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Examining the Intergenerational Cascade from Parent Adverse Childhood Experiences to Child Chronic Pain: The Mediating Roles of Parent Chronic Pain and PTSD Symptoms

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Examining the Intergenerational Cascade from Parent Adverse Childhood Experiences to Child Chronic Pain: The Mediating Roles of Parent Chronic Pain and PTSD Symptoms

by

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Abstract

Introduction: Adverse childhood experiences (ACEs; exposure to abuse, neglect, household dysfunction as a child) are common and associated with poor mental and physical health outcomes in adolescence and adulthood. Emerging research suggests ACEs can also confer risk for the next generation. Indeed, *parent* ACEs have been found to relate to children's general health, development, and psychosocial functioning. Research has yet to examine ACEs among parents of youth with chronic pain. Parent ACEs may play an important role in pediatric chronic pain, given their association with physical (e.g., parent chronic pain) and mental (e.g., parent PTSD) health conditions that are related to the functioning of youth with chronic pain. The current study sought to examine the relation between parent ACEs and child chronic pain as well as the potential mediating roles of parent chronic pain and PTSD symptoms in this relation. **Methods:** Parent-child dyads were recruited from tertiary-level pediatric chronic pain clinics in Canada. At baseline, parents completed self-report measures of exposure to ACEs, chronic pain status, and current PTSD symptoms. At 3-month follow-up, youth completed self-report measures of pain intensity and pain interference. The final sample included 195 youth with chronic pain (75.9% female, M_{age} = 14.39 years) and one of their parents (92.3% female, M_{age} = 44.91 years). **Results:** Over two-thirds (67.7%) of parents reported one or more ACEs and almost one-quarter (22.1%) reported four or more ACEs. Parent ACEs (total score, maltreatment score, and household dysfunction score) were related to parent chronic pain status but not parent PTSD symptoms. Moreover, parent ACEs were not related to youth pain outcomes either directly or indirectly through parent chronic pain or PTSD symptoms. **Conclusions:** These findings suggest that ACEs are prevalent among parents of youth seeking treatment for their chronic pain but are not directly related to the youth's pain or impairment. Further research that

examines the role of parent ACEs in the *development* of child chronic pain as well as other potential mediators of this association is needed to inform interventions that prevent the intergenerational transmission of risk for chronic pain.

Preface

Portions of this thesis have been submitted to a peer-reviewed journal for publication as J.K. Beveridge, K.S. Dobson, S. Madigan, K.O. Yeates, A.L. Stone, A.C. Wilson, S. Salberg, R. Mychasiuk, and M. Noel, “Adverse childhood experiences in parents of youth with chronic pain: Prevalence and comparison to a community-based sample”. The study reported was covered by Ethics Certificate numbers REB15-3100 (issued by the University of Calgary Conjoint Health Research Ethics Board on June 23, 2016), 1023148 (issued by the IWK Research Ethics Board on February 15, 2018), and 100005648 (issued by the SickKids Research Ethics Board on June 14, 2018), for the project “Pain and Mental Health in Youth (PATH)”.

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ACEs	Adverse childhood experiences
ACH	Alberta Children's Hospital
HPA axis	Hypothalamic-pituitary-adrenal axis
IWK	IWK Health Centre
M	Mean
OR	Odds ratio
PTSD	Posttraumatic stress disorder
SD	Standard deviation
SE	Standard error
SickKids	Hospital for Sick Children
95% CI	95% confidence interval

Introduction

Chronic pain (i.e., pain that recurs or persists for longer than three months; Treede et al., 2015) is prevalent among children and adolescents and causes significant impairment for a subsample of youth (Huguet & Miró, 2008; King et al., 2011; Stanford, Chambers, Biesanz, & Chen, 2008). Unfortunately, current psychological treatments for the management of pediatric chronic pain are often ineffective (Fisher et al., 2018) and if pediatric chronic pain is not successfully treated, many youth will continue to experience chronic pain and mental health problems into adulthood (Fearon & Hotopf, 2001; Shelby et al., 2013; Walker, Sherman, Bruehl, Garber, & Smith, 2012). As such, a better understanding of factors that contribute to pediatric chronic pain, and which may be effective targets for intervention, is needed.

A substantial literature has examined the role of parent factors in pediatric chronic pain (Donnelly, Palermo, & Newton-John, 2020). This research has largely focused on how parents respond to their child's chronic pain and has found that responses that reinforce child pain behaviours (e.g., protectiveness) are related to worse pain and impairment in youth (e.g., Claar, Simons, & Logan, 2008; Cunningham et al., 2014; Walker & Zeman, 1992). However, recent research suggests that parents' own physical (e.g., chronic pain) and mental (e.g., anxiety and depressive symptoms) health may be *more* important for children's pain-related functioning than their responses (Poppert Cordts, Stone, Beveridge, Wilson, & Noel, 2019; Stone, Bruehl, Smith, Garber, & Walker, 2018). In addition, research has found that posttraumatic stress disorder (PTSD) symptoms are prevalent among parents of youth with chronic pain and related to worse child pain outcomes (Neville, Soltani, Pavlova, & Noel, 2018; Noel et al., 2016). These findings have important treatment implications, as psychological interventions for pediatric chronic pain

often focus on reducing maladaptive parent responses and do not address parents' own physical or psychological functioning (Eccleston, Fisher, Law, Bartlett, & Palermo, 2015).

Despite increasing attention to the role of parent mental and physical health in pediatric chronic pain, research has yet to examine adverse childhood experiences (ACEs; exposure to abuse, neglect, or household dysfunction as a child) among parents of youth with chronic pain. ACEs have been widely studied in other populations and tied to negative physical and mental health outcomes in adults (Anda et al., 2006; Felitti et al., 1998; Hughes et al., 2017; Petruccelli, Davis, & Berman, 2019), including those identified as parent risk factors for the development and maintenance of pediatric chronic pain (e.g., chronic pain, PTSD symptoms). Thus, the aims of the current study were to examine the relation between parent ACEs and child pain outcomes, as well as the mediating roles of parent chronic pain and parent PTSD symptoms in this relation, in a clinical sample of youth with chronic pain and their parents.

Pediatric Chronic Pain

Chronic pain is highly prevalent among children and adolescents, affecting between 11% to 38% of youth, a rate that has increased in the last several decades (King et al., 2011; Stanford et al., 2008). For a smaller proportion of youth, approximately 5%, their chronic pain causes moderate to severe impairment (Huguet & Miró, 2008). Chronic pain may be due to an injury, surgical procedure, chronic health condition, or internal disease process (Treede et al., 2015). However, chronic pain may not be associated with an identifiable injury or disease, such as in fibromyalgia or functional abdominal pain. In these cases, pain is considered the primary chronic health condition (Treede et al., 2019). The most common primary pain complaints among youth are headache, abdominal pain, back pain, and limb pain; however, many youth with chronic pain report pain in multiple locations (King et al., 2011; Merlijn et al., 2003; Stanford et al., 2008).

The annual health care cost of pediatric chronic pain in Canada has been estimated to be $144 million, which is largely due to physician visits and hospitalizations (Hogan, Taddio, Katz, Shah, & Krahn, 2016). In addition to its economic cost, pediatric chronic pain can also have a significant personal impact. Youth with chronic pain may miss significantly more school than youth with other chronic conditions, have sleep disturbances that interfere with daytime function, report reductions in peer activities and sports, and experience mental health problems including clinically-elevated depressive, anxiety, and PTSD symptoms (Palermo, 2000; Vinall, Pavlova, Asmundson, Rasic, & Noel, 2016). Pediatric chronic pain can also have a negative impact on the social, emotional, and financial well-being of parents and can interfere with family functioning (Lewandowski, Palermo, Stinson, Handley, & Chambers, 2010; Palermo & Eccleston, 2009).

Current treatments for pediatric chronic pain include psychological interventions, which focus on helping children manage their pain, disability, and distress and include behavioural strategies such as relaxation training and cognitive strategies such as cognitive restructuring. Unfortunately, research suggests these psychological treatments are often ineffective, with small and short-lasting effects (Fisher et al., 2018). Importantly, if pediatric chronic pain is not successfully treated, up to two-thirds of youth will continue to have chronic pain into adulthood and are at increased risk for mental health problems including anxiety and depressive disorders as adults (Fearon & Hotopf, 2001; Shelby et al., 2013; Walker et al., 2012). As such, a better understanding of factors that contribute to the development and maintenance of pediatric chronic pain, and which may be effective targets for intervention, is needed.

Parent Factors in Pediatric Chronic Pain

Parents play a critically important role in the development and health of their children, including their experiences of chronic pain (Palermo & Eccleston, 2009). Indeed, a large body of

research has shown that parent factors, including behavioural (e.g., protective responses), cognitive (e.g., catastrophic thinking about child pain), and affective (e.g., depressive symptoms) factors, are related to children's chronic pain outcomes (Donnelly et al., 2020). The majority of this research has focused on the behavioural responses of parents. This research has shown that parent responses that provide special attention or rewards to the child in pain (e.g., protective and monitoring responses), and thus reinforce the child's pain behaviours, are related to poor outcomes for youth with chronic pain including greater pain and impairment (Claar et al., 2008; Cunningham et al., 2014). Importantly, the majority of evidence-based psychological treatments for the management of pediatric chronic pain also focus on these parent responses (Eccleston et al., 2015). However, a recent trial that examined the efficacy of an internet-delivered cognitive-behavioural therapy (I-CBT) for pediatric chronic pain found that reductions in maladaptive parent responses were not related to decreases in children's self-reported disability (Law et al., 2017), suggesting that these parent responses may not be the most important treatment target.

Instead, increasing research is demonstrating the importance of parents' own functioning in pediatric chronic pain. For example, a recent study that used structural equation modelling to examine the interrelationships between parent factors and their differential associations with pain-related outcomes in youth with chronic pain found that parents' self-reported chronic pain features (e.g., intensity, number of locations), physical functioning (e.g., pain interference), and psychological functioning (e.g., anxiety and depressive symptoms) were all related to children's pain-related outcomes (Poppert Cordts et al., 2019). Importantly, parent responses to child pain were not related to children's pain-related outcomes in this multivariate model. Moreover, the study by Law et al. (2017) that examined the efficacy of I-CBT for pediatric chronic pain found that youth whose parents had higher distress at pre-treatment had less improvement in their self-

reported disability during treatment than youth whose parents had lower distress at pre-treatment. These recent findings suggest that parents' own physical and psychological functioning may be more important for the maintenance of pediatric chronic pain than their responses to child pain. Indeed, research has increasingly demonstrated the high prevalence of physical (e.g., chronic pain) and mental (e.g., clinically-elevated anxiety, depressive, PTSD symptoms) health problems among parents of youth with chronic pain as well as their impact on the functioning of youth with chronic pain (Beveridge, Neville, Wilson, & Noel, 2018; Birnie et al., 2020; Campo et al., 2007; Eccleston, Crombez, Scotford, Clinch, & Connell, 2004; Noel et al., 2016; Stone et al., 2018). Despite recent attention to the role of parent health in pediatric chronic pain, research has yet to examine the prevalence of ACEs among parents of youth with chronic pain or the impact of parent ACEs on the outcomes of youth with chronic pain. Parent ACEs may play an important role in the development and maintenance of chronic pain in youth, given their association with physical (e.g., chronic pain) and mental (e.g., PTSD symptoms) health conditions that have been implicated as parent factors contributing to pediatric chronic pain.

Adverse Childhood Experiences (ACEs)

ACEs represent a set of adverse or traumatic events that an individual may experience in the first 18 years of their life. There are typically ten ACEs that are studied, including five types of maltreatment (physical, emotional, and sexual abuse; physical and emotional neglect) and five types of household dysfunction (lived with someone with substance use problems, mental illness, or who went to jail; witnessed physical violence between parents; parents separated or divorced). Felitti, Anda, and colleagues (1998) were the first to study the impact of these adverse events in their seminal ACE Study and found that individuals exposed to ACEs were more likely to report negative mental and physical health conditions and behaviours as adults including depression

and suicidality, cancer and stroke, problematic substance use, and cardiovascular and respiratory diseases. Since this study, ACEs have been widely researched and their association with poor health outcomes has been well-established in adults as well as children and adolescents (Anda et al., 2006; Hughes et al., 2017; Liming & Grube, 2018; Oh et al., 2018; Petruccelli et al., 2019; Scully, McLaughlin, & Fitzgerald, 2020). These associations tend to occur in a dose-response fashion, such that individuals exposed to more types of ACEs (e.g., 4 or more) are at greater risk for poor health outcomes than individuals with lower or no exposure to ACEs (Anda et al., 2006; Felitti et al., 1998; Hughes et al., 2017; Petruccelli et al., 2019). Importantly, research has also shown that ACEs are common. For example, 64% of adults in the ACE Study reported at least one ACE and 12.5% reported 4 or more ACEs (CDC-Kaiser Permanente, 2016). Moreover, most participants (i.e., 81-98%) in this study who reported one ACE reported at least one other ACE (Dong et al., 2004), suggesting that someone who experiences one type of childhood adversity will likely experience multiple types.

Numerous studies have shown that individual ACEs (e.g., sexual abuse) and total ACE scores are related to the occurrence and severity of chronic pain in adolescents and adults (Anda, Tietjen, Schulman, Felitti, & Croft, 2010; Davis, Luecken, & Zautra, 2005; Juang, Wang, Fuh, Lu, & Chen, 2004; McCall-Hosenfeld, Winter, Heeren, & Liebschutz, 2014; Nelson, Simons, & Logan, 2018; Olivieri, Solitar, & Dubois, 2012; Park et al., 2016; Scott et al., 2011). In fact, a meta-analysis examining the association between childhood maltreatment and physical health in adulthood found that neurological problems (e.g., migraines) had the strongest relation with childhood maltreatment, more so than cardiovascular, respiratory, and metabolic problems (Wegman & Stetler, 2009). ACEs are also more prevalent among individuals with chronic pain. For example, one study found 84% of women with chronic pain reported one or more ACEs and

44.5% reported 4 or more ACEs (Dennis, Clohessy, Stone, Darnall, & Wilson, 2019). Similar rates have been found among youth with chronic pain, with 81.6% reporting one or more ACEs and 23.4% reporting 3 or more ACEs (Nelson et al., 2018). Importantly, emerging research suggests that experiences of maltreatment may be stronger predictors of chronic pain than experiences of household dysfunction. For example, a prospective study that examined pain in young women with substantiated reports of childhood maltreatment found that maltreatment, but not other types of adversity, predicted women's pain reports (Beal et al., 2020). Thus, some types of ACEs may be more important for chronic pain than others.

Research has also found a significant association between ACEs and PTSD. Specifically, studies with community and clinical samples of youth and adults have shown that exposure to more types of ACEs increases one's likelihood of meeting diagnostic criteria for PTSD as well as reporting more severe PTSD symptoms (Grasso, Dierkhising, Branson, Ford, & Lee, 2016; Kalmakis, Chiodo, Kent, & Meyer, 2020; Leardmann, Smith, & Ryan, 2010; Wu, Schairer, Dellor, & Grella, 2010). For example, one study found that rates of PTSD increased as ACE scores increased, with 42.3% of participants who were exposed to 4 or more ACEs meeting criteria for PTSD in their lifetime, compared to only 2.8% of participants who were not exposed to ACEs (Sachs-Ericsson, Sheffler, Stanley, Piazza, & Preacher, 2017). Similar to chronic pain, individual ACEs (e.g., physical neglect) have also been shown to relate to more severe PTSD symptoms in adulthood (Schalinski et al., 2016), and research suggests that experiences of maltreatment may be stronger predictors of elevated PTSD symptoms than experiences of household dysfunction (Atzl, Narayan, Rivera, & Lieberman, 2019).

Thus, ACEs may be related to the occurrence of chronic pain and elevated PTSD symptoms in parents of youth with chronic pain. Conceptual and empirical literature posit that

ACEs contribute to these physical and mental health conditions through both biological and psychosocial pathways. First, neurobiological research suggests that experiences of toxic stress in childhood can alter neural structures and systems that are involved in the stress response, mood and impulse control, and learning and memory (Anda et al., 2006; Shonkoff et al., 2012). These alterations can lead to lasting physiological and physical changes (e.g., increased cortisol) and/or high-risk health behaviours (e.g., substance use), which can both increase risk for health conditions. Epigenetic research suggests that early life stress can also alter genes that regulate the stress response, in ways that increase risk for poor health (Provençal & Binder, 2015). In terms of psychosocial pathways, conceptual models suggest that ACEs can influence cognitive and interpersonal styles, which then indirectly increase risk for poor health through increased mental health problems and poor social connections (Kendall-Tackett, 2002). In line with these models, research has found that psychological and social factors in adolescence and adulthood, including elevated symptoms of mental health disorders (e.g., PTSD, depression) and more life stressors (e.g., divorce), mediate the relation between childhood maltreatment and adult physical health (Beal et al., 2020; Min, Minnes, Kim, & Singer, 2013). In this way, childhood adversity may influence biological and psychological processes, which, in turn, may increase an individual's risk for poor physical and mental health outcomes throughout his or her life.

