THE RELATION BETWEEN PARENTAL CHRONIC PAIN HISTORIES AND PEDIATRIC CHRONIC ABDOMINAL PAIN OUTCOMES

By

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CHAPTER I

INTRODUCTION

Chronic abdominal pain (CAP) is a common childhood chronic pain complaint and frequently results in pain-related disability, school absenteeism, and increased health care utilization (Campo, Comer, Jansen-McWilliams, Gardner, & Kelleher, 2002; Hoftun, Romundstad, Zwart, & Rygg, 2011; Saps et al., 2009). Children with CAP are at greater risk for developing functional gastrointestinal disorders (FGIDs), including irritable bowel syndrome (IBS) and functional dyspepsia, as well as other forms of chronic pain in adulthood (Walker, Dengler-Crish, Rippel, & Bruehl, 2010; Walker, Guite, Duke, Barnard, & Greene, 1998). Parental history of FGIDs or chronic pain may affect the development or pain experience of children with CAP. Prior research has found a higher prevalence of FGIDs and chronic pain in parents of children with CAP than controls (Buonavolontà et al., 2010; Campo et al., 2007; Saito et al., 2008). Additionally, Levy et al (2004) found that children of mothers with IBS had more frequent stomachaches, non-GI symptoms, school absences, and physician visits than children of mothers without IBS.

Biological, contextual, and psychological factors could all contribute to this parent-child pain association. The fact that FGIDs tend to cluster in families could implicate either a genetic or an environmental basis for this association. Twin studies have estimated the genetic heritability of FGIDs to be between 22% and 57% (Saito, Mitra, & Mayer, 2010). One genetic factor that could contribute to the heritability of CAP could be a biological vulnerability for greater pain sensitivity. Researchers have found that a catechol-O-methyltransferase (COMT)

gene polymorphism may contribute to greater pain sensitivity in individuals with fibromyalgia, chronic headaches, and facial pain (Diatchenko et al., 2005; Gürsoy et al., 2003; Hagen, Pettersen, Stovner, Skorpen, & Zwart, 2006). Individuals with FGIDs are a heterogeneous group and there are likely many different genes that could contribute to the development of symptoms and pain experience (Saito, et al., 2010).

While genetics may contribute to this parent-child pain association, social learning may play as great a role as genetics in influencing child pain experience. One twin study found that having a mother with IBS or a father with IBS, independently, were stronger predictors of IBS than having a dizygotic (DZ) twin with IBS (Levy et al., 2001). Because DZ twins and their parents roughly should share the same amount of genetic material, this finding suggests that, in comparison to genetic heritability, social learning may have an equal or greater effect on the development of IBS. Social learning theory posits that behavior is developed and maintained largely through social rewards (Bandura, 1977). In the case of child pain behavior, a child could learn behaviors either through parental reinforcement of child pain behaviors and/or observational learning (Levy, 2011). Evidence from experimental pain studies supports the hypothesis that maternal modeling of pain behavior influences child pain response. For example, children whose mothers were instructed to exaggerate their own responses to the cold pressor task exhibited lower pain thresholds than children whose mothers were not given the same instructions (Goodman, 2003).

Additionally, individual psychological factors, such as the child's attention towards pain, may help explain a parent-child pain relationship. Specifically, a child's attention towards pain could bias his or her perception of parental chronic pain complaints. For example, somatic hypervigilance may help explain the relationship between self-reported family history of chronic

pain and recent pain complaints in young adult volunteers (Fillingim, Edwards, & Powell, 2000). In addition to a possible bias in perception of parental chronic pain complaints, children with an attentional bias towards pain could also be more susceptible to the social learning of pain because of their attentional bias towards threatening pain information (Goubert, Vlaeyen, Crombez, & Craig, 2011).

Some studies have shown an association between parents' and children's experiences of pain, as well as potential mechanisms that support the development of this association (Evans et al., 2008; Laurell, Larsson, & Eeg-Olofsson, 2005; Mallen, Peat, Thomas, & Croft, 2006; Schanberg, Keefe, Lefebvre, Kredich, & Gil, 1998). However, no studies have examined whether the existence of parental pain may influence the persistence of children's pain over time. Any of the biological, contextual, and psychological factors that account for an association between parent and child pain at a single point in time, could also contribute to an association between parental pain and persistence of children's pain. For example, children who inherit a genetic vulnerability for pain sensitivity could be more likely to experience persistent pain symptoms. Long-term parental modeling of pain behaviors also could continually reinforce child pain behaviors. Finally, children who attend to threatening pain information in the environment may likely maintain their pain symptoms as well as a bias in their perception of parental chronic pain.

The purpose of this study is to investigate within a young adult population with a history of CAP, the relationship between parental chronic pain history and the persistence and severity of their own symptoms over a ten year period. We hypothesize that a greater proportion of young adults with persistent CAP will report a positive parental chronic pain history than those whose pain has resolved and controls. Additionally, within the former CAP patient population,

we expect young adults with a positive parental chronic pain history to report more severe symptoms, more disability, and greater sensitivity to laboratory pain than young adults who do not report a positive parental chronic pain history. Finally, we predict that, of former CAP patients, those with a positive parental chronic pain history will have higher odds of developing other non-abdominal chronic pain sites, utilizing more health services, and reporting a greater impact of health on their ability to work.

There is some evidence of the influence of gender in the parent-child pain relationship. Chambers et al (2002) found that girls whose mothers interacted with them in a pain promoting way reported more pain than daughters of mothers in control and pain reducing groups. However, this effect was not significant for boys. Similarly, Walker et al (2006) found that the effect of parental attention on symptom complaints was greater for girl pain patients than for boy pain patients or well children. These studies suggest that maternal modeling and reinforcement may have a greater impact on girls than boys. Gender role modeling suggests that, likewise, fathers may have a greater impact on male pain behaviors. However, no studies have examined the differential impact of mother's versus father's on girl's versus boy's pain (Evans, et al., 2008). This study will investigate the influence of gender on the relationship between parental pain history and child pain experience and persistence. We hypothesize that positive maternal pain history will have a greater impact on pain experience and persistence in girls than in boys.

CHAPTER II

METHOD

Participants

Chronic abdominal pain (CAP) patients. CAP patients in this current study originally participated in studies conducted by Walker and colleagues (Walker, Garber, Smith, Van Slyke, & Claar, 2001; Walker, Smith, Garber, & Claar, 2005) between 1993 and 2004. These studies enrolled consecutive new patients who presented to the Vanderbilt Pediatric Gastroenterology Clinic for evaluation of abdominal pain when they were 8–18 years old. Patients were eligible for participation in the original studies if they lived with parent(s) or parent figure, reported abdominal pain of at least 3 months' duration, had no history of chronic illness or disability, and no organic disease diagnosis for abdominal pain from the referring physician. Participants were excluded from the current study if they were less than 18 years of age, less than four years had elapsed since initial study enrollment, evidence of significant organic disease was found in the CAP evaluation at Vanderbilt, or they reported other significant chronic disease.

Healthy Control participants. Healthy Control participants in this study participated in control groups drawn from local schools for the original studies when they were between the ages of 8 and 16 years (Walker, et al., 2001; Walker, et al., 2005; Walker