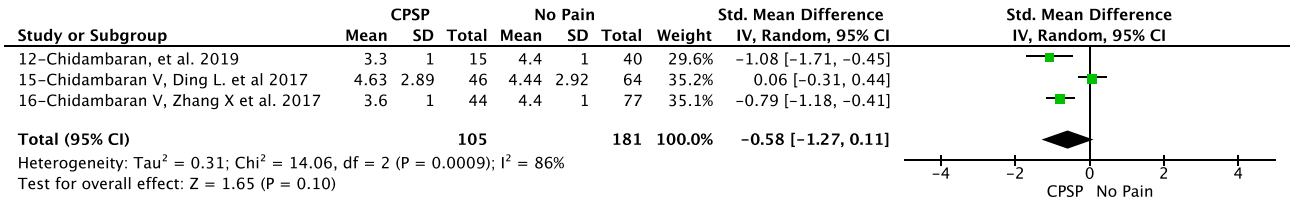


Figure 8 Differences in child pain anxiety between patients who developed chronic postsurgical pain (CPSP) and those without pain at 12 months after surgery Forest plot showing standardized mean differences (SMD) in child pain anxiety between children, adolescents and young adults who developed chronic postsurgical pain (CPSP) and those who did not report pain at 12 months after surgery. A random-effects meta-analysis was conducted. Negative values indicate lower levels of pain anxiety in the CPSP group compared with the no-pain group. Squares represent individual study effect sizes weighted by sample size, horizontal lines indicate 95% confidence intervals, and the diamond represents the pooled effect estimate. Between-study heterogeneity is reported using the I^2 statistic.

A meta-analysis could be performed only for the 12-month follow-up using binary CPSP outcomes. Based on three studies (n = 286),[50–52] the pooled results showed no significant difference in pain anxiety between children who developed CPSP and those who did not (p =0.10), with high heterogeneity (I^2 = 86%; see Figure 8).

Taken together, current evidence does not support child pain anxiety as a reliable prognostic factor for CPSP. The certainty of the evidence was rated as moderate, given the predominance of studies reporting null findings (Table 3).

Genetic & DNA Methylation Association

Three studies investigated genetic and epigenetic contributors to CPSP development.

The first study[51] (n = 133 adolescents undergoing spinal fusion) examined DNA methylation in the promoter region of the μ-opioid receptor gene (OPRM1). Higher methylation levels at multiple CpG sites—particularly CpG13 and CpG22—were significantly associated with an increased risk of CPSP after adjusting for preoperative pain and postoperative morphine consumption (p <0.05). These findings suggest that methylation-dependent downregulation of OPRM1 expression may reduce opioid system efficacy and increase vulnerability to persistent postsurgical pain.

The second study by Chidambaran et al[50] identified 637 differentially methylated positions (DMPs) associated with CPSP (p <0.05). Key enriched pathways included GABA receptor hypofunction, indicating altered inhibitory neurotransmission, and dopamine–DARPP32 feedback in cAMP signaling, a pathway implicated in emotional modulation, reward processing, and pain persistence. These results point to widespread epigenetic alterations potentially involved in the transition from acute to chronic postsurgical pain.

A third study[49] investigated genetic–epigenetic interactions by examining methylation quantitative trait loci (meQTLs). A total of 2,753 meQTLs were identified, associated with methylation levels at 480 CpG sites, 127 of which mediated relationships between 470 single nucleotide polymorphisms (SNPs) and CPSP (p <0.05). Notable associations included meQTLs in the PARK16 locus, as well as variants in PM20D1 (rs960603; OR 4.87) and RAB29 (rs708723; OR 3.19), both significantly associated with CPSP risk. These findings suggest that non-coding genomic variation may influence CPSP susceptibility through methylation-dependent regulatory mechanisms.

Although these studies provide compelling preliminary evidence linking genetic and epigenetic factors to CPSP risk, they are limited by small sample sizes, observational designs, and lack of replication. Consequently, the certainty of the evidence was rated as low (Table 3). Nonetheless, these early-phase findings highlight promising biological pathways warranting further investigation in larger, rigorously designed studies.