This risk that ACEs confer for poor outcomes may extend across generations. Research on the intergenerational impact of ACEs has found that children of parents exposed to more types of ACEs are rated as having more developmental concerns, poorer general health, greater internalizing and externalizing behaviours, and more socio-emotional difficulties than children of parents exposed to less or no ACEs (Cooke, Racine, Plamondon, Tough, & Madigan, 2019; Lê-Scherban, Wang, Boyle-Steed, & Pachter, 2018; Madigan, Wade, Plamondon, Maguire, &

Jenkins, 2017; McDonnell & Valentino, 2016; Plant, Pawlby, Pariante, & Jones, 2018; Racine, Plamondon, Madigan, McDonald, & Tough, 2018; Schickedanz, Halfon, Sastry, & Chung, 2018; Treat, Sheffield-Morris, Williamson, & Hays-Grudo, 2019). Similar to literature on the impact of ACEs on an individual's health, biological and psychosocial processes may play a role in the transmission of ACEs risk from parent to child. First, recent research has shown that maternal ACEs are related to maternal HPA axis function during pregnancy, which in turn, is related to newborn HPA axis function and toddler behavioural problems (Thomas-Argyriou et al., 2020; Thomas, Letourneau, Campbell, & Giesbrecht, 2018). Maternal ACEs have also been found to indirectly relate to the physical health of 18-month-old children through biomedical risks during the pre- and peri- natal periods (Madigan et al., 2017). In these ways, parent ACEs may increase child risk for poor outcomes through biological processes. Research has also found that parents' mental health (e.g., anxiety and depressive symptoms) and parenting practices (e.g., harsh discipline) mediate the relation between parent ACEs and child outcomes (Cooke et al., 2019; Madigan, Wade, Plamondon, & Jenkins, 2015; Madigan et al., 2017; McDonnell & Valentino, 2016; Plant et al., 2018; Schickedanz et al., 2018; Treat et al., 2019), which suggests that psychosocial processes also contribute to the intergenerational transmission of risk from ACEs.

The above findings suggest that parent ACEs may be related to child chronic pain. Alterations in maternal HPA axis function during pregnancy, and the resulting disruptions to fetal development, have been posited as risk factors for the development of chronic pain in youth (Stone & Wilson, 2016) and, as noted, substantial research has demonstrated the association between parent psychosocial factors (e.g., mental health symptoms, parenting behaviours) and the functioning of youth with chronic pain (Donnelly et al., 2020). To date, only one study has examined parent ACEs in the context of chronic pain. In this study, Dennis and colleagues

(2019) examined mothers with chronic pain and their children and found that maternal ACEs were not related to child pain or physical function but were indirectly related to child depressive symptoms through the mother's depressive symptoms. However, child pain frequency was low in this sample as children were intentionally recruited prior to adolescence, the period when chronic pain often first emerges (King et al., 2011). To more fully understand the role of parent ACEs in pediatric chronic pain, research that examines parent ACEs, as well as potential factors mediating their relation with child chronic pain, in clinical samples of youth with chronic pain is needed. The current study examined two factors that may be key mediators of the parent ACEs-child chronic pain relation, given their association with both ACEs and pediatric chronic pain: parent chronic pain status and parent PTSD symptoms.

Parent Chronic Pain

It is well documented that chronic pain tends to aggregate in families, such that children of parents with chronic pain are significantly more likely to report pain than children of parents without chronic pain (Higgins et al., 2015). In line with these findings, parent chronic pain is prevalent in clinical samples of youth with chronic pain, with approximately 50% to 75% of parents of youth with chronic pain reporting their own chronic pain problem (Beveridge et al., 2018; Stone et al., 2018). Moreover, parent chronic pain is related to worse outcomes for youth with chronic pain including greater impairment and lower quality of life (Beveridge et al., 2018). This intergenerational transmission of risk for chronic pain and poor outcomes is likely due to a combination of genetic and environmental factors (Stone & Wilson, 2016). In fact, research using the classic twin study design suggests genetic factors account for approximately 40% to 70% of the variance in pediatric chronic pain conditions (e.g., headache, abdominal pain, low back pain, neck pain, widespread pain) while shared and unique environmental factors account

for approximately 30% to 60% of the variance (Hestbaek, Iachine, Leboeuf-Yde, Kyvik, & Manniche, 2007; Ståhl et al., 2013; Svensson, Larsson, Bille, & Lichtenstein, 1999).

One environmental factor that may be particularly important for pediatric chronic pain is parent modeling of pain behaviours. Research on this topic is guided by social learning theory, which posits that individuals learn through observing the behaviours of others (Bandura, 1977). Parents with chronic pain may model pain behaviours (i.e., how to perceive and react to pain sensations) to their child, which could, in turn, contribute to the development of the same pain behaviours and thus pain problems in the child. This hypothesis is supported by empirical research that has shown children frequently report the same pain locations (e.g., headaches) and pain-related behaviours (e.g., physician visits for pain symptoms) as their parents with chronic pain (Arruda, Guidetti, Galli, Albuquerque, & Bigal, 2010; Campo et al., 2007; Evans, Meldrum, Tsao, Fraynt, & Zeltzer, 2010; Laurell, Larsson, & Eeg-Olofsson, 2005; Levy, 2011; Osborne, Hatcher, & Richtsmeier, 1989). Research has shown that parent modeling of pain behaviours is also related to the *maintenance* of chronic pain and impairment in youth. For example, a recent study found that parents with chronic pain modeled more pain behaviours to their children, and these pain behaviours were, in turn, related to worse pain-related outcomes for adolescents with chronic pain (Stone et al., 2018). In these ways, parent chronic pain may mediate the potential relation between parent ACEs and child chronic pain. That is, parent ACEs may confer risk for parent chronic pain, which in turn, may contribute to chronic pain and impairment in the child.

Parent PTSD Symptoms

Research has shown that elevated PTSD symptoms are also prevalent among parents of youth with chronic pain and associated with poor child outcomes. Specifically, Noel et al. (2016) found that parents of youth with chronic pain reported higher PTSD symptoms, and were more

likely to meet clinical cut-offs for PTSD, than parents of youth without chronic pain. Moreover, higher PTSD symptoms in parents have been found to relate to more severe pain and impairment among youth with chronic pain (Neville et al., 2018; Noel et al., 2016). Similar to parents with chronic pain, parents with elevated PTSD symptoms may model maladaptive coping strategies (e.g., avoidance of stressful situations, hypervigilance to threatening situations) to their child that increase the child's risk for greater pain and impairment. Symptoms of PTSD (e.g., avoidance, negative evaluations of self and world, increased negative emotions) could also interfere with parenting abilities and the parent-child relationship (Creech & Misca, 2017), which could impact the child's ability to cope with their chronic pain. Lastly, it has been posited that higher levels of distress in parents may interfere with their ability to implement treatment recommendations that aim to improve the functioning of their child with chronic pain (Law et al., 2017; Stone et al., 2018). Thus, parent PTSD symptoms may mediate the potential relation between parent ACEs and child chronic pain, with parent ACEs increasing parent risk for elevated PTSD symptoms, and parent PTSD symptoms increasing child risk for poor chronic pain-related outcomes.

Present Study

The research discussed herein suggests that parent ACEs may play a distal role in child chronic pain, through increased risk for parent chronic pain and elevated PTSD symptoms. The broad aim of the current study was to examine this intergenerational cascade from parent ACEs to child chronic pain, in a clinical sample of youth with chronic pain and their parents.

Aims and Hypotheses

Aim 1: To report the prevalence of ACEs in parents of youth with chronic pain. Hypothesis 1: Exposure to ACEs, including maltreatment and household dysfunction, will be common among parents of youth with chronic pain.

Aim 2: To examine the associations between parent ACEs (total ACE score, maltreatment score, and household dysfunction score) and parent chronic pain status and parent PTSD symptoms.

Hypothesis 2: Parents with higher (versus lower) total ACE scores will be more likely to report chronic pain and higher PTSD symptoms. These associations will be driven by experiences of maltreatment, such that parent maltreatment scores will be stronger predictors of parent chronic pain and parent PTSD symptoms than parent household dysfunction scores.

Aim 3: To examine the association between parent ACEs (total ACE score, maltreatment score, and household dysfunction score) and youth pain outcomes (pain intensity and interference), and the mediating roles of parent chronic pain status and parent PTSD symptoms in this association.

Hypothesis 3: Higher total ACE scores in parents will be related to worse pain outcomes in youth (i.e., higher pain intensity and greater pain interference). This association will be stronger for parent maltreatment than household dysfunction. Moreover, parent chronic pain status and parent PTSD symptoms will mediate the association between parent ACEs and youth pain outcomes.

Methods

This study is part of a broader program of research, entitled the Pain and Mental Health in Youth (PATH) Study, that is examining a myriad of cognitive, behavioural, neurobiological, and social factors in the co-occurrence of chronic pain and internalizing mental health disorders in youth. The data for this research program is being collected across multiple sites in Canada. The primary data collection site is Alberta Children's Hospital (ACH) in Calgary, Alberta. The secondary data collection sites are IWK Health Centre (the IWK) in Halifax, Nova Scotia and the Hospital for Sick Children (SickKids) in Toronto, Ontario. The PATH Study has been approved by the University of Calgary Research Ethics Board (REB15-3100), the IWK Research Ethics

Board (ID: 1023148), and the SickKids Research Ethics Board (ID: 100005648). This study used data collected at the baseline and 3-month follow-up timepoints.

Participants

Youth with chronic pain and one of their parents were recruited from tertiary-level, outpatient chronic pain clinics at pediatric hospitals in Canada (i.e., Vi Riddell Outpatient Pain and Rehabilitation Program at ACH, Pediatric Complex Pain Clinic at the IWK, and Chronic Pain Clinic at SickKids). Youth were eligible to participate if they were 10 to 18 years of age and had chronic pain (i.e., pain that persists or recurs for ≥ 3 months) at the time of recruitment that was not associated with an underlying disease (e.g., juvenile idiopathic arthritis, cancer). Youth were not eligible if they were unable to read/speak English, did not have access to the internet, or had any of the following: severe cognitive impairment or developmental disorder, schizophrenia spectrum or other psychotic disorder, or presence of a serious chronic health or life-threatening condition (e.g., cancer). Parents were eligible to participate if they were the legal guardian of the youth, could read/speak English, and had access to the internet.

The final sample for the current study included 195 parent-child dyads. At ACH, 360 families were contacted and invited to participate. Of these, 63 were not eligible and 107 either did not want to participate or could not be reached after initial contact to be enrolled in the study. Thus, 190 parent-child dyads were enrolled in the study at ACH. At the IWK, 76 families were contacted about the study. Of those, five were not eligible, 34 declined participation, five could not be reached after initial contact to be enrolled, and two did not consent. Thus, 30 parent-child dyads were enrolled at the IWK. At SickKids, 22 families were screened for inclusion. Of those, two were not eligible, six declined participation, and eight could not be reached after initial contact to be enrolled. Thus, six parent-child dyads were enrolled in the study at SickKids. Of

the 226 families enrolled at all sites, eight could not be reached after enrolment to complete the online consent forms, six withdrew before baseline, eleven parents either did not complete the baseline survey or the ACE Questionnaire, two parents enrolled twice (with a different child), one family was not able to participate due to the COVID-19 pandemic, and three parent-child dyads were excluded from the current study because they were not biologically related.

Procedure

Recruitment

The clinic staff of the Vi Riddell Outpatient Pain and Rehabilitation Program at ACH obtained consent from parents of new patients to be contacted by the research team. Clinic staff provided the contact information of consenting families, as well as families who had received care in the program within the last two years, to the research team of the Alberta Children's Pain Research Laboratory. The research team also compiled a list of participants who were previously recruited for a Clinical Outcomes Study (REB14-0162) and consented to be contacted for future research studies. The research team contacted potential participants via email or telephone with information about the study and invited them to participate. Interested parent-child dyads were screened for eligibility over the phone and a verbal informed consent procedure was conducted with both the parent and youth wherein the information contained in the consent and/or assent forms was explained, including details of the study and limits to confidentiality, any questions were answered, and agreement to participate was obtained. Online versions of the consent and/or assent forms were also emailed to parent-child dyads to obtain their written consent.

At the IWK and SickKids, clinic staff of the Pediatric Complex Pain Clinic and Chronic Pain Clinic asked families who were currently receiving care if they were interested in being contacted about a potential research opportunity. Clinic staff provided the contact information of

interested families to the research teams at these sites. The research teams also had access to lists of families who had previously attended appointments in the clinics. The research team at the IWK also had access to a registry of families who were interested in being contacted for research studies. A member of the research team screened these lists and mailed an informational letter to families who were potentially eligible to participate. If families did not decline being contacted, a member of the research team would contact parent-child dyads over the phone with information about the study and invite them to participate. Recruitment cards with the contact information of the research team were also provided to patients in the Pediatric Complex Pain Clinic at the IWK so families could contact the research team directly to learn about the study. Similar to ACH, all interested parent-child dyads were screened for eligibility over the phone and both verbal and written informed consent and/or assent was obtained from parents and youth.

Data Collection

All forms and measures were administered and completed through REDCap (Research Electronic Data Capture), a secure web-based data collection site (Harris et al., 2009, 2019). At the baseline and 3-month follow-up timepoints, parents and youth were each sent a battery of self-report measures. Parents were asked to provide sociodemographic information and complete measures of their exposure to ACEs, chronic pain status and characteristics, and current PTSD symptoms. Youth were asked to complete measures of their pain characteristics. Parents and youth received an honorarium (i.e., $10 or $20 CAD gift cards) for their participation at each timepoint. This study used baseline data for the sample characteristics (i.e., sociodemographic variables, youth pain characteristics) and the parent variables of interest (i.e., ACEs, chronic pain, PTSD symptoms) and follow-up data for the youth variables of interest (i.e., pain intensity and pain interference). Data were collected between February 2017 and June 2020.

Measures

Sociodemographic Information

Parents completed a sociodemographic questionnaire that asked about their own age, gender, race/ethnicity, marital status, relationship to child, education, employment status, and annual household income as well as their child's age, gender, and race/ethnicity.

Parent ACEs

The Adverse Childhood Experiences (ACE) Questionnaire retrospectively assessed parent exposure to 10 categories of ACEs (i.e., emotional, physical, and sexual abuse; emotional and physical neglect; five types of household dysfunction) in the first 18 years of their life. This 28-item measure was developed for the original ACE Study by adapting items from existing measures of childhood abuse, neglect, and household dysfunction (Bernstein et al., 2003; Straus, 1979; Wyatt, 1985). ACE categories are assessed with one or more items, which are rated on dichotomous (yes/no) or 5-point Likert-type (0 = 'never true' or 'never' to 4 = 'very often true' or 'very often') scales. If at least one item of the category was endorsed, the ACE was coded as present. Total ACE scores were obtained by summing responses to the 10 categories. Maltreatment and household dysfunction scores were obtained by summing responses to the relevant 5 categories. Higher scores indicate exposure to more categories of ACEs. The ACE Questionnaire has demonstrated good psychometric properties in community and high-risk populations (e.g., low income women, individuals with major depression; Dube, Williamson, Thompson, Felitti, & Anda, 2004; Ford et al., 2014; Frampton, Poole, Dobson, & Pusch, 2018; Mersky, Janczewski, & Topitzes, 2017) and studies evaluating its factor structure support the subscales of maltreatment and household dysfunction (Ford et al., 2014; Mersky et al., 2017). The measure demonstrated excellent internal consistency (a = .92-.94) in the current study.

Parent Chronic Pain

Similar to previous research (Beveridge et al., 2018), and consistent with the current definition of chronic pain (Treede et al., 2015), parent chronic pain status was assessed with a dichotomous item (yes/no) that asked about the presence of pain for at least three months in a row. If parents selected yes, a follow-up item assessed the duration of their pain in months and years. The intensity and locations of their pain in the past week were also assessed with items from the Pain Questionnaire. Pain intensity was measured using a validated and reliable 11-point Numerical Rating Scale (0 = 'no pain' to 10 = 'worst pain possible'; von Baeyer et al., 2009) and pain locations were assessed with an item that asked about the parts of their body where they experienced the most aches or pains from a checklist of six options (e.g., stomach, head, other).

Parent PTSD Symptoms

Parent PTSD symptoms were assessed with the PTSD Checklist for DSM-5 (PCL-5) with Life Events Checklist for DSM-5 (LEC-5) and Criterion A (Weathers et al., 2013). The LEC-5 is a checklist of traumatic events that asks respondents to indicate if events have happened to them personally, if they witnessed them happen to someone else or learned they happened to someone close to them, or if they were exposed to them as part of their job. Respondents are then asked to describe the worst event they experienced in a textbox and answer questions about that event, including how long ago it happened. The 20-item PCL-5 then asks respondents to think about this event and rate how much PTSD-specific symptoms have bothered them in the past month on a 5-point Likert-type scale (0 = 'not at all' to 4 = 'extremely'). A total symptom severity score is obtained by summing the ratings for each item. Higher scores indicate greater PTSD symptoms, with a score of 33 suggested as the clinical cut-off (National Center for PTSD, n.d.). The PCL-5 has excellent reliability and validity (Blevins, Weathers, Davis, Witte, & Domino, 2015) and has

been previously used in research with parents of youth with chronic pain (Beveridge et al., 2018; Noel et al., 2016). It demonstrated excellent internal consistency (a = .94) in the current study.

Youth Pain Characteristics

Youth pain characteristics were assessed with the pain frequency, pain locations, and pain duration items of the commonly-used Pain Questionnaire (Palermo, Witherspoon, Valenzuela, & Drotar, 2004). The pain frequency item measures how often the respondent had pain in the past week on a 5-point Likert-type scale from 'not at all' to 'daily'. The pain location item asks respondents to select the parts of their body where they experienced the most aches or pains in the past week from a checklist of six options (e.g., stomach, head, other). The pain duration item asks respondents to indicate how long they have had pain in years and months. This measure has been used in previous research with youth with chronic pain (Beveridge et al., 2018; Neville et al., 2018; Noel et al., 2016).

Youth Pain Outcomes

Youth pain intensity was measured using a validated and reliable 11-point Numerical Rating Scale (0 = 'no pain' to 10 = 'worst pain possible'; von Baeyer et al., 2009). Youth pain interference was assessed with the Patient Reported Outcomes Measurement Information System (PROMIS) Pain Interference - Short Form. This 4-item measure asks youth to rate the extent to which pain interfered with daily activities such as sleeping in the past week on a 5-point Likert-type scale (1 = 'never' to 5 = 'almost always'). A total score is obtained by summing the ratings for each item and then translating the raw score into a standardized T-score. Higher scores indicate greater pain interference. This measure was developed by the National Institutes of Health using item response theory and has been validated in youth with chronic pain (Kashikar-Zuck et al., 2016). It demonstrated good internal consistency (a = .85) in this study.

Statistical Analyses

Statistical analyses were performed using IBM SPSS Statistics (Version 24). Pearson's correlations and χ^2 tests were conducted to examine associations between sociodemographic variables (parent age, gender, race/ethnicity, education, employment status, household income; child age, gender, race/ethnicity) and key study variables to inform covariates to enter in models. These sociodemographic variables were identified from previous ACEs research (Felitti et al., 1998; Poole, Dobson, & Pusch, 2017; Schickedanz et al., 2018; Thomas et al., 2018). Descriptive statistics were used to characterize the sociodemographic and pain characteristics of the sample and calculate mean scores on key study variables. Paired samples *t*-tests were used to compare youth scores on pain intensity and pain interference at baseline to their scores on these measures at follow-up. Pearson's correlations were used to examine relations between key study variables.

To address hypothesis 1, descriptive statistics were used to characterize the prevalence of ACEs among parents. To address hypothesis 2, binary logistic regression was used to examine the association between parent ACEs and parent chronic pain status and linear regression was used to examine the association between parent ACEs and parent PTSD symptoms. Specifically, three unadjusted (without covariates) and three adjusted (with covariates) models assessed the associations between parent ACEs (first model: parent total ACE score; second model: parent maltreatment score; third model: parent household dysfunction score) and the outcome variable (parent chronic pain status or parent PTSD symptoms). In the adjusted models, covariates were entered in the first step and parent ACEs were entered in the second step. To further explore the relation between parent ACEs and parent chronic pain and parent PTSD symptoms, descriptive statistics and Pearson's correlations were used to examine the relation between parent ACEs and parent chronic pain characteristics (i.e., duration, intensity, locations) and the traumatic events

described by parents on the PCL-5 were coded to identify the prevalence of parents reporting an ACE as their 'worst' event. Descriptive statistics were used to report the percentage of parents describing an ACE as their worst event and the percentage meeting the clinical cut-off for PTSD.

To address hypothesis 3, mediation analyses were conducted when significant relations between key variables were present (i.e., predictor variable was related to the mediator variable and mediator variable was related to the outcome variable). For simple mediation analyses with a continuous mediator variable, use of the PROCESS Macro for SPSS (Version 3.5) was planned (Hayes, 2017). This analysis uses a bootstrap estimation approach to test for the indirect (i.e., mediation) effect. Bootstrapped confidence intervals that do not cross zero are indicative of a statistically significant indirect effect. A bootstrapping sample of 5000 was used in the current analyses. For simple mediation analyses with a dichotomous mediator variable, use of Valeri and VanderWeele's (2013) SPSS Macro for nonlinear mediation models was planned. This technique is based on the counterfactual approach and uses a logistic regression model to allow for binary mediators. The macro generates estimates and confidence intervals for direct and indirect effects and can generate bootstrap confidence intervals to test for the significance of the indirect effect.

Results

Data Preparation

Data Screening

In total, 195 parent-child dyads were included in analyses that used baseline data (hypothesis 1 and 2) and 151 parent-child dyads were included in analyses that used follow-up data (hypothesis 3). However, *n*s in each analysis vary slightly due to missing data (see below). The lower sample size for the follow-up analyses was due to attrition ($n = 3$ families withdrew from the study before follow-up, $n = 3$ families could not be reached for follow-up), incomplete

data on the follow-up survey (n = 13), and families having not reached follow-up yet (n = 25). The baseline sample size met recommendations for the minimum sample size for the logistic regression analyses (i.e., 30 times as many cases as parameters being estimated; Pedhazur, 1997 as cited in Meyers, Gamst, & Guarino, 2017) and the linear regression analyses. Specifically, for the linear regression analyses, an a priori power analysis based on previously published data (Beal et al., 2020) suggested a sample size of 55 was required to detect a medium effect (f^2 = .15, α = .05, 1 – β = .80; G*Power; Faul, Erdfelder, Buchner, & Lang, 2009). The follow-up sample size also met that required for the mediation analyses. Specifically, an a priori power analysis based on previously published data (Beal et al., 2020; Beveridge et al., 2018; Neville et al., 2018) suggested a sample size of 103 was required to detect a medium effect (f^2 = .15, α = .05, 1 – β = .80; G*Power; Faul et al., 2009; Schoemann, Boulton, & Short, 2017) in the mediation analyses.

Missing data on baseline variables ranged from 0% (parent ACEs, parent chronic pain status) to 7.7% (n = 15; parent PTSD symptoms). Missing data on follow-up variables ranged from 4.0% (n = 6; youth pain intensity) to 6.6% (n = 10; youth pain interference). Little's MCAR test, which included the key continuous variables, was non-significant, $\chi^2(9) = 14.39$, p = .109, indicating that the missing data was not inconsistent with being missing completely at random (MCAR). Within-person mean imputation was used for item-level missing data on the measure of parent PTSD symptoms (n = 10 had one missing item, n = 1 had two missing items), but not on the measures of youth pain intensity or youth pain interference given the short length of these measures. No imputation procedures were used for variable-level missing data. Given the low percentage of missing data, and results of Little's MCAR test, pairwise deletion was used for all analyses. For the regression analyses, data were screened for influential cases (i.e., Mahalanobis $d^2 > \chi^2$-critical = .001; leverage > $3(k + 1)/n$; Cook's distance > 1; DFBeta > 1; Field, 2018) and

analyses were conducted with and without these cases to determine their influence on the model. When these cases had an appreciable influence on the model, both sets of results (i.e., with and without the influential cases) are reported (Aguinis, Gottfredson, & Joo, 2013).

Covariate Screening

Parent age was significantly related to parent ACE scores (total score: $r = -.26$, $p < .001$; maltreatment score: $r = -.19$, $p = .007$; household dysfunction score: $r = -.28$, $p < .001$). Parent race/ethnicity (other categories vs. White) was significantly related to parent maltreatment score, $r = .17$, $p = .019$. Parent education (no post-secondary degree vs. post-secondary degree) was significantly related to parent ACE scores (total score: $r = .31$, $p < .001$; maltreatment score: $r = .26$, $p < .001$; household dysfunction score: $r = .30$, $p < .001$). Household income (<$90,000 vs. >$90,000) was significantly related to parent ACE scores (total score: $r = .30$, $p < .001$; maltreatment score: $r = .29$, $p < .001$; household dysfunction score: $r = .24$, $p = .002$), parent chronic pain status, $\chi^2(1) = 12.90$, $p < .001$, and parent PTSD symptoms, $r = .26$, $p = .001$. Parent gender (female vs. male) and employment status (not working vs. working) were not related to any parent variables. Thus, parent age, race/ethnicity, education, and household income were included as covariates in analyses examining parent variables, with 'other categories' as the focus group and 'White' as the reference group for race/ethnicity, 'no post-secondary degree' as the focus group and 'post-secondary degree' as the reference group for education, and '<$90,000' as the focus group and '>$90,000' as the reference group for household income.

Parent age, race/ethnicity, gender, education, employment status, and household income were not related to youth pain outcomes. Youth age and gender were related to youth pain interference (age: $r = .20$, $p = .018$; gender: $r = .22$, $p = .009$). Youth race/ethnicity was not related to youth pain outcomes. Thus, youth age and gender and parent age, race/ethnicity,

education, and household income were included in analyses examining parent and youth variables, with female as the focus group and male as the reference group for youth gender.

Sample Characteristics

Sociodemographic characteristics of the sample are reported in Tables 1 (total sample) and 2 (by site). Parents ranged in age from 32 to 63 years old (M = 44.91, SD = 5.14) and were predominately female (92.3%), White (86.2%), and married or common-law (81.0%). The majority of parents had a college or University degree (69.7%), were currently employed full-time (55.4%), and had an annual household income >$90,000 CAD (60.5%). Youth ranged in age from 10 to 18 years old (M = 14.39, SD = 2.20) and were also predominately female (75.9%) and White (81.5%). Youth baseline pain characteristics are reported in Table 3. Youth reported an average pain duration of 3.40 years (SD = 3.14; range: 3 months to 17 years and 1 month). Their most frequently reported pain locations were head (67.2%), other (28.7%), and muscle and joints (26.2%). Almost half of youth reported more than one pain location (44.6%) and that their pain occurred on a daily basis (49.7%). At baseline, youth reported an average pain intensity of 5.52 (SD = 1.82) out of 10 and pain interference score of 55.85 (SD = 9.32) out of 74. Youth scores on pain intensity and pain interference were significantly higher at baseline than at follow-up, pain intensity: $t(142) = 2.21$, $p = .029$, pain interference: $t(138) = 3.55$, $p = .001$. Mean scores on study variables and correlations between study variables are reported in Table 4.

Hypothesis 1: Prevalence of ACEs in Parents of Youth with Chronic Pain

Prevalence rates of ACEs among parents are reported in Tables 5 and 6. Overall, 67.7% of parents reported one or more ACEs and 22.1% reported four or more ACEs. Their average ACE score was 2.08 (SD = 2.34) out of 10. Household dysfunction was reported at higher rates than maltreatment, with 59.0% of parents reporting at least one type of household dysfunction

and 44.6% of parents reporting at least one type of maltreatment. The most frequently reported types of household dysfunction were mental illness in the household (37.4%), parent separation/ divorce (33.3%), and problematic substance use in the household (31.3%). The most frequently reported types of maltreatment were sexual (27.7%) and emotional (24.1%) abuse.

Hypothesis 2a: Association Between Parent ACEs and Parent Chronic Pain

Logistic Regression Between Parent Total ACEs and Parent Chronic Pain Status

Table 7 reports the logistic regression results for parent total ACE score predicting parent chronic pain status. The omnibus test for the unadjusted model was significant, χ^2 (1, N = 195) = 7.43, p = .006, with parent total ACE score accounting for 5.0% of the total variance in parent chronic pain status (Nagelkerke pseudo R^2 = .05). The Wald test indicated that parent total ACE score was a significant predictor of parent chronic pain status. Specifically, for every additional ACE reported, parents were 1.19 times (95% CI [1.05, 1.36]) more likely to report chronic pain. The omnibus tests for the adjusted model were also significant, Block 1 (covariates): χ^2 (4, N = 166) = 15.05, p = .005, Block 2 (total ACE score): χ^2 (1, N = 166) = 6.71, p = .010, Model: χ^2 (5, N = 166) = 21.76, p = .001. The Nagelkerke pseudo R^2 indicated that the adjusted model, with parent total ACE score and covariates, accounted for 16.4% of the variance in parent chronic pain status. The Wald test indicated that parent total ACE score remained a significant predictor of parent chronic pain status when covariates were included in the model. Specifically, for every additional ACE reported, parents were 1.24 times (95% CI [1.05, 1.46]) more likely to report chronic pain while controlling for parent age, race/ethnicity, education, and household income.

Logistic Regression Between Parent Maltreatment and Parent Chronic Pain Status

Table 8 presents the logistic regression results for parent maltreatment score predicting parent chronic pain status. The omnibus test for the unadjusted model was significant, χ^2 (1, N =

195) = 7.51, p = .006, with parent maltreatment score accounting for 5.0% of the total variance in parent chronic pain status (Nagelkerke pseudo R^2 = .05). The Wald test indicated that parent maltreatment score was a significant predictor of parent chronic pain status. For every additional type of maltreatment reported, parents were 1.37 times (95% CI [1.08, 1.73]) more likely to report chronic pain. The omnibus tests for the adjusted model were also significant, Block 1 (covariates): χ^2 (4, N = 166) = 15.05, p = .005, Block 2 (maltreatment score): χ^2 (1, N = 166) = 5.64, p = .018, Model: χ^2 (5, N = 166) = 20.69, p = .001. The Nagelkerke pseudo R^2 indicated that the adjusted model, with parent maltreatment score and covariates, accounted for 15.6% of the variance in parent chronic pain status. The Wald test indicated that parent maltreatment score remained a significant predictor of parent chronic pain status when covariates were included in the model. Specifically, for every additional type of maltreatment reported, parents were 1.40 times (95% CI [1.05, 1.88]) more likely to report chronic pain, while controlling for parent age, race/ethnicity, education, and household income.

Logistic Regression Between Parent Household Dysfunction and Parent Chronic Pain Status

Table 9 presents the logistic regression results for parent household dysfunction score predicting parent chronic pain status. The omnibus test for the unadjusted model was significant, χ^2 (1, N = 195) = 4.40, p = .036, with parent household dysfunction accounting for 3.0% of the total variance in parent chronic pain (Nagelkerke pseudo R^2 = .03). The Wald test indicated that parent household dysfunction was a significant predictor of parent chronic pain status. For every additional household dysfunction reported, parents were 1.27 times (95% CI [1.01, 1.58]) more likely to report chronic pain. The omnibus tests for the adjusted model were also significant, Block 1 (covariates): χ^2 (4, N = 166) = 15.05, p = .005, Block 2 (household dysfunction score): χ^2 (1, N = 166) = 4.49, p = .034, Model: χ^2 (5, N = 166) = 19.54, p = .002. The Nagelkerke pseudo

R^2 indicated that the adjusted model, which included parent household dysfunction score and covariates, accounted for 14.8% of the variance in parent chronic pain status. The Wald test indicated that parent household dysfunction score remained a significant predictor of parent chronic pain status when covariates were included. For every additional type of household dysfunction reported, parents were 1.34 times (95% CI [1.02, 1.77]) more likely to report chronic pain, controlling for parent age, race/ethnicity, education, and household income.

Relations Between Parent ACEs and Parent Chronic Pain Characteristics

In total, 100 parents (51.3% of the sample) reported the presence of chronic pain. The average pain duration reported by these parents was 12.53 years (*SD* = 12.09 years; range: 4 months to 48 years). Interestingly, 15.0% of parents with chronic pain indicated their chronic pain began in childhood and 31.0% indicated their chronic pain was present before their child (i.e., the study child with chronic pain) was born. Parents with chronic pain reported an average pain intensity of 4.88 (*SD* = 2.10) out of 10 and an average of 1.82 pain locations (*SD* = 1.04) out of 6. Among the parents with chronic pain, total ACE scores and maltreatment scores were not related to pain intensity (total score: $r = .17$, $p = .086$; maltreatment score: $r = .10$, $p = .324$), pain duration (total score: $r = -.04$, $p = .688$; maltreatment score: $r = -.001$, $p = .996$) or number of pain locations (total score: $r = .15$, $p = .138$; maltreatment score: $r = .07$, $p = .483$). Household dysfunction scores were related to parent pain intensity, $r = .21$, $p = .034$, and number of pain locations, $r = .20$, $p = .046$, but not pain duration, $r = -.08$, $p = .467$.

Hypothesis 2b: Association Between Parent ACEs and Parent PTSD Symptoms

Linear Regression Between Parent Total ACEs and Parent PTSD Symptoms

Linear regression results for parent total ACE score predicting parent PTSD symptoms are reported in Table 10. The unadjusted model was significant, $F(1, 189) = 8.11$, $p = .005$, and

indicated that parent total ACE scores accounted for approximately 4% of the variance in parent PTSD symptoms (R^2 = .04, adjusted R^2 = .04). Higher parent total ACE scores predicted higher parent PTSD symptoms. The adjusted model was also significant, $F(5, 156) = 3.56$, $p = .004$, and accounted for approximately 10% of the variance in parent PTSD symptoms (R^2 = .10, adjusted R^2 = .07). However, the addition of parent total ACE score in the second step of the model did not significantly increase the predictive power of the model, $\Delta F(1, 156) = 1.97$, $p = .163$, ΔR^2 = .01, and parent total ACE score was not a significant predictor of parent PTSD symptoms when covariates were included. Instead, it was the first step of the model, which only included the covariates, that was significant, $F(4, 157) = 3.94$, $p = .005$, and accounted for the majority of the variance (R^2 = .09, adjusted R^2 = .07) in parent PTSD symptoms.

Linear Regression Between Parent Maltreatment and Parent PTSD Symptoms

Table 11 presents the linear regression results for parent maltreatment score predicting parent PTSD symptoms. The unadjusted model was significant, $F(1, 189) = 6.94$, $p = .009$, and indicated that parent maltreatment scores accounted for approximately 3.5% of the variance in parent PTSD symptoms (R^2 = .04, adjusted R^2 = .03). Overall, higher parent maltreatment scores predicted higher parent PTSD symptoms. However, when influential cases were removed, the model was not significant, $F(1, 184) = 2.25$, $p = .136$, R^2 = .01, adjusted R^2 = .01. The adjusted model was significant, $F(5, 156) = 3.38$, $p = .006$, and accounted for approximately 10% of the variance in parent PTSD symptoms (R^2 = .10, adjusted R^2 = .07). Parent maltreatment score did not significantly increase the predictive power of the model when added in the second step of the model, however, $\Delta F(1, 156) = 1.16$, $p = .284$, ΔR^2 = .01, and was not a significant predictor of parent PTSD symptoms when covariates were included. It was the first step of the model, which

included only covariates, that was significant, $F(4, 157) = 3.94$, $p = .005$, and accounted for the majority of the variance ($R^2 = .09$, adjusted $R^2 = .07$) in parent PTSD symptoms.