Parent Symptoms When Children are in Pain

Parent Pain Catastrophizing in CPSP

Seven studies (n = 1,119) examined parent pain catastrophizing as a potential prognostic factor for CPSP.[43,44,46,50,52,53,60] A meta-analysis could be conducted only for the 3-month follow-up using binary CPSP outcomes. Based on two studies (n = 233),[43,52] the pooled results showed a non-significant difference in parent catastrophizing between children who

developed CPSP and those who did not (p =0.41), with moderate heterogeneity (I^2 = 66%; see Figure 9 and Supplementary Document III).

Taken together, the evidence does not support parent pain catastrophizing as a prognostic factor for CPSP. The certainty of the evidence was rated as moderate, given the consistent null findings across studies (Table 3).

Parent Pain Anxiety in CPSP

Four studies (n = 587) evaluated parent pain anxiety as a potential prognostic factor for CPSP.[50–52,63] A meta-analysis could be performed for the 3-month and 12-month follow-ups, including only studies that assessed parent pain anxiety using the VAS. Pooled results from two studies at 3 months (n = 248)[51,52] and two studies at 12 months (n = 165)[50,52] showed no significant differences between parents of children who developed CPSP and those who did not (3 months: p =0.35; 12 months: p =0.26). Heterogeneity was high at both time points (I^2 = 73% and 96%, respectively; see Figure 10 and Supplementary Document III).

Overall, available evidence does not support parent pain anxiety as a prognostic factor for CPSP. The certainty of the evidence was rated as moderate, given the consistent null findings across studies (Table 3).

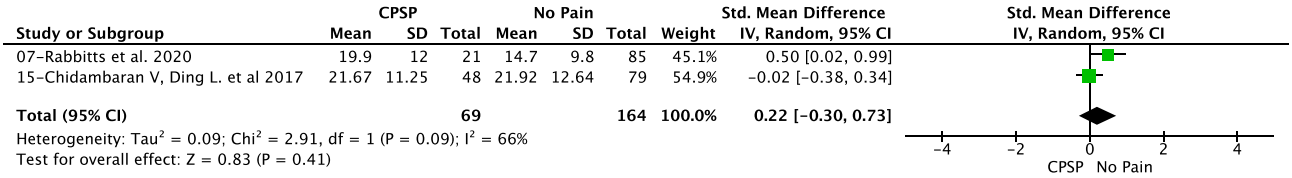
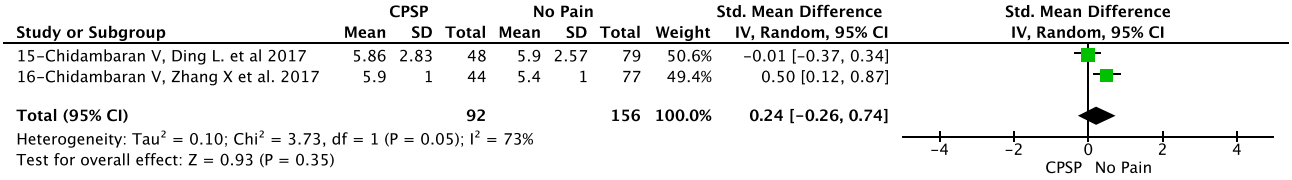


Figure 9 Differences in parent pain catastrophizing between patients who developed chronic postsurgical pain (CPSP) and those without pain at 3 months after surgery Forest plot showing standardized mean differences (SMD) in parent pain catastrophizing between families of children, adolescents and young adults who developed chronic postsurgical pain (CPSP) and those without pain at 3 months after surgery. A random-effects meta-analysis was conducted. Positive values indicate higher levels of parent pain catastrophizing in the CPSP group compared with the no-pain group. Squares represent individual study effect sizes weighted by sample size, horizontal lines indicate 95% confidence intervals, and the diamond represents the pooled effect estimate. Between-study heterogeneity is reported using the I^2 statistic.