Linear Regression Between Parent Household Dysfunction and Parent PTSD Symptoms

Table 12 presents the linear regression results for parent household dysfunction score predicting parent PTSD symptoms. The unadjusted model was significant, $F(1, 189) = 5.81$, $p = .017$, and indicated that parent household dysfunction scores accounted for approximately 3% of the variance in parent PTSD symptoms ($R^2 = .03$, adjusted $R^2 = .03$). Higher parent household dysfunction scores predicted higher parent PTSD symptoms. The adjusted model was also significant, $F(5, 156) = 3.54$, $p = .005$, accounting for approximately 10% of the variance in parent PTSD symptoms ($R^2 = .10$, adjusted $R^2 = .07$). However, parent household dysfunction score did not significantly increase the predictive power of the model when included in the second step, $\Delta F(1, 156) = 1.87$, $p = .174$, $\Delta R^2 = .01$, and was not a unique predictor of parent PTSD symptoms when controlling for the covariates. Instead, it was the first step, which included only covariates, that was significant, $F(4, 157) = 3.94$, $p = .005$, and accounted for the majority of the variance ($R^2 = .09$, adjusted $R^2 = .07$) in parent PTSD symptoms.

Exploration of Parent ACEs as the 'Worst' Traumatic Event Experienced

The types of traumatic events reported by parents to be their worst event are presented in Table 13. Thirteen (6.7%) parents reported an ACE as their worst event. The ACEs included sexual abuse, physical abuse, physical violence between parents, and parent separation/divorce. Interestingly, 16 (8.2%) parents reported an event from their childhood that is not captured by the traditional ACE categories as their worst event. These included the death of family or friends, witnessing accidents that resulted in death, being involved in a serious accident, physical illness or medical emergency involving a parent, witnessing physical violence against a sibling, being

bullied or socially excluded, and experiencing a natural disaster or house fire. The remaining parents either reported an event from their adulthood (n = 148; 75.9%) or did not report an event (n = 18; 9.2%). In total, 11 (5.6%) parents met the clinical cut-off for PTSD.

Hypothesis 3: Mediation Analyses

Given the lack of associations between parent chronic pain status and youth pain outcomes, and between parent PTSD symptoms and youth pain intensity, mediation analyses focused on the associations between parent ACEs (i.e., total ACE score, maltreatment score, and household dysfunction score), parent PTSD symptoms, and youth pain interference. Results of the mediation analyses are displayed in Table 14 and Figures 1 through 6. Overall, results were similar across all mediation models. Specifically, there were no indirect effects of parent ACEs on youth pain interference through parent PTSD symptoms in the unadjusted or adjusted models; thus, mediation was not established in any model. In the unadjusted models, higher parent ACE scores were not related to greater parent PTSD symptoms or greater youth pain interference, but greater parent PTSD symptoms were related to greater youth pain interference. In the adjusted models, when covariates were included, all paths between parent ACEs, parent PTSD symptoms, and youth pain interference were non-significant.

Discussion

This is the first study to examine the prevalence of ACEs among parents of youth with chronic pain as well as the associations between parent ACEs, parent chronic pain and PTSD symptoms, and child pain outcomes in a clinical sample of youth with chronic pain and their parents. As expected, ACEs were found to be common among parents of youth with chronic pain and related to parent chronic pain status. Contrary to hypotheses, parent ACEs were not associated with parent PTSD symptoms or youth pain outcomes. Moreover, parent chronic pain

and parent PTSD symptoms did not mediate the association between parent ACEs and youth pain outcomes. These findings add to a growing body of research on the relation between trauma and pain that have been observed in both individuals and parent-child dyads (e.g., Asmundson, Coons, Taylor, & Katz, 2002; Davis et al., 2005; Nelson et al., 2018; Noel et al., 2016).

Despite the higher socioeconomic status of the sample, ACEs were found to be prevalent among parents of youth presenting to tertiary-level pediatric chronic pain clinics in Canada. Over two-thirds (67.7%) of parents reported one or more ACEs and almost one-quarter (22.1%) reported four or more ACEs. Moreover, approximately half of parents reported at least one type of maltreatment (44.6%) or household dysfunction (59.0%). The most frequently reported ACEs were types of household dysfunction, with approximately one-third of parents reporting mental illness in the household (37.4%), parent separation/divorce (33.3%), or problematic substance use in the household (31.3%). However, types of maltreatment were also frequently reported, with approximately one-quarter of parents reporting sexual (27.7%) or emotional (24.1%) abuse. These rates are generally higher than the rates reported in the original ACE Study. For example, 12.5% of respondents in the ACE Study reported four or more ACEs, 19.4% reported household mental illness, 23.3% reported parent separation/divorce, and 10.6% reported emotional abuse (CDC-Kaiser Permanente, 2016). However, the rates of ACEs reported in the current study are largely similar to a recent study that examined ACEs in a treatment-seeking sample of adults from the same city as the majority of the current sample (i.e., Calgary). In this study, 19.7% of participants reported four or more ACEs, 42.4% reported at least one type of maltreatment, and 60.8% reported at least one type of household dysfunction (Beveridge et al., under review). Each category of ACE was also reported at similar rates, with the exception of physical neglect, which was more prevalent in the current sample of parents than the sample of adults (14.4% vs. 9.3%).

The high prevalence of ACEs among parents of youth with chronic pain may represent heightened risk in these families. Youth with chronic pain tend to report greater symptoms of depression, anxiety, and PTSD, and are more likely to meet clinical cut-offs for these mental health disorders, than youth without chronic pain (Vinall et al., 2016). Parents of youth with chronic pain also report high rates of chronic pain as well as clinically-elevated symptoms of depression, anxiety, and PTSD (Beveridge et al., 2018; Campo et al., 2007; Eccleston et al., 2004; Noel et al., 2016; Stone et al., 2018). These findings suggest that some families may have a vulnerability to develop physical and mental health conditions. Importantly, this vulnerability appears to be more general rather than a specific vulnerability for chronic pain. Indeed, findings from a systematic review that examined a wide range of outcomes in children of parents with chronic pain suggest that the risk transmitted from a parent with chronic pain to their child is not specific to pain but is a broader risk for poor outcomes (Higgins et al., 2015). This review found that children of parents with chronic pain reported poorer general health, more hospitalizations, greater externalizing and internalizing problems, and poorer social abilities than children of parents without chronic pain. In line with these findings, a study by van Tilburg et al. (2015) found that children of mothers with irritable bowel syndrome (IBS) had more health care visits for a variety of physical health complaints (i.e., not just gastrointestinal symptoms), and had higher scores on measures of depression and anxiety, than control children. In this study, the health care utilization and mental health symptoms of mothers with IBS and their children were significantly related. Together, these results suggest that a broader vulnerability for poor health outcomes may be transmitted through families with chronic pain. This vulnerability may be due to genetic factors, such as a predisposition to an anxious temperament, but may also be caused by environmental factors, including exposure to ACEs. That is, ACEs may initiate a developmental

cascade that alters biological and psychological processes in the parent, increasing their risk for poor health outcomes, which in turn, increases their child's risk for poor outcomes.

In line with this suggestion, parent ACEs (including their total ACE score, maltreatment score, and household dysfunction score) were significant predictors of parent chronic pain status in the current sample. Specifically, for every additional type of ACE reported, parents were 1.19 to 1.40 times more likely to report chronic pain. These results are consistent with a large body of research that has shown ACEs confer risk for the occurrence of chronic pain conditions in adults (Anda et al., 2010; Davis et al., 2005; Olivieri et al., 2012; Park et al., 2016; Scott et al., 2011). Contrary to some studies (Dennis et al., 2019; McCall-Hosenfeld et al., 2014), total ACE scores and maltreatment scores were not associated with the severity of parents' chronic pain (i.e., pain intensity, pain duration, number of pain locations). Unexpectedly, household dysfunction scores were positively related to the pain intensity and number of pain locations reported by parents with chronic pain. However, these correlations were small (i.e., *r*s = .20 and .21). Overall, these results suggest that ACEs may be more closely related to the occurrence of chronic pain than its severity in parents of youth with chronic pain.

Contrary to hypotheses, parent ACEs were not related to their current PTSD symptoms when key sociodemographic variables were controlled for in the analyses. Parent scores on the measure of PTSD symptoms were generally low (i.e., *M* = 10.69 [*SD* = 12.39] out of 80) and only 5.6% of parents met the clinical cut-off for a diagnosis of PTSD. Thus, clinically-elevated PTSD symptoms may not have been as prevalent in the current sample as previous studies that have found an association between ACEs and current PTSD symptoms (Atzl et al., 2019; Grasso et al., 2016; Leardmann et al., 2010; Schalinski et al., 2016; Wu et al., 2010). Of note, most of these previous studies included high risk samples (e.g., psychiatric inpatients, military personnel,

low income women) who may be more likely to experience traumatic events that meet the diagnostic criteria for PTSD (i.e., exposure to actual or threatened death, serious injury, or sexual violence; American Psychiatric Association, 2013), and may also be more at risk to develop clinically-elevated PTSD symptoms, than the current sample of parents. The percentage of parents that met the clinical cut-off for PTSD in the current study was also lower than a previous study from the United States that found that 20% of parents of youth with chronic pain met the clinical cut-off for PTSD (Noel et al., 2016). Thus, research with other samples of parents of youth with chronic pain (e.g., from other geographical regions) may reveal associations between parent ACEs and parent PTSD symptoms that differ from the current study.

Several studies have also shown that PTSD can mediate and/or moderate the association between childhood adversity and chronic pain in adulthood. For example, a study by Beal et al. (2020) found that PTSD symptoms in adolescence mediated the association between childhood maltreatment and pain in young adulthood among women with substantiated reports of childhood maltreatment. Another study by Raphael and Widom (2011) found that PTSD diagnosed in young adulthood moderated the association between childhood maltreatment and pain in middle adulthood, such that adults with a history of both maltreatment and PTSD reported significantly more pain symptoms, pain problems, and pains related to illness or injury than adults without a history of both maltreatment and PTSD. Thus, parent PTSD symptoms may be a more important mediator/moderator of the parent ACEs-parent chronic pain relation than the parent ACEs-child chronic pain relation. Indeed, parent PTSD symptoms were significantly related to parent chronic pain status in the current study. Future prospective research is needed to extend the above findings into the next generation, and more comprehensively examine the cascade from parent ACEs to parent lifetime PTSD to parent chronic pain to child chronic pain.

Parent ACEs were not associated with youth pain outcomes in the current study, either directly or indirectly through parent chronic pain or parent PTSD symptoms, suggesting that parent ACEs may not be related to the *maintenance* of chronic pain and related impairment in youth. Instead, similar to the finding that parent ACEs were related to the occurrence but not severity of parent chronic pain, parent ACEs may be more related to the *development* of child chronic pain. First, research on the intergenerational impact of ACEs has shown that parent ACEs are related to child factors that may increase children's risk for developing chronic pain, including poorer general health and more internalizing behaviours (Dennis et al., 2019; Lê-Scherban et al., 2018; Madigan et al., 2017; Schickedanz et al., 2018). Both of these outcomes have been posited as risk factors for the development of pediatric chronic pain (McKillop & Banez, 2016; Stone & Wilson, 2016). Moreover, research has shown that children of parents with chronic pain are at risk for developing their own chronic pain (Higgins et al., 2015). In the current study, almost one-third of parents with chronic pain reported that their pain began before their child was born. Thus, at least for some families in the current study, parent chronic pain status may have contributed to the *onset* of the child's chronic pain, likely through biological (e.g., disrupted fetal development) and psychosocial (e.g., parent modeling of pain behaviours) mechanisms. In this way, parent chronic pain status may play a more important mediating role in the relation between parent ACEs and the *development* of chronic pain and impairment in youth.

Moreover, the current study investigated the simple mediating roles of two parent factors that have been implicated in the maintenance of pediatric chronic pain (i.e., chronic pain and PTSD symptoms). However, the indirect effect of parent ACEs on child chronic pain outcomes may be more complex and involve other factors. Indeed, recent studies that have used structural equation modelling to examine factors contributing to the maintenance of chronic pain in youth

have found that parent health (e.g., global physical health, global mental health, chronic pain status) is indirectly related to the functioning of youth with chronic pain through cognitive and behavioural factors (e.g., parent catastrophizing about child pain, parent protective responses to child pain, parent pain behaviours, child pain catastrophizing, child pain threat appraisals; Birnie et al., 2020; Stone et al., 2018). ACEs may impact parent cognitive and behavioural factors, either directly or indirectly through experiences with their own health conditions. For example, research has shown that parents with chronic pain and parents with higher distress (including greater PTSD symptoms) report higher levels of catastrophizing about their child's pain and more maladaptive responses to their child's pain than parents without chronic pain or with lower levels of distress (Birnie et al., 2020; Neville et al., 2018; Sieberg, Williams, & Simons, 2011; Stone et al., 2018). Thus, future research should examine more comprehensive models, that include these key parent factors, to better understand the pathways that may lead from parent ACEs to poor outcomes for youth with chronic pain. In particular, research that uses daily diaries or ecological momentary assessment may help elucidate nuanced associations between parent ACEs, parent responses, and youth pain outcomes. A recent study that used daily diaries found that parent catastrophizing about child pain at baseline moderated the daily associations between parent behavioural responses and child pain outcomes (Neville et al., 2020). It may be that parent ACEs influence the day-to-day responses of parents to their child's chronic pain.

The hypothesis that parent maltreatment scores would be stronger predictors of parent and child outcomes than parent household dysfunction scores was not supported in the current study. In fact, parent experiences of maltreatment had a comparable relation with parent chronic pain status, parent PTSD symptoms, and youth pain outcomes as parent experiences of household dysfunction. These findings are inconsistent with emerging research that has found

maltreatment to be a stronger predictor of poor health outcomes in adults, including chronic pain and elevated PTSD symptoms, than household dysfunction (Atzl et al., 2019; Beal et al., 2020) but are in line with other studies that have found that individual types of household dysfunction (e.g., mental illness in the household) have similar relations with chronic pain and PTSD as individual types of maltreatment (e.g., sexual abuse; Gonzalez et al., 2012; Park et al., 2016; Schalinski et al., 2016; Scott et al., 2011). For example, a study by Anda et al. (2010) found that the odds ratios for individual ACEs predicting frequent headaches in adults ranged from 1.2 to 1.4 (95% CIs [1.1, 1.5]) for types of maltreatment (i.e., physical and sexual abuse) and types of household dysfunction (i.e., witnessing domestic violence, problematic substance use and mental illness in the household, household member imprisoned). Thus, the literature is currently mixed in regard to the impact that specific types of ACEs have on later physical and mental health.

Clinical Implications

The current study demonstrated that ACEs are common among parents of youth seeking treatment for their chronic pain. These results add to a growing body of research that has found that experiences of adversity and symptoms of traumatic stress are prevalent among youth with chronic pain as well as their parents (Nelson et al., 2018; Noel et al., 2016), and suggest that the assessment and treatment of trauma in this population may be needed. It is important to note, however, that many researchers and clinicians caution against routine screening for ACEs in health care settings given the potential for patients to be retraumatized as well as the current lack of evidence-based interventions for ACEs (Finkelhor, 2018; Racine, Killam, & Madigan, 2020). Moreover, several researchers, including one of the lead investigators of the original ACE Study, have noted that the ACE Questionnaire and the ‘ACE score’ are limited in their ability to identify childhood adversity and its risk for health outcomes (Anda, Porter, & Brown, 2020; Finkelhor,

2018; Racine et al., 2020). For instance, the 10 categories of ACEs that are traditionally assessed were not chosen through a rigorous scientific process and thus may not be the most (or the only) critical events to assess. In line with this critique, the current study found that, when asked about the worst traumatic event experienced in their life, many parents reported childhood events that are not captured by the ACE Questionnaire including the death of a family member, being in a serious accident, and experiencing a natural disaster. Thus, instead of a narrow focus on ACEs, Racine and colleagues (2020) have recommended that health care settings adopt the broader approach of trauma-informed care, which 1) realizes the high prevalence of trauma and its effect on health, 2) recognizes how trauma can present in individuals, and 3) responds in a matter that resists retraumatization (Substance Abuse and Mental Health Services Administration, 2014). Given previous research that has found high rates of ACEs in clinical samples of youth with chronic pain, as well as the current finding that parents of these youth also report high rates of ACEs, a trauma-informed approach may be particularly important in tertiary-level pediatric chronic pain clinics.

Strengths and Limitations

A strength of the current study was the use of well-validated and widely-used self-report measures for the key study variables, as these features help ensure that the findings are accurate and can be compared to other studies that have used the same measures. As well, the temporal ordering of study variables in the current study allowed for a longitudinal examination of the indirect effect of parent ACEs (occurring in childhood) on youth pain outcomes (at 3-month follow-up) through parent chronic pain status and PTSD symptoms (at baseline). The use of longitudinal, as opposed to cross-sectional, data has been recommended for mediation analyses as it allows for a stronger examination of the *temporal* relations between variables (Jose, 2016).

Nonetheless, self-report measures are subject to response biases (e.g., social desirability) that may impact the accuracy of the results. In particular, concerns have been raised about using self-report measures to retrospectively assess ACEs (Widom, Raphael, & DuMont, 2004). For example, it has been suggested that the current mental or physical health status of the respondent could influence his or her responses on the ACE measure and a review of the relevant literature found that adults tend to underestimate their experiences of childhood adversity (Hardt & Rutter, 2004). However, this review cautioned against the complete dismissal of retrospective reports of childhood adversity and suggested that clear operationalization of adversities and high-quality measurement methods can improve the accuracy of these reports. Moreover, recent research with a sample similar to the current sample (i.e., treatment-seeking adults in Calgary) found that scores on the ACE measure were not influenced by current depressive symptoms (Frampton et al., 2018). Prospective, longitudinal research that examines the developmental cascade from parent childhood adversity to parent mental and physical health to child chronic pain could help confirm the current results.

In addition, the results of the current study may not generalize to other populations, such as youth (and their parents) who are *not* receiving tertiary-level treatment for their chronic pain. Indeed, these results may not be applicable to youth with chronic pain that is less severe and does not require treatment from a multidisciplinary team. However, the results may be *more* robust for families who do not have the resources to seek out a referral for tertiary-level treatment for the child's chronic pain. The current sample was also predominately White, female, and of higher socioeconomic status. Future research that examines the role of parent ACEs in pediatric chronic pain in a more diverse sample from the general population is needed to explicate the relevance of the current findings to a broader population of youth with chronic pain.

Conclusions and Future Directions

In line with previous studies that have found high rates of ACEs among individuals with chronic pain (Dennis et al., 2019; Nelson et al., 2018), the current study found that ACEs were also prevalent among *parents* of youth with chronic pain. Given that ACEs are associated with poor outcomes for the individual and their child (Hughes et al., 2017; Lê-Scherban et al., 2018; Liming & Grube, 2018; Oh et al., 2018; Plant et al., 2018), the prevalence of ACEs in parents of youth with chronic pain may signify a vulnerability in these families for poor health outcomes. Consistent with this suggestion, parent ACEs were found to be associated with parent chronic pain status. However, parent ACEs were not related to parent PTSD symptoms or youth pain outcomes, and parent chronic pain status and PTSD symptoms did not mediate the association between parent ACEs and youth pain outcomes. Parent ACEs may play a more important role in the *development* of child chronic pain than its *maintenance*. Indeed, parent ACEs may be a distal risk factor for the intergenerational transmission of risk for chronic pain, increasing parent risk for chronic pain and, in turn, child risk for chronic pain. Further research on the role of childhood adversity in chronic pain is needed. Specifically, given the substantial body of research that has found an association between ACEs and chronic pain, research that identifies the neurobiological and psychosocial mechanisms mediating this association, in individuals and parent-child dyads, is needed to inform interventions that prevent chronic pain. Since ACEs are not deterministic of poor outcomes, with factors such as supportive relationships moderating the intergenerational continuity of ACEs (Schofield, Lee, & Merrick, 2013), research that identifies risk (e.g., elevated mental health symptoms) *and* protective (e.g., positive childhood experiences; Bethell, Jones, Gombojav, Linkenbach, & Sege, 2019) factors will be crucial for designing interventions that halt the continuation of poor health outcomes across generations.

References

Aguinis, H., Gottfredson, R. K., & Joo, H. (2013). Best-practice recommendations for defining, identifying, and handling outliers. *Organizational Research Methods*, *16*(2), 270–301. doi:10.1177/1094428112470848

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Association.

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., … Giles, W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood: A convergence of evidence from neurobiology and epidemiology. *European Archives of Psychiatry and Clinical Neuroscience*, *256*(3), 174–186. doi:10.1007/s00406-005-0624-4

Anda, R. F., Porter, L. E., & Brown, D. W. (2020). Inside the Adverse Childhood Experience score: Strengths, limitations, and misapplications. *American Journal of Preventive Medicine*, *59*(2), 293–295. doi:10.1016/j.amepre.2020.01.009

Anda, R. F., Tietjen, G., Schulman, E., Felitti, V., & Croft, J. (2010). Adverse childhood experiences and frequent headaches in adults. *Headache*, *50*(9), 1473–1481. doi:10.1111/j.1526-4610.2010.01756.x

Arruda, M. A., Guidetti, V., Galli, F., Albuquerque, R. C. A. P., & Bigal, M. E. (2010). Frequency of headaches in children is influenced by headache status in the mother. *Headache*, *50*(6), 973–980. doi:10.1111/j.1526-4610.2010.01677.x

Asmundson, G. J. G., Coons, M. J., Taylor, S., & Katz, J. (2002). PTSD and the experience of pain: Research and clinical implications of shared vulnerability and mutual maintenance models. *Canadian Journal of Psychiatry*, *47*(10), 930–937. doi:10.1177/070674370204701004

Atzl, V. M., Narayan, A. J., Rivera, L. M., & Lieberman, A. F. (2019). Adverse childhood experiences and prenatal mental health: Type of ACEs and age of maltreatment onset. *Journal of Family Psychology*, *33*(3), 304–314. doi:10.1037/fam0000510

Bandura, A. (1977). *Social learning theory*. Englewood Cliffs, NJ: Prentice Hall.

Beal, S. J., Kashikar-Zuck, S., King, C., Black, W., Barnes, J., & Noll, J. G. (2020). Heightened risk of pain in young adult women with a history of childhood maltreatment: A prospective longitudinal study. *Pain*, *161*(1), 156–165. doi:10.1097/j.pain.0000000000001706

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., … Zule, W. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse and Neglect*, *27*(2), 169–190. doi:10.1016/S0145-2134(02)00541-0

Bethell, C., Jones, J., Gombojav, N., Linkenbach, J., & Sege, R. (2019). Positive childhood experiences and adult mental and relational health in a statewide sample: Associations across adverse childhood experiences levels. *JAMA Pediatrics*, *173*(11), e193007. doi:10.1001/jamapediatrics.2019.3007

Beveridge, J. K., Dobson, K. S., Madigan, S., Yeates, K. O., Stone, A. L., Wilson, A. C., … Noel, M. (under review). Adverse childhood experiences in parents of youth with chronic pain: Prevalence and comparison to a community-based sample.

Beveridge, J. K., Neville, A., Wilson, A. C., & Noel, M. (2018). Intergenerational examination of pain and posttraumatic stress disorder symptoms among youth with chronic pain and their parents. *Pain Reports*, *3*, e667. doi:10.1097/PR9.0000000000000667

Birnie, K. A., Heathcote, L. C., Bhandari, R. P., Feinstein, A., Yoon, I. A., & Simons, L. E. (2020). Parent physical and mental health contributions to interpersonal fear avoidance

processes in pediatric chronic pain. *Pain*, *161*(6), 1202–1211. doi:10.1097/j.pain.0000000000001820

Blevins, C. A., Weathers, F. W., Davis, M. T., Witte, T. K., & Domino, J. L. (2015). The Posttraumatic Stress Disorder Checklist for DSM-5 (PCL-5): Development and initial psychometric evaluation. *Journal of Traumatic Stress*, *28*, 489–498. doi:10.1002/jts

Campo, J. V., Bridge, J., Lucas, A., Savorelli, S., Walker, L., Di Lorenzo, C., … Brent, D. A. (2007). Physical and emotional health of mothers of youth with functional abdominal pain. *Archives of Pediatrics and Adolescent Medicine*, *161*(2), 131–137. doi:10.1001/archpedi.161.2.131

Centers for Disease Control and Prevention-Kaiser Permanente. (2016). *The ACE Study survey data*. https://www.cdc.gov/violenceprevention/childabuseandneglect/acestudy/about.html

Claar, R. L., Simons, L. E., & Logan, D. E. (2008). Parental response to children's pain: The moderating impact of children's emotional distress on symptoms and disability. *Pain*, *138*(1), 172–179. doi:10.1016/j.pain.2007.12.005

Cooke, J. E., Racine, N., Plamondon, A., Tough, S., & Madigan, S. (2019). Maternal adverse childhood experiences, attachment style, and mental health: Pathways of transmission to child behavior problems. *Child Abuse and Neglect*, *93*, 27–37. doi:10.1016/j.chiabu.2019.04.011

Creech, S. K., & Misca, G. (2017). Parenting with PTSD: A review of research on the influence of PTSD on parent-child functioning in military and veteran families. *Frontiers in Psychology*, *8*, 1101. doi:10.3389/fpsyg.2017.01101

Cunningham, N. R., Lynch-Jordan, A., Barnett, K., Peugh, J., Sil, S., Goldschneider, K., & Kashikar-Zuck, S. (2014). Child pain catastrophizing mediates the relation between parent

responses to pain and disability in youth with functional abdominal pain. *Gastroenterology*, *59*(6), 732–738. doi:10.1097/MPG.0000000000000529

Davis, D. A., Luecken, L. J., & Zautra, A. J. (2005). Are reports of childhood abuse related to the experience of chronic pain in adulthood? A meta-analytic review of the literature. *Clinical Journal of Pain*, *21*(5), 398–405. doi:10.1097/01.ajp.0000149795.08746.31

Dennis, C. H., Clohessy, D. S., Stone, A. L., Darnall, B. D., & Wilson, A. C. (2019). Adverse childhood experiences in mothers with chronic pain and intergenerational impact on children. *The Journal of Pain*, *20*(10), 1209–1217. doi:10.1016/j.jpain.2019.04.004

Dong, M., Anda, R. F., Felitti, V. J., Dube, S. R., Williamson, D. F., Thompson, T. J., … Giles, W. H. (2004). The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse and Neglect*, *28*(7), 771–784. doi:10.1016/j.chiabu.2004.01.008

Donnelly, T. J., Palermo, T. M., & Newton-John, T. R. O. (2020). Parent cognitive, behavioural, and affective factors and their relation to child pain and functioning in pediatric chronic pain. *Pain*, *161*(7), 1401–1419. doi:10.1097/j.pain.0000000000001833

Dube, S. R., Williamson, D. F., Thompson, T., Felitti, V. J., & Anda, R. F. (2004). Assessing the reliability of retrospective reports of adverse childhood experiences among adult HMO members attending a primary care clinic. *Child Abuse and Neglect*, *28*(7), 729–737. doi:10.1016/j.chiabu.2003.08.009

Eccleston, C., Crombez, G., Scotford, A., Clinch, J., & Connell, H. (2004). Adolescent chronic pain: Patterns and predictors of emotional distress in adolescents with chronic pain and their parents. *Pain*, *108*(3), 221–229. doi:10.1016/j.pain.2003.11.008

Eccleston, C., Fisher, E., Law, E., Bartlett, J., & Palermo, T. M. (2015). Psychological

interventions for parents of children and adolescents with chronic illness. *Cochrane Database of Systematic Reviews*. doi:10.1002/14651858.CD009660.pub3

Evans, S., Meldrum, M., Tsao, J. C. I., Fraynt, R., & Zeltzer, L. K. (2010). Associations between parent and child pain and functioning in a pediatric chronic pain sample: A mixed methods approach. *International Journal on Disability and Human Development*, *9*(1), 11–21. doi:10.1515/IJDHD.2010.003

Faul, F., Erdfelder, E., Buchner, A., & Lang, A. G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*, *41*(4), 1149–1160. doi:10.3758/BRM.41.4.1149

Fearon, P., & Hotopf, M. (2001). Relation between headache in childhood and physical and psychiatric symptoms in adulthood: National birth cohort study. *BMJ*, *322*, 1–6. doi:10.1136/bmj.322.7295.1145

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., … Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, *14*(4), 245–258. doi:10.1177/002248717802900215

Field, A. (2018). *Discovering statistics using IBM SPSS Statistics* (5th ed.). Thousand Oaks, CA: SAGE Publications.

Finkelhor, D. (2018). Screening for adverse childhood experiences (ACEs): Cautions and suggestions. *Child Abuse and Neglect*, *85*, 174–179. doi:10.1016/j.chiabu.2017.07.016

Fisher, E., Law, E., Dudeney, J., Palermo, T. M., Stewart, G., & Eccleston, C. (2018). Psychological therapies for the management of chronic and recurrent pain in children and

adolescents. *Cochrane Database of Systematic Reviews*. doi:10.1002/14651858.cd003968

Ford, D. C., Merrick, M. T., Parks, S. E., Breiding, M. J., Gilbert, L. K., Edwards, V. J., … Thompson, W. W. (2014). Examination of the factorial structure of adverse childhood experiences and recommendations for three subscale scores. *Psychology of Violence*, *4*(4), 432–444. doi:10.1037/a0037723

Frampton, N. M. A., Poole, J. C., Dobson, K. S., & Pusch, D. (2018). The effects of adult depression on the recollection of adverse childhood experiences. *Child Abuse and Neglect*, *86*, 45–54. doi:10.1016/j.chiabu.2018.09.006

Gonzalez, A., Boyle, M. H., Kyu, H. H., Georgiades, K., Duncan, L., & MacMillan, H. L. (2012). Childhood and family influences on depression, chronic physical conditions, and their comorbidity: Findings from the Ontario Child Health Study. *Journal of Psychiatric Research*, *46*(11), 1475–1482. doi:10.1016/j.jpsychires.2012.08.004

Grasso, D. J., Dierkhising, C. B., Branson, C. E., Ford, J. D., & Lee, R. (2016). Developmental patterns of adverse childhood experiences and current symptoms and impairment in youth referred for trauma-specific services. *Journal of Abnormal Child Psychology*, *44*(5), 871–886. doi:10.1007/s10802-015-0086-8

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: Review of the evidence. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *45*(2), 260–273. doi:10.1111/j.1469-7610.2004.00218.x

Harris, P. A., Taylor, R., Minor, B. L., Elliott, V., Fernandez, M., O'Neal, L., … Duda, S. N. (2019). The REDCap consortium: Building an international community of software platform partners. *Journal of Biomedical Informatics*, *95*, 103208. doi:10.1016/j.jbi.2019.103208

Harris, P. A., Taylor, R., Thielke, R., Payne, J., Gonzalez, N., & Conde, J. G. (2009). Research Electronic Data Capture (REDCap) - A metadata-driven methodology and workflow process for providing translation informatics support. *Journal of Biomedical Informatics*, *42*(2), 377–381. doi:10.1016/j.jbi.2008.08.010

Hayes, A. F. (2017). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach* (2nd ed.). New York, NY: Guilford Publications.

Hestbaek, L., Iachine, I. A., Leboeuf-Yde, C., Kyvik, K. O., & Manniche, C. (2007). Heredity of low back pain in a young population: A classical twin study. *Twin Research*, *7*(01), 16–26. doi:10.1375/twin.7.1.16

Higgins, K. S., Birnie, K. A., Chambers, C. T., Wilson, A. C., Caes, L., Clark, A. J., … Campbell-Yeo, M. (2015). Offspring of parents with chronic pain: A systematic review and meta-analysis of pain, health, psychological, and family outcomes. *Pain*, *156*, 2256–2266. doi:10.1097/j.pain.0000000000000293

Hocking, L. J., Morris, A. D., Dominiczak, A. F., Porteous, D. J., & Smith, B. H. (2012). Heritability of chronic pain in 2195 extended families. *European Journal of Pain (United Kingdom)*, *16*(7), 1053–1063. doi:10.1002/j.1532-2149.2011.00095.x

Hogan, M. E., Taddio, A., Katz, J., Shah, V., & Krahn, M. (2016). Incremental health care costs for chronic pain in Ontario, Canada: A population-based matched cohort study of adolescents and adults using administrative data. *Pain*, *157*(8), 1626–1633. doi:10.1097/j.pain.0000000000000561

Hughes, K., Bellis, M. A., Hardcastle, K. A., Sethi, D., Butchart, A., Mikton, C., … Dunne, M. P. (2017). The effect of multiple adverse childhood experiences on health: A systematic review and meta-analysis. *The Lancet Public Health*, *2*(8), e356–e366. doi:10.1016/S2468-

2667(17)30118-4

Huguet, A., & Miró, J. (2008). The severity of chronic pediatric pain: An epidemiological study. *Journal of Pain*, *9*(3), 226–236. doi:10.1016/j.jpain.2007.10.015

Jose, P. E. (2016). The merits of using longitudinal mediation. *Educational Psychologist*, *51*(3–4), 331–341. doi:10.1080/00461520.2016.1207175

Juang, K. D., Wang, S. J., Fuh, J. L., Lu, S. R., & Chen, Y. S. (2004). Association between adolescent chronic daily headache and childhood adversity: A community-based study. *Cephalalgia*, *24*(1), 54–59. doi:10.1111/j.1468-2982.2004.00643.x

Kalmakis, K. A., Chiodo, L. M., Kent, N., & Meyer, J. S. (2020). Adverse childhood experiences, post-traumatic stress disorder symptoms, and self-reported stress among traditional and nontraditional college students. *Journal of American College Health*, *68*(4), 411–418. doi:10.1080/07448481.2019.1577860

Kashikar-Zuck, S., Carle, A., Barnett, K., Goldschneider, K. R., Sherry, D. D., Mara, C. A., … DeWitt, E. M. (2016). Longitudinal evaluation of Patient Reported Outcomes Measurement Information Systems (PROMIS) measures in pediatric chronic pain. *Pain*, *157*(2), 339–347. doi:10.1097/j.pain.0000000000000378

Kendall-Tackett, K. (2002). The health effects of childhood abuse: Four pathways by which abuse can influence health. *Child Abuse and Neglect*, *26*(6–7), 715–729. doi:10.1016/S0145-2134(02)00343-5

King, S., Chambers, C. T., Huguet, A., MacNevin, R. C., McGrath, P. J., Parker, L., & MacDonald, A. J. (2011). The epidemiology of chronic pain in children and adolescents revisited: A systematic review. *Pain*, *152*(12), 2729–2738. doi:10.1016/j.pain.2011.07.016

Laurell, K., Larsson, B., & Eeg-Olofsson, O. (2005). Headache in schoolchildren: Association

with other pain, family history and psychosocial factors. *Pain*, *119*(1–3), 150–158. doi:10.1016/j.pain.2005.09.030

Law, E. F., Fisher, E., Howard, W. J., Levy, R., Ritterband, L., & Palermo, T. M. (2017). Longitudinal change in parent and child functioning after internet-delivered cognitive-behavioral therapy for chronic pain. *Pain*, *158*, 1992–2000. doi:10.1097/j.pain.0000000000000999

Lê-Scherban, F., Wang, X., Boyle-Steed, K. H., & Pachter, L. M. (2018). Intergenerational associations of parent adverse childhood experiences and child health outcomes. *Pediatrics*, *141*(6), e20174274. doi:10.1542/peds.2017-4274

Leardmann, C. A., Smith, B., & Ryan, M. A. (2010). Do adverse childhood experiences increase the risk of postdeployment posttraumatic stress disorder in US Marines? *BMC Public Health*, *10*, 437. doi:10.1186/1471-2458-10-437

Levy, R. L. (2011). Exploring the intergenerational transmission of illness behavior: From observations to experimental intervention. *Annals of Behavioral Medicine*, *41*(2), 174–182. doi:10.1007/s12160-010-9254-9

Lewandowski, A. S., Palermo, T. M., Stinson, J., Handley, S., & Chambers, C. T. (2010). Systematic review of family functioning in families of children and adolescents with chronic pain. *Journal of Pain*, *11*(11), 1027–1038. doi:10.1016/j.jpain.2010.04.005

Liming, K. W., & Grube, W. A. (2018). Wellbeing outcomes for children exposed to multiple adverse experiences in early childhood: A systematic review. *Child and Adolescent Social Work Journal*, *35*(4), 317–335. doi:10.1007/s10560-018-0532-x

Madigan, S., Wade, M., Plamondon, A., & Jenkins, J. (2015). Maternal abuse history, postpartum depression, and parenting: Links with preschoolers' internalizing problems.