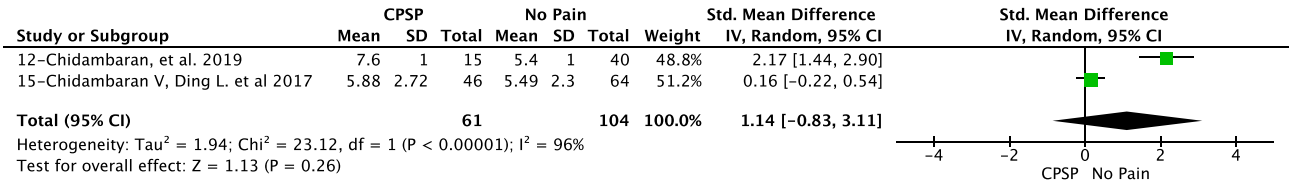


Figure 10 Differences in parent pain anxiety between patients who developed chronic postsurgical pain (CPSP) and those without pain at 3 and 12 months after surgery Forest plots showing standardized mean differences (SMD) in parent pain anxiety between families of children, adolescents and young adults who developed chronic postsurgical pain (CPSP) and those without pain at 3 and 12 months after surgery. Random-effects meta-analyses are presented separately for each follow-up period. Positive values indicate higher levels of parent pain anxiety in the CPSP group compared with the no-pain group. Squares represent individual study effect sizes weighted by sample size, horizontal lines indicate 95% confidence intervals, and diamonds represent pooled effect estimates. Between-study heterogeneity is reported using the I^2 statistic.

Discussion

The aim of this review was to identify presurgical prognostic factors for CPSP in children, adolescents, and young adults. Although 44 potential predictors were identified, inconsistent reporting across studies allowed meta-analysis of only eight factors. Taken together, the findings show that only baseline pain intensity demonstrates consistent prognostic value, whereas sociodemographic and psychological variables—both in children and in parents—show limited and inconsistent associations with CPSP. Importantly, by examining a developmental window spanning childhood through young adulthood, this review provides new insight into how pain-related vulnerabilities may evolve across the lifespan, rather than being confined to traditional pediatric age cutoffs.

With respect to age, the results of this review indicate that children, adolescents, and young adults who developed CPSP at 3 months were, on average, older than those who did not. This aligns with evidence showing that the likelihood of chronic pain increases across adolescence[22,23,70–75] and into early adulthood.[13] Adolescents typically have more extensive pain histories, greater exposure to medical procedures, and heightened emotional reactivity, all of which may sensitize pain pathways and increase vulnerability to CPSP.[14] Integrating a lifespan perspective, young adulthood may represent a developmental stage in which pain-related cognitive, affective, and neural patterns become more stable Evidence from chronic pain research indicates that once these patterns are established in older adolescents and young adults, they tend to persist and are less likely to resolve spontaneously.[8] From this perspective, adolescence appears to be a sensitive period during which risk factors consolidate, while young adulthood reflects the strengthening rather than the emergence of these vulnerabilities. Further longitudinal studies following patients from childhood into adulthood are needed to fully understand how risk evolves across developmental transition.

Consistent with previous work,[13] sex was not associated with CPSP at any time point. This finding contrasts with broader pediatric pain literature showing that girls experience greater acute postoperative pain,[76] higher pain unpleasantness,[76] more frequent chronic pain,[70,77] and greater pain impact, psychological burden, and sleep disturbances.[23,68,71,73,78,79] The absence of sex differences in CPSP may partly reflect sample imbalances across studies and methodological variability, but it also suggests that the transition from acute to chronic postsurgical pain may be driven more strongly by nociceptive and procedural factors than by sex-associated biopsychosocial differences. These findings underscore the need for future studies to examine sex effects with adequately powered and balanced samples.

Baseline presurgical pain intensity emerged as one of the strongest and most consistent predictors of CPSP, particularly at 3 and 6 months. This aligns with previous research showing that high presurgical pain contributes to more severe acute postoperative pain[69] and increases the likelihood of persistent pain, disability, and pain interference.[42,46,47] These findings emphasize the clinical relevance of implementing systematic preoperative pain assessment and multimodal analgesic preparation.[14,80] Future randomized trials should investigate whether presurgical pain optimization reduces CPSP incidence.[26] Notably, baseline pain intensity did not predict CPSP at 12 months, suggesting that its influence is strongest during the early stages of pain chronification and may diminish over longer follow-up periods.