Infant Mental Health Journal, *36*(2), 146–155. doi:10.1002/imhj.20077

Madigan, S., Wade, M., Plamondon, A., Maguire, J. L., & Jenkins, J. M. (2017). Maternal adverse childhood experience and infant health: Biomedical and psychosocial risks as intermediary mechanisms. *Journal of Pediatrics*, *187*, 282–289. doi:10.1016/j.jpeds.2017.04.052

McCall-Hosenfeld, J., Winter, M., Heeren, T., & Liebschutz, J. M. (2014). The association of interpersonal trauma with somatic symptom severity in a primary care population with chronic pain: Exploring the role of gender and the mental health sequelae of trauma. *Journal of Psychosomatic Research*, *77*(3), 196–204. doi:10.1016/j.jpsychores.2014.07.011

McDonnell, C. G., & Valentino, K. (2016). Intergenerational effects of childhood trauma: Evaluating pathways among maternal ACEs, perinatal depressive symptoms, and infant outcomes. *Child Maltreatment*, *21*(4), 317–326. doi:10.1177/1077559516659556

McKillop, H., & Banez, G. (2016). A broad consideration of risk factors in pediatric chronic pain: Where to go from here? *Children*, *3*(4), 38. doi:10.3390/children3040038

Merlijn, V. P., Hunfeld, J. A., Van Der Wouden, J. C., Hazebroek-Kampschreur, A. A. J. M., Koes, B. W., & Passchier, J. (2003). Psychosocial factors associated with chronic pain in adolescents. *Pain*, *101*, 33–43. doi:10.1016/S0304-3959(02)00289-0

Mersky, J. P., Janczewski, C. E., & Topitzes, J. (2017). Rethinking the measurement of adversity: Moving toward second-generation research on adverse childhood experiences. *Child Maltreatment*, *22*(1), 58–68. doi:10.1177/1077559516679513

Meyers, L. S., Gamst, G., & Guarino, A. J. (2017). *Applied multivariate research: Design and interpretation* (3rd ed.). Thousand Oaks, CA: SAGE Publications.

Min, M. O., Minnes, S., Kim, H., & Singer, L. T. (2013). Pathways linking childhood

maltreatment and adult physical health. *Child Abuse and Neglect*, *37*(6), 361–373. doi:10.1016/j.chiabu.2012.09.008

National Center for PTSD. (n.d.). *PTSD checklist for DSM-5 (PCL-5)*. http://www.ptsd.va.gov/professional/

Nelson, S., Simons, L. E., & Logan, D. (2018). The incidence of adverse childhood experiences (ACEs) and their association with pain-related and psychosocial impairment in youth with chronic pain. *Clinical Journal of Pain*, *34*(5), 402–408. doi:10.1097/AJP.0000000000000549

Neville, A., Griep, Y., Palermo, T. M., Vervoort, T., Schulte, F., Yeates, K. O., … Noel, M. (2020). A "dyadic dance": Pain catastrophizing moderates the daily relationships between parent mood and protective responses and child chronic pain. *Pain*, *161*, 1072–1082. doi:10.1097/j.pain.0000000000001799

Neville, A., Soltani, S., Pavlova, M., & Noel, M. (2018). Unravelling the relationship between parent and child PTSD and pediatric chronic pain: The mediating role of pain catastrophizing. *Journal of Pain*, *19*(2), 196–206. doi:10.1016/j.jpain.2017.10.004

Noel, M., Wilson, A. C., Lewandowski Holley, A., Durkin, L., Patton, M., & Palermo, T. M. (2016). Post-traumatic stress disorder symptoms in youth with versus without chronic pain. *Pain*, *157*(10), 2277–2284. doi:10.1097/j.pain.0000000000000642

Oh, D. L., Jerman, P., Silvério Marques, S., Koita, K., Purewal Boparai, S. K., Burke Harris, N., & Bucci, M. (2018). Systematic review of pediatric health outcomes associated with childhood adversity. *BMC Pediatrics*, *18*, 83. doi:10.1186/s12887-018-1037-7

Olivieri, P., Solitar, B., & Dubois, M. (2012). Childhood risk factors for developing fibromyalgia. *Open Access Rheumatology: Research and Reviews*, *4*, 109–114.

doi:10.2147/OARRR.S36086

Osborne, R. B., Hatcher, J. W., & Richtsmeier, A. J. (1989). The role of social modeling in unexplained pediatric pain. *Journal of Pediatric Psychology*, *14*(1), 43–61. doi:10.1093/jpepsy/14.1.43

Palermo, T. M. (2000). Impact of recurrent and chronic pain on child and family daily functioning: A critical review of the literature. *Developmental and Behavioral Pediatrics*, *21*(1), 58–69. doi:10.1097/00004703-200002000-00011

Palermo, T. M., & Eccleston, C. (2009). Parents of children and adolescents with chronic pain. *Pain*, *146*(1), 15–17. doi:10.1016/j.pain.2009.05.009

Palermo, T. M., Witherspoon, D., Valenzuela, D., & Drotar, D. D. (2004). Development and validation of the Child Activity Limitations Interview: A measure of pain-related functional impairment in school-age children and adolescents. *Pain*, *109*(3), 461–470. doi:10.1016/j.pain.2004.02.023

Park, S. H., Videlock, E. J., Shih, W., Presson, A. P., Mayer, E. A., & Chang, L. (2016). Adverse childhood experiences are associated with irritable bowel syndrome and gastrointestinal symptom severity. *Neurogastroenterology and Motility*, *28*(8), 1252–1260. doi:10.1111/nmo.12826

Pedhazur, E. J. (1997). *Multiple regression in behavioral research* (3rd ed.). Fort Worth, TX: Harcourt Brace.

Petruccelli, K., Davis, J., & Berman, T. (2019). Adverse childhood experiences and associated health outcomes: A systematic review and meta-analysis. *Child Abuse and Neglect*, *97*, 104127. doi:10.1016/j.chiabu.2019.104127

Plant, D. T., Pawlby, S., Pariante, C. M., & Jones, F. W. (2018). When one childhood meets

another – maternal childhood trauma and offspring child psychopathology: A systematic review. *Clinical Child Psychology and Psychiatry*, *23*(3), 483–500. doi:10.1177/1359104517742186

Poole, J. C., Dobson, K. S., & Pusch, D. (2017). Childhood adversity and adult depression: The protective role of psychological resilience. *Child Abuse & Neglect*, *64*, 89–100. doi:10.1016/j.chiabu.2016.12.012

Poppert Cordts, K. M., Stone, A. L., Beveridge, J. K., Wilson, A. C., & Noel, M. (2019). The (parental) whole is greater than the sum of its parts: A multifactorial model of parent factors in pediatric chronic pain. *Journal of Pain*, *20*(7), 786–795. doi:10.1016/j.jpain.2019.01.004

Provençal, N., & Binder, E. B. (2015). The effects of early life stress on the epigenome: From the womb to adulthood and even before. *Experimental Neurology*, *268*, 10–20. doi:10.1016/j.expneurol.2014.09.001

Racine, N., Killam, T., & Madigan, S. (2020). Beyond the Adverse Childhood Experiences Questionnaire: Trauma-informed care as a universal precaution. *JAMA Pediatrics*, *174*(1), 5–6. doi:10.1093/pch/pxz043

Racine, N., Plamondon, A., Madigan, S., McDonald, S., & Tough, S. (2018). Maternal adverse childhood experiences and infant development. *Pediatrics*, *141*(4), e20172495. doi:10.1542/peds.2017-2495

Raphael, K. G., & Widom, C. S. (2011). Post-traumatic stress disorder moderates the relation between documented childhood victimization and pain 30 years later. *Pain*, *152*(1), 163–169. doi:10.1016/j.pain.2010.10.014

Sachs-Ericsson, N. J., Sheffler, J. L., Stanley, I. H., Piazza, J. R., & Preacher, K. J. (2017). When emotional pain becomes physical: Adverse childhood experiences, pain, and the role of

mood and anxiety disorders. *Journal of Clinical Psychology*, *73*(10), 1403–1428. doi:10.1002/jclp.22444

Schalinski, I., Teicher, M. H., Nischk, D., Hinderer, E., Müller, O., & Rockstroh, B. (2016). Type and timing of adverse childhood experiences differentially affect severity of PTSD, dissociative and depressive symptoms in adult inpatients. *BMC Psychiatry*, *16*(1), 1–15. doi:10.1186/s12888-016-1004-5

Schickedanz, A., Halfon, N., Sastry, N., & Chung, P. J. (2018). Parents' adverse childhood experiences and their children's behavioral health problems. *Pediatrics*, *142*(2), e20180023. doi:10.1542/peds.2018-0023

Schoemann, A. M., Boulton, A. J., & Short, S. D. (2017). Determining power and sample size for simple and complex mediation models. *Social Psychological and Personality Science*, *8*(4), 379–386. doi:10.1177/1948550617715068

Schofield, T. J., Lee, R. D., & Merrick, M. T. (2013). Safe, stable, nurturing relationships as a moderator of intergenerational continuity of child maltreatment: A meta-analysis. *Journal of Adolescent Health*, *53*, S32–S38. doi:10.1016/j.jadohealth.2013.05.004

Scott, K. M., Von Korff, M., Angermeyer, M. C., Benjet, C., Bruffaerts, R., De Girolamo, G., … Kessler, R. C. (2011). Association of childhood adversities and early-onset mental disorders with adult-onset chronic physical conditions. *Archives of General Psychiatry*, *68*(8), 838–844. doi:10.1001/archgenpsychiatry.2011.77

Scully, C., McLaughlin, J., & Fitzgerald, A. (2020). The relationship between adverse childhood experiences, family functioning, and mental health problems among children and adolescents: A systematic review. *Journal of Family Therapy*, *42*(2), 291–316. doi:10.1111/1467-6427.12263

Shelby, G. D., Shirkey, K. C., Sherman, A. L., Beck, J. E., Haman, K., Shears, A. R., … Walker, L. S. (2013). Functional abdominal pain in childhood and long-term vulnerability to anxiety disorders. *Pediatrics*, *132*(3), 475–482. doi:10.1542/peds.2012-2191

Shonkoff, J. P., & Garner, A. S. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, *129*, e232. doi:10.1542/peds.2011-2663

Sieberg, C. B., Williams, S., & Simons, L. E. (2011). Do parent protective responses mediate the relation between parent distress and child functional disability among children with chronic pain? *Journal of Pediatric Psychology*, *36*(9), 1043–1051. doi:10.1093/jpepsy/jsr043

Ståhl, M. K., El-Metwally, A. A., Mikkelsson, M. K., Salminen, J. J., Pulkkinen, L. R., Rose, R. J., & Kaprio, J. A. (2013). Genetic and environmental influences on non-specific neck pain in early adolescence: A classical twin study. *European Journal of Pain (United Kingdom)*, *17*(6), 791–798. doi:10.1002/j.1532-2149.2012.00247.x

Stanford, E. A., Chambers, C. T., Biesanz, J. C., & Chen, E. (2008). The frequency, trajectories and predictors of adolescent recurrent pain: A population-based approach. *Pain*, *138*(1), 11–21. doi:10.1016/j.pain.2007.10.032

Stone, A. L., Bruehl, S., Smith, C. A., Garber, J., & Walker, L. S. (2018). Social learning pathways in the relation between parental chronic pain and daily pain severity and functional impairment in adolescents with functional abdominal pain. *Pain*, *159*(2), 298–305. doi:10.1097/j.pain.0000000000001085

Stone, A. L., & Wilson, A. C. (2016). Transmission of risk from parents with chronic pain to offspring: An integrative conceptual model. *Pain*, *157*(12), 2628–2639. doi:10.1097/j.pain.0000000000000637

Straus, M. A. (1979). Measuring intrafamily conflict and violence: The Conflict Tactics (CT)

Scales. *Journal of Marriage and Family*, *41*(1), 75–88. doi:10.4324/9781315126401

Substance Abuse and Mental Health Services Administration. (2014). *SAMHSA's concept of trauma and guidance for a trauma-informed approach*. https://ncsacw.samhsa.gov/userfiles/files/SAMHSA_Trauma.pdf

Svensson, D. A., Larsson, B., Bille, B., & Lichtenstein, P. (1999). Genetic and environmental influences on recurrent headaches in eight to nine-year-old twins. *Cephalalgia*, *19*(10), 866–872. doi:10.1046/j.1468-2982.1999.1910866.x

Thomas-Argyriou, J. C., Letourneau, N., Dewey, D., Campbell, T. S., Giesbrecht, G. F., & the APrON Study Team. (2020). The role of HPA-axis function during pregnancy in the intergenerational transmission of maternal adverse childhood experiences to child behavior problems. *Development and Psychopathology*, 1–17. doi:10.1017/S0954579419001767

Thomas, J. C., Letourneau, N., Campbell, T. S., & Giesbrecht, G. F. (2018). Social buffering of the maternal and infant HPA axes: Mediation and moderation in the intergenerational transmission of adverse childhood experiences. *Development and Psychopathology*, *30*(3), 921–939. doi:10.1017/S0954579418000512

Treat, A. E., Sheffield-Morris, A., Williamson, A. C., & Hays-Grudo, J. (2019). Adverse childhood experiences and young children's social and emotional development: The role of maternal depression, self-efficacy, and social support. *Early Child Development and Care*, 1–15. doi:10.1080/03004430.2019.1578220

Treede, R.-D., Rief, W., Barke, A., Aziz, Q., Bennett, M. I., Benoliel, R., … Wang, S.-J. (2015). A classification of chronic pain for ICD-11. *Pain*, *156*(6), 1003–1007. doi:10.1097/j.pain.0000000000000160

Treede, R.-D., Rief, W., Barke, A., Aziz, Q., Bennett, M. I., Benoliel, R., … Wang, S.-J. (2019).

Chronic pain as a symptom or a disease. *Pain*, *160*(1), 19–27. doi:10.1097/j.pain.0000000000001384

Valeri, L., & VanderWeele, T. J. (2013). Mediation analysis allowing for exposure-mediator interactions and causal interpretation: Theoretical assumptions and implementation with SAS and SPSS macros. *Psychological Methods*, *18*(2), 137–150. doi:10.1037/a0031034

van Tilburg, M. A. L., Levy, R. L., Walker, L. S., Von Korff, M., Feld, L. D., Garner, M., … Whitehead, W. E. (2015). Psychosocial mechanisms for the transmission of somatic symptoms from parents to children. *World Journal of Gastroenterology*, *21*(18), 5532–5541. doi:10.3748/wjg.v21.i18.5532

Vinall, J., Pavlova, M., Asmundson, G., Rasic, N., & Noel, M. (2016). Mental health comorbidities in pediatric chronic pain: A narrative review of epidemiology, models, neurobiological mechanisms and treatment. *Children*, *3*(4), 40. doi:10.3390/children3040040

von Baeyer, C. L., Spagrud, L. J., McCormick, J. C., Choo, E., Neville, K., & Connelly, M. A. (2009). Three new datasets supporting use of the Numerical Rating Scale (NRS-11) for children's self-reports of pain intensity. *Pain*, *143*(3), 223–227. doi:10.1016/j.pain.2009.03.002

Walker, L. S., Sherman, A. L., Bruehl, S., Garber, J., & Smith, C. A. (2012). Functional abdominal pain patient subtypes in childhood predict functional gastrointestinal disorders with chronic pain and psychiatric comorbidities in adolescence and adulthood. *Pain*, *153*(9), 1798–1806. doi:10.1016/j.pain.2012.03.026

Walker, L. S., & Zeman, J. L. (1992). Parental response to child illness behavior. *Journal of Pediatric Psychology*, *17*(1), 49–71. doi:10.1093/jpepsy/17.1.49

Weathers, F. W., Litz, B. T., Keane, T. M., Palmieri, P. A., Marx, B. P., & Schnurr, P. P. (2013). *The PTSD Checklist for DSM-5 with Life Events Checklist for DSM-5 and Criterion A*. https://www.ptsd.va.gov/professional/assessment/documents/PCL5_LEC_criterionA.PDF

Wegman, H. L., & Stetler, C. (2009). A meta-analytic review of the effects of childhood abuse on medical outcomes in adulthood. *Psychosomatic Medicine*, *71*(8), 805–812. doi:10.1097/PSY.0b013e3181bb2b46

Widom, C. S., Raphael, K. G., & DuMont, K. A. (2004). The case for prospective longitudinal studies in child maltreatment research: Commentary on Dube, Williamson, Thompson, Felitti, and Anda (2004). *Child Abuse and Neglect*, *28*(7), 715–722. doi:10.1016/j.chiabu.2004.03.009

Wu, N. S., Schairer, L. C., Dellor, E., & Grella, C. (2010). Childhood trauma and health outcomes in adults with comorbid substance abuse and mental health disorders. *Addictive Behavior*, *35*(1), 68–71. doi:10.1016/j.addbeh.2009.09.003

Wyatt, G. E. (1985). The sexual abuse of Afro-American and White-American women in childhood. *Child Abuse and Neglect*, *9*(4), 507–519. doi:10.1016/0145-2134(85)90060-2

Appendix A: Tables and Figures

Table 1

Sociodemographic Characteristics of the Total Sample

Variable	*n*	*M* (*SD*) or %
Parent age, years	-	44.91 (5.14)
Parent gender		
Female	180	92.3
Male	14	7.2
Other	1	0.5
Parent race/ethnicity		
White/Caucasian	168	86.2
Biracial/multiracial	12	6.2
Latin American	4	2.1
Arab/West Asian	3	1.5
South Asian	2	1.0
Aboriginal	1	0.5
Black	1	0.5
Chinese	1	0.5
Filipino	1	0.5
Other	1	0.5
Did not answer	1	0.5
Parent marital status		
Married/common-law	158	81.0

Divorced or separated	27	13.8
Single	7	3.6
Widowed	3	1.5
Parent education		
High school or less	18	9.2
Vocational school or some college (no degree)	41	21.0
College or Bachelor's degree	109	55.9
Graduate/Professional school (Master's, PhD)	27	13.8
Parent employment status		
Full-time	108	55.4
Part-time	50	25.6
Not working	35	17.9
Did not answer	2	1.0
Annual household income, CAD		
$0 - $29,999	11	5.6
$30,000 - $59,999	18	9.2
$60,000 - $89,999	23	11.8
>$90,000	118	60.5
Did not answer	25	12.8
Youth age, years	-	14.39 (2.20)
Youth gender		
Female	148	75.9
Male	44	22.6

Other	3	1.5
Youth race/ethnicity		
White/Caucasian	159	81.5
Biracial/multiracial	16	8.2
Arab/West Asian	3	1.5
South Asian	3	1.5
Aboriginal	2	1.0
Black	2	1.0
Latin American	2	1.0
Filipino	1	0.5
Other	6	3.1
Did not answer	1	0.5

Note. *M* = mean, *SD* = standard deviation.

Table 2

Sociodemographic Characteristics of the Sample by Site

	ACH (*n* = 168)		IWK (*n* = 24)		SickKids (*n* = 3)	
Variable	*n*	*M* (*SD*) or %	*n*	*M* (*SD*) or %	*n*	*M* (*SD*) or %
Parent age, years	-	44.94 (5.14)	-	44.54 (5.32)	-	45.67 (5.03)
Parent gender						
Female	153	91.1	24	100	3	100
Male	14	8.3	0	0.0	0	0.0
Other	1	0.6	0	0.0	0	0.0
Parent race/ethnicity						
White/Caucasian	142	84.5	23	95.8	3	100
Biracial/multiracial	11	6.5	1	4.2	0	0.0
Latin American	4	2.4	0	0.0	0	0.0
Arab/West Asian	3	1.8	0	0.0	0	0.0
South Asian	2	1.2	0	0.0	0	0.0
Aboriginal	1	0.6	0	0.0	0	0.0
Black	1	0.6	0	0.0	0	0.0
Chinese	1	0.6	0	0.0	0	0.0
Filipino	1	0.6	0	0.0	0	0.0
Other	1	0.6	0	0.0	0	0.0
Did not answer	1	0.6	0	0.0	0	0.0
Parent marital status						
Married/common-law	135	80.4	20	83.3	3	100

Divorced or separated	24	14.3	3	12.5	0	0.0
Single	6	3.6	1	4.2	0	0.0
Widowed	3	1.8	0	0.0	0	0.0
Parent education						
High school or less	17	10.1	1	4.2	0	0.0
Vocational school or some college (no degree)	38	22.6	3	12.5	0	0.0
College or Bachelor's degree	93	55.4	13	54.2	3	100
Graduate/Professional school (Master's, PhD)	20	11.9	7	29.2	0	0.0
Parent employment status						
Full-time	88	52.4	17	70.8	3	100
Part-time	49	29.2	1	4.2	0	0.0
Not working	31	18.5	4	16.7	0	0.0
Did not answer	0	0.0	2	8.3	0	0.0
Annual household income, CAD						
$0 - $29,999	9	5.4	2	8.3	0	0.0
$30,000 - $59,999	15	8.9	3	12.5	0	0.0
$60,000 - $89,999	20	11.9	3	12.5	0	0.0
>$90,000	101	60.1	14	58.3	3	100
Did not answer	23	13.7	2	8.3	0	0.0

Youth age, years	-	14.30 (2.18)	-	14.92 (2.26)	-	15.17 (2.84)
Youth gender						
Female	122	72.6	23	95.8	3	100
Male	43	25.6	1	4.2	0	0.0
Other	3	1.8	0	0.0	0	0.0
Youth race/ethnicity						
White/Caucasian	136	81.0	21	87.5	2	66.7
Biracial/multiracial	13	7.7	2	8.3	1	33.3
Arab/West Asian	3	1.8	0	0.0	0	0.0
South Asian	3	1.8	0	0.0	0	0.0
Aboriginal	2	1.2	0	0.0	0	0.0
Latin American	2	1.2	0	0.0	0	0.0
Black	1	0.6	1	4.2	0	0.0
Filipino	1	0.6	0	0.0	0	0.0
Other	6	3.6	0	0.0	0	0.0
Did not answer	1	0.6	0	0.0	0	0.0

Note. *M* = mean, *SD* = standard deviation.

Table 3

Youth Pain Characteristics at Baseline

Variable	*n*	*M* (*SD*) or %
Pain duration, years	-	3.40 (3.14)
Pain locations		
Head	131	67.2
Muscle and joints	51	26.2
Stomach	38	19.5
Legs	36	18.5
Chest	22	11.3
Other	56	28.7
Two or more locations	87	44.6
Pain frequency		
Not at all	4	2.1
1 time per week	14	7.2
2 to 3 times per week	53	27.2
4 to 6 times per week	26	13.3
Daily	97	49.7
Pain intensity, out of 10	-	5.52 (1.82)
Pain interference, out of 74	-	55.85 (9.32)

Note. *M* = mean, *SD* = standard deviation.

Table 4

Means, Standard Deviations, and Correlation Coefficients for Key Study Variables

Variable	2	3	4	5	6	7	*M* (*SD*)	*n*
1. Parent total ACE score	.89***	.89***	.19**	.20**	-.05	.12	2.08 (2.34)	195
2. Parent maltreatment score	-	.59***	.19**	.19**	-.09	.09	0.89 (1.32)	195
3. Parent household dysfunction score		-	.15*	.17*	.01	.13	1.19 (1.31)	195
4. Parent chronic pain status			-	.26***	-.06	.05	-	195
5. Parent PTSD symptoms				-	.08	.21*	10.69 (12.39)	191
6. Youth pain intensity					-	.47***	5.26 (2.09)	145
7. Youth pain interference						-	52.84 (9.64)	141

Note. ACE = Adverse childhood experience, *M* = mean, *SD* = standard deviation.
*$p < .05$, **$p < .01$, ***$p < .001$.

Table 5

Prevalence Rates of ACE Scores in Parents of Youth with Chronic Pain

Variable	*n*	%
Total ACE score		
0	63	32.3
1	40	20.5
2	31	15.9
3	18	9.2
4	12	6.2
5	11	5.6
6	8	4.1
7	4	2.1
8	3	1.5
9	3	1.5
10	2	1.0
Total maltreatment score		
0	108	55.4
1	47	24.1
2	14	7.2
3	10	5.1
4	11	5.6
5	5	2.6
Total household dysfunction score		

0	80	41.0
1	48	24.6
2	34	17.4
3	21	10.8
4	7	3.6
5	5	2.6

Note. ACE = Adverse childhood experience.

Table 6

Prevalence Rates of Individual ACEs in Parents of Youth with Chronic Pain

ACE category	*n*	%
Maltreatment categories		
Emotional abuse	47	24.1
Physical abuse	13	6.7
Sexual abuse	54	27.7
Emotional neglect	32	16.4
Physical neglect	28	14.4
Household dysfunction categories		
Problematic substance use in household	61	31.3
Mental illness in household	73	37.4
Physical violence between parents	24	12.3
Household member in prison	9	4.6
Parents separated/divorced	65	33.3

Note. ACE = Adverse childhood experience.

Table 7

Results from Logistic Regression of Parent Total ACE Score Predicting Parent Chronic Pain Status

Model	Predictor variable	*b*	*SE-b*	Wald	*p*	OR	95% CI
1a	(Intercept)	-0.30	0.20	2.41	.121	0.74	-
	Parent total ACEs	0.18	0.07	6.83	.009	1.19	1.05, 1.36
1b	Block 1						
	(Intercept)	-0.20	1.51	0.02	.896	0.82	-
	Parent age	-0.001	0.03	0.001	.972	1.00	0.94, 1.07
	Parent race/ethnicity	0.38	0.47	0.63	.426	1.46	0.58, 3.69
	Parent education	-0.41	0.40	1.03	.310	0.67	0.30, 1.46
	Parent income	1.40	0.41	12.02	.001	4.07	1.84, 8.99
	Block 2						
	(Intercept)	-1.39	1.62	0.74	.391	0.25	-
	Parent age	0.02	0.04	0.29	.594	1.02	0.95, 1.09
	Parent race/ethnicity	0.28	0.48	0.33	.564	1.32	0.51, 3.42
	Parent education	-0.61	0.42	2.07	.150	0.55	0.24, 1.25
	Parent income	1.26	0.42	9.20	.002	3.52	1.56, 7.95
	Parent total ACEs	0.21	0.09	6.17	.013	1.24	1.05, 1.46

Note. df = 1; Model 1a: Nagelkerke R^2 = .05; Model 1b, Block 1: Nagelkerke R^2 = .12, Model 1b, Block 2: Nagelkerke R^2 = .16; The target category for the dependent variable was presence of chronic pain and the reference category was absence of chronic pain; focus groups of covariates are listed in the results section of the thesis document. ACE = Adverse childhood experience, *SE* = standard error, OR = odds ratio, CI = confidence interval.

Table 8

Results from Logistic Regression of Parent Maltreatment Score Predicting Parent Chronic Pain Status

Model	Predictor variable	*b*	*SE-b*	Wald	*p*	OR	95% CI
2a	(Intercept)	-0.22	0.18	1.55	.213	0.80	-
	Parent maltreatment	0.31	0.12	6.82	.009	1.37	1.08, 1.73
2b	Block 1						
	(Intercept)	-0.20	1.51	0.02	.896	0.82	-
	Parent age	-0.001	0.03	0.001	.972	1.00	0.94, 1.07
	Parent race/ethnicity	0.38	0.47	0.63	.426	1.46	0.58, 3.69
	Parent education	-0.41	0.40	1.03	.310	0.67	0.30, 1.46
	Parent income	1.40	0.41	12.02	.001	4.07	1.84, 8.99
	Block 2						
	(Intercept)	-0.85	1.57	0.29	.588	0.43	-
	Parent age	0.01	0.03	0.08	.781	1.01	0.95, 1.08
	Parent race/ethnicity	0.22	0.49	0.20	.652	1.25	0.48, 3.27
	Parent education	-0.55	0.42	1.74	.187	0.58	0.26, 1.31
	Parent income	1.27	0.41	9.39	.002	3.55	1.58, 7.98
	Parent maltreatment	0.34	0.15	5.17	.023	1.40	1.05, 1.88

Note. df = 1; Model 2a: Nagelkerke R^2 = .05; Model 2b, Block 1: Nagelkerke R^2 = .12, Model 2b, Block 2: Nagelkerke R^2 = .16; The target category for the dependent variable was presence of chronic pain and the reference category was absence of chronic pain; focus groups of covariates are listed in the results section of the thesis document. *SE* = standard error, OR = odds ratio, CI = confidence interval.

Table 9

Results from Logistic Regression of Parent Household Dysfunction Score Predicting Parent Chronic Pain Status

Model	Predictor variable	*b*	*SE-b*	Wald	*p*	OR	95% CI
3a	(Intercept)	-0.22	0.20	1.32	.251	0.80	-
	Parent household dysfunction	0.24	0.11	4.23	.040	1.27	1.01, 1.58
3b	Block 1						
	(Intercept)	-0.20	1.51	0.02	.896	0.82	-
	Parent age	-0.001	0.03	0.001	.972	1.00	0.94, 1.07
	Parent race/ethnicity	0.38	0.47	0.63	.426	1.46	0.58, 3.69
	Parent education	-0.41	0.40	1.03	.310	0.67	0.30, 1.46
	Parent income	1.40	0.41	12.02	.001	4.07	1.84, 8.99
	Block 2						
	(Intercept)	-1.29	1.62	0.63	.426	0.28	-
	Parent age	0.02	0.03	0.24	.623	1.02	0.95, 1.09
	Parent race/ethnicity	0.39	0.48	0.66	.417	1.47	0.58, 3.76
	Parent education	-0.57	0.42	1.84	.176	0.57	0.25, 1.29
	Parent income	1.32	0.41	10.20	.001	3.73	1.66, 8.36
	Parent household dysfunction	0.29	0.14	4.27	.039	1.34	1.02, 1.77

Note. df = 1; Model 3a: Nagelkerke R^2 = .03; Model 3b, Block 1: Nagelkerke R^2 = .12, Model 3b, Block 2: Nagelkerke R^2 = .15; The target category for the dependent variable was presence of chronic pain and the reference category was absence of chronic pain; focus groups of covariates are listed in the results section of the thesis document. *SE* = standard error, OR = odds ratio, CI = confidence interval.

Table 10

Results from Linear Regression of Parent Total ACE Score Predicting Parent PTSD Symptoms

Model	Predictor variable	*b*	95% CI	*SE-b*	*Beta*	*p*	*r*	sr^2
1a	(Constant)	8.44	6.11, 10.77	1.18	-	-	-	-
	Parent total ACEs	1.07	0.33, 1.80	0.37	.20	.005	.20	.04
1b	Step 1							
	(Constant)	-1.61	-17.90, 14.69	8.25	-	-	-	-
	Parent age	0.24	-0.11, 0.60	0.18	.11	.180	.11	.01
	Parent race/ethnicity	-0.68	-5.56, 4.21	2.47	-.02	.785	.02	<.01
	Parent education	-2.68	-6.81, 1.44	2.09	-.11	.201	-.02	.01
	Parent income	7.66	3.57, 11.74	2.07	.31	<.001	.26	.08
	Step 2							
	(Constant)	-4.95	-21.86, 11.97	8.56	-	-	-	-
	Parent age	0.30	-0.07, 0.67	0.19	.13	.107	.11	.02
	Parent race/ethnicity	-1.01	-5.90, 3.89	2.48	-.03	.685	.02	<.01
	Parent education	-3.19	-7.36, 0.99	2.11	-.13	.133	-.02	.01
	Parent income	7.00	2.82, 11.18	2.11	.28	.001	.26	.06
	Parent total ACEs	0.58	-0.24, 1.39	0.41	.12	.163	.13	.01

Note. Model 1a R^2 = .04, adjusted R^2 = .04; Model 1b, Step 1 R^2 = .09, adjusted R^2 = .07; Model 1b, Step 2 R^2 = .10, adjusted R^2 = .07. ACE = Adverse childhood experience, *SE* = standard error, CI = confidence interval, *r* = zero-order correlation, sr^2 = squared semi-partial correlation.

Table 11

Results from Linear Regression of Parent Maltreatment Score Predicting Parent PTSD Symptoms

Model	Predictor variable	*b*	95% CI	*SE-b*	*Beta*	*p*	*r*	sr^2
2a	(Constant)	9.10	6.99, 11.21	1.07	-	-	-	-
	Parent maltreatment	1.75	0.44, 3.07	0.67	.19	.009	.19	.04
2a*	(Constant)	9.41	7.37, 11.45	1.04	-	-	-	-
	Parent maltreatment	1.10	-0.35, 2.55	0.74	.11	.136	.11	.01
2b	Step 1							
	(Constant)	-1.61	-17.90, 14.69	8.25	-	-	-	-
	Parent age	0.24	-0.11, 0.60	0.18	.11	.180	.11	.01
	Parent race/ethnicity	-0.68	-5.56, 4.21	2.47	-.02	.785	.02	<.01
	Parent education	-2.68	-6.81, 1.44	2.09	-.11	.201	-.02	.01
	Parent income	7.66	3.57, 11.74	2.07	.31	<.001	.26	.08
	Step 2							
	(Constant)	-3.11	-19.62, 13.41	8.36	-	-	-	-
	Parent age	0.27	-0.09, 0.63	0.18	.12	.143	.11	.01
	Parent race/ethnicity	-1.12	-6.07, 3.83	2.51	-.04	.655	.02	<.01
	Parent education	-2.99	-7.15, 1.17	2.11	-.12	.158	-.02	.01
	Parent income	7.16	2.98, 11.34	2.12	.29	.001	.26	.07
	Parent maltreatment	0.77	-0.64, 2.18	0.72	.09	.284	.12	.01

Note. *Influential cases removed from model; Model 2a R^2 = .04, adjusted R^2 = .03; Model 2a* R^2 = .01, adjusted R^2 = .01; Model 2b, Step 1 R^2 = .09, adjusted R^2 = .07; Model 2b, Step 2 R^2 = .10, adjusted R^2 = .07. *SE* = standard error, CI = confidence interval, *r* = zero-order correlation, sr^2 = squared semi-partial correlation.

Table 12

Results from Linear Regression of Parent Household Dysfunction Score Predicting Parent PTSD Symptoms

Model	Predictor variable	*b*	95% CI	*SE-b*	*Beta*	*p*	*r*	sr^2
3a	(Constant)	8.74	6.37, 11.10	1.20	-	-	-	-
	Parent h. dysfunction	1.62	0.29, 2.95	0.67	.17	.017	.17	.03
3b	Step 1							
	(Constant)	-1.61	-17.90, 14.69	8.25	-	-	-	-
	Parent age	0.24	-0.11, 0.60	0.18	.11	.180	.11	.01
	Parent race/ethnicity	-0.68	-5.56, 4.21	2.47	-.02	.785	.02	<.01
	Parent education	-2.68	-6.81, 1.44	2.09	-.11	.201	-.02	.01
	Parent income	7.66	3.57, 11.74	2.07	.31	<.001	.26	.08
	Step 2							
	(Constant)	-5.38	-22.52, 11.76	8.68	-	-	-	-
	Parent age	0.31	-0.06, 0.68	0.19	.13	.101	.11	.02
	Parent race/ethnicity	-0.67	-5.54, 4.20	2.47	-.02	.787	.02	<.01
	Parent education	-3.15	-7.32, 1.02	2.11	-.13	.137	-.02	.01
	Parent income	7.17	3.04, 11.31	2.09	.29	.001	.26	.07
	Parent h. dysfunction	0.98	-0.44, 2.40	0.72	.11	.174	.11	.01

Note. Model 3a R^2 = .03, adjusted R^2 = .03; Model 3b, Step 1 R^2 = .09, adjusted R^2 = .07; Model 3b, Step 2 R^2 = .10, adjusted R^2 = .07; h. dysfunction = household dysfunction, *SE* = standard error, CI = confidence interval, *r* = zero-order correlation, sr^2 = squared semi-partial correlation.

Table 13

Traumatic Events Reported by Parents of Youth with Chronic Pain

Traumatic event	*n*	%
Traditional ACE category	13	6.7
Sexual abuse	9	4.6
Physical abuse	2	1.0
Physical violence between parents	1	0.5
Parent separation/divorce	1	0.5
Other childhood event	16	8.2
Death of family or friend	6	3.1
Witnessed fatal accident	2	1.0
Parent physical illness/medical emergency	2	1.0
Bullied or socially excluded	2	1.0
Involved in serious accident	1	0.5
Physical violence against sibling	1	0.5
Natural disaster	1	0.5
Fire	1	0.5
Adulthood event	148	75.9
No event reported	18	9.2

Note. ACE = Adverse childhood experience.

Table 14

Results from Mediation Analyses of Parent ACEs Predicting Youth Pain Interference through Parent PTSD Symptoms

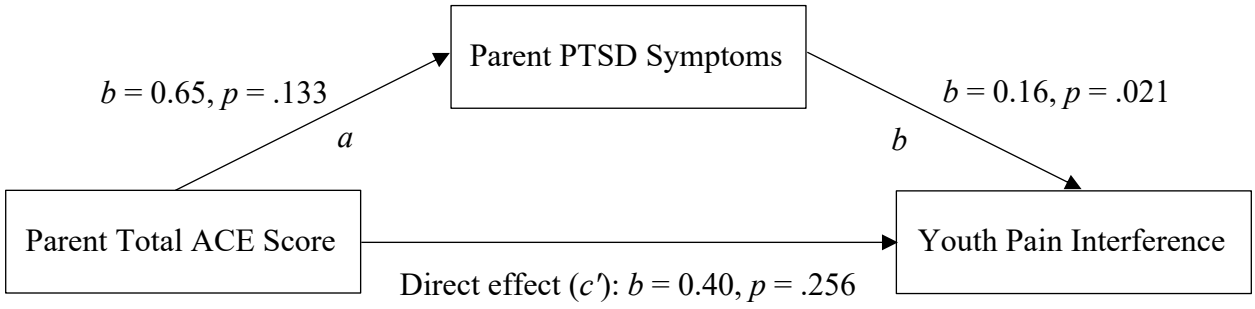
Model	*n*	Path	*b*	95% CI	*SE-b*	*Beta*	*p*
1a	140	Total ACEs → PTSD symptoms (*a*)	0.65	-0.20, 1.50	0.43	.13	.133
		PTSD symptoms → Pain interference (*b*)	0.16	0.02, 0.30	0.07	.20	.021
		Total ACEs → Pain interference (*c′*)	0.40	-0.30, 1.10	0.35	.10	.256
		Total ACEs → PTSD symptoms → Pain interference (*ab*)	0.11	-0.02, 0.30	0.08	.02	-
1b	114	Total ACEs → PTSD symptoms (*a*)	0.73	-0.26, 1.72	0.50	.15	.149
		PTSD symptoms → Pain interference (*b*)	0.10	-0.06, 0.25	0.08	.12	.213
		Total ACEs → Pain interference (*c′*)	0.28	-0.53, 1.09	0.41	.07	.496
		Total ACEs → PTSD symptoms → Pain interference (*ab*)	0.07	-0.03, 0.28	0.08	.02	-
2a	140	Maltreatment → PTSD symptoms (*a*)	0.92	-0.57, 2.40	0.75	.10	.225
		PTSD symptoms → Pain interference (*b*)	0.17	0.03, 0.30	0.07	.20	.018
		Maltreatment → Pain interference (*c′*)	0.49	-0.73, 1.71	0.62	.07	.426

		Maltreatment → PTSD symptoms → Pain interference (*ab*)	0.15	-0.08, 0.48	0.14	.02	-
2b	114	Maltreatment → PTSD symptoms (*a*)	0.92	-0.85, 2.69	0.89	.11	.304
		PTSD symptoms → Pain interference (*b*)	0.11	-0.05, 0.26	0.08	.13	.181
		Maltreatment → Pain interference (*c′*)	0.04	-1.39, 1.47	0.72	.01	.957
		Maltreatment → PTSD symptoms → Pain interference (*ab*)	0.10	-0.08, 0.37	0.11	.01	-
3a	140	Household dysfunction → PTSD symptoms (*a*)	1.11	-0.41, 2.63	0.77	.12	.150
		PTSD symptoms → Pain interference (*b*)	0.16	0.02, 0.30	0.07	.20	.021
		Household dysfunction → Pain interference (*c′*)	0.77	-0.48, 2.01	0.63	.10	.226
		Household dysfunction → PTSD symptoms → Pain int. (*ab*)	0.18	-0.04, 0.53	0.15	.02	-
3b	114	Household dysfunction → PTSD symptoms (*a*)	1.30	-0.42, 3.02	0.87	.15	.136
		PTSD symptoms → Pain interference (*b*)	0.09	-0.06, 0.25	0.08	.11	.237
		Household dysfunction → Pain interference (*c′*)	0.80	-0.60, 2.19	0.70	.11	.260
		Household dysfunction → PTSD symptoms → Pain int. (*ab*)	0.12	-0.05, 0.48	0.14	.02	-

Note. 'a' models = unadjusted models, 'b' models = adjusted models; 95% CI for indirect effect (path ab) based on 5000 bootstrap samples.

Figure 1

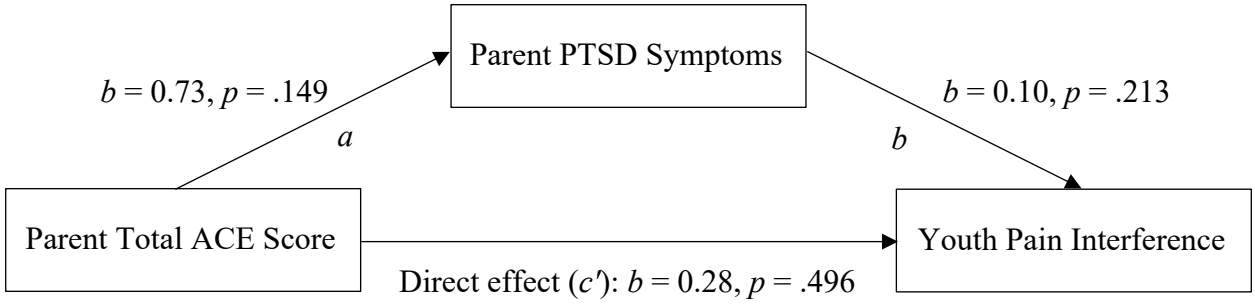
Direct and Indirect Effects of the Unadjusted Mediation Model for Parent Total ACE Score Predicting Youth Pain Interference, Mediated by Parent PTSD Symptoms (Model 1a).



Indirect effect (*ab*): *b* = 0.11, 95% CI [-0.02, 0.30]

Figure 2

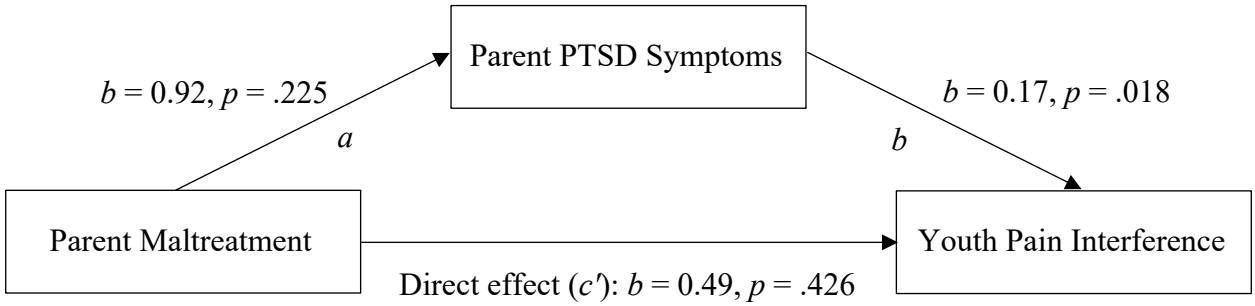
Direct and Indirect Effects of the Adjusted Mediation Model for Parent Total ACE Score Predicting Youth Pain Interference, Mediated by Parent PTSD Symptoms (Model 1b).



Indirect effect (*ab*): *b* = 0.07, 95% CI [-0.03, 0.28]

Figure 3

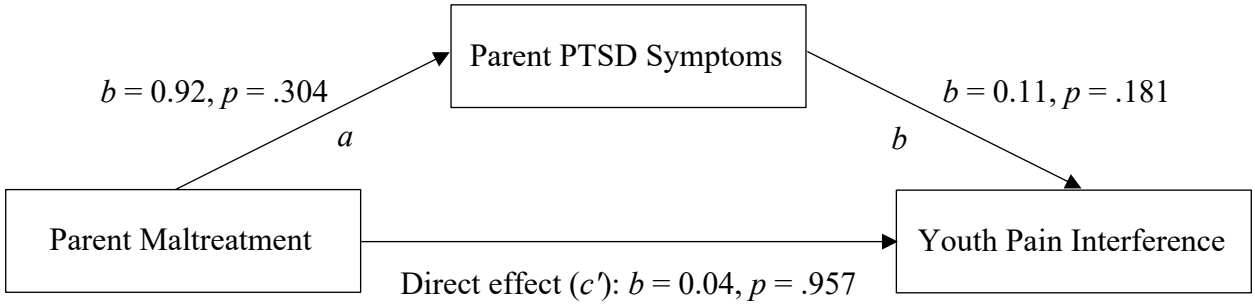
Direct and Indirect Effects of the Unadjusted Mediation Model for Parent Maltreatment Score Predicting Youth Pain Interference, Mediated by Parent PTSD Symptoms (Model 2a).



Indirect effect (*ab*): *b* = 0.15, 95% CI [-0.08, 0.48]

Figure 4

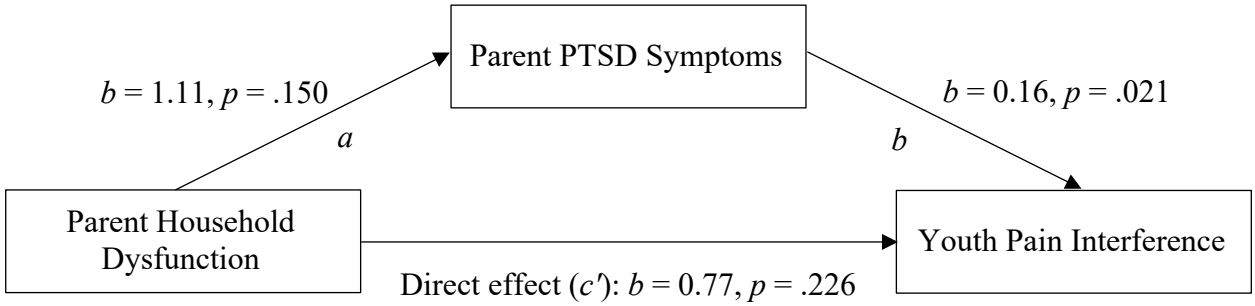
Direct and Indirect Effects of the Adjusted Mediation Model for Parent Maltreatment Score Predicting Youth Pain Interference, Mediated by Parent PTSD Symptoms (Model 2b).



Indirect effect (*ab*): *b* = 0.10, 95% CI [-0.08, 0.37]

Figure 5

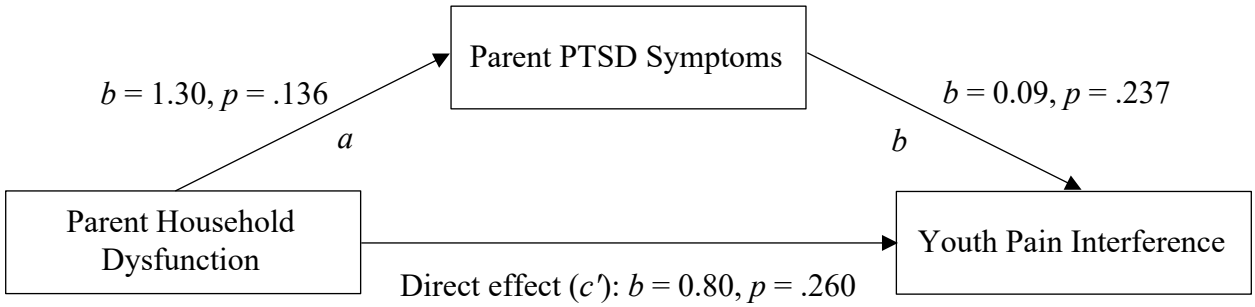
Direct and Indirect Effects of the Unadjusted Mediation Model for Parent Household Dysfunction Score Predicting Youth Pain Interference, Mediated by Parent PTSD Symptoms (Model 3a).



Indirect effect (*ab*): *b* = 0.18, 95% CI [-0.04, 0.53]

Figure 6

Direct and Indirect Effects of the Adjusted Mediation Model for Parent Household Dysfunction Score Predicting Youth Pain Interference, Mediated by Parent PTSD Symptoms (Model 3b).



Indirect effect (*ab*): *b* = 0.12, 95% CI [-0.05, 0.48]

Appendix B: Letter of Permission

September, 2020

Letter of Permission

I hereby provide permission to use the following co-authored work as part of Jaimie Beveridge's Master's thesis:

Beveridge, J.K., Dobson, K.S., Madigan, S., Yeates, K.O., Stone, A.L., Wilson, A.C., Salberg, S., Mychasiuk, R., & Noel, M. Adverse childhood experiences in parents of youth with chronic pain: Prevalence and comparison to a community-based sample.

I am of the understanding that the purpose of this request is to include the material in Jaimie Beveridge's thesis, and that the thesis will be added to the institutional repository at the University of Calgary and the Library and Archives Canada.

University of Calgary Theses Repository – The Vault: http://theses.ucalgary.ca/Library

Archives Canada: http://collectionscanada.gc.ca/obj/s4/f2/frm-nl59-2-e.pdf

Dr. Keith S. Dobson	Dr. Sheri Madigan	Dr. Keith O. Yeates
Dr. Amanda L. Stone	Dr. Anna C. Wilson	Ms. Sabrina Salberg
Dr. Richelle Mychasiuk	Dr. Melanie Noel	