Regarding psychological factors, child pain catastrophizing and anxiety-related constructs (pain anxiety and anxiety sensitivity) showed limited and inconsistent prognostic value. Although children and adolescents who developed CPSP sometimes showed higher scores at 3 months, meta-analytic results did not support a consistent association. These findings contrast with the robust literature demonstrating that catastrophizing and anxiety sensitivity strongly influence pain maintenance, disability, and emotional distress in pediatric chronic pain.[81–83] In this context, it may be important to distinguish anxiety from anxiety sensitivity. While anxiety reflects a general emotional state characterized by worry and heightened arousal, anxiety sensitivity is conceptualized as a more stable cognitive–affective trait involving the fear of anxiety-related bodily sensations and their perceived consequences.[84–89] This construct may be particularly relevant in chronic pain, as anxiety sensitivity shares key mechanisms with pain catastrophizing, including heightened interoceptive threat appraisal and increased attentional focus on internal sensations. These overlapping processes may contribute to pain persistence and amplification, helping to explain why anxiety sensitivity emerges as a meaningful risk factor in the perioperative trajectory toward chronic postsurgical pain.

Interestingly, presurgical psychological factors did not emerge as strong predictors of CPSP onset. This contrasts with the well-established role of psychological processes in general pediatric chronic pain. One possible explanation is that early CPSP development may be more strongly driven by surgery-specific nociceptive input and perioperative inflammatory mechanisms, whereas psychological factors may exert greater influence during later stages of pain persistence. In this context, presurgical psychological vulnerability may contribute more substantially to the maintenance and functional impact of pain over time rather than to its initial transition. Importantly, this does not diminish the clinical value of preoperative screening, which has demonstrated utility in identifying at-risk patients,[90,91] but rather highlights the need for future research examining psychological processes during the postoperative period as potential mechanisms of long-term pain maintenance.

Taken together, the evidence suggests that psychological factors may exert greater influence on the persistence and impact of chronic pain rather than on its initial onset. This pattern aligns with theoretical models positing that early biological and nociceptive processes are central during the transition from acute to chronic pain, whereas psychological factors become more prominent in shaping long-term trajectories once persistent pain is established.[44,63,66] This distinction has important clinical implications: presurgical interventions may need to prioritize nociceptive modulation, while postoperative rehabilitation can focus more on psychological flexibility, coping, and parent–child interaction patterns.

Finally, parental pain catastrophizing and parental pain anxiety were not found to be significant prognostic factors for CPSP. Despite extensive evidence demonstrating strong parent–child pain dynamics in chronic pain populations[92,93]—through both actor and partner effects, behavioral reinforcement, and parental influence on pain[92,94] —these influences do not appear to translate into presurgical predictors of CPSP. The lack of association suggests that parental cognitions may shape children's pain experiences during recovery or long-term adjustment but are unlikely to determine whether acute postoperative pain becomes chronic.[5] Nevertheless, understanding dyadic processes remains fundamental for multidisciplinary pain treatment programs.

Limitations and Future Directions

The findings of this review should be interpreted considering several limitations. The limited availability of raw data and inconsistent reporting prevented a comprehensive meta-analysis of effect sizes such as odds ratios, restricting the ability to quantify absolute risk. Heterogeneity in CPSP definitions, follow-up durations, and surgical procedures further complicates direct comparisons across studies. Additionally, more than 60% of prognostic factors were examined in only one or two cohorts, which limits the strength of conclusions. The underrepresentation of non-White populations also restricts generalizability. Finally, all included studies focused on major surgeries requiring hospitalization, leaving open the question of whether similar risk patterns apply to minor surgeries, which are highly prevalent in pediatric populations.

Future studies should adopt standardized CPSP definitions, report prognostic variables consistently, include more diverse populations, and follow patients across the transition from adolescence into young adulthood. Clinical trials should evaluate whether optimizing presurgical pain and improving perioperative management can reduce CPSP incidence. Furthermore, expanding research to include minor surgeries could clarify whether risk factors differ across surgical severity.

Conclusions

Baseline pain intensity emerged as the most consistent presurgical prognostic factor for CPSP across children, adolescents, and young adults, particularly during the early months following surgery. Age showed a small association with CPSP at 3 months, whereas sex and psychological variables—including catastrophizing, pain anxiety, and anxiety sensitivity—were not reliable predictors. Parental psychological factors similarly showed no prognostic value. However, these findings are based on a limited number of studies restricting the strength and generalizability of conclusions. Larger, methodologically rigorous prospective studies are needed to clarify the prognostic role of presurgical factors across developmental stages.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors have no conflicts of interest to declare.

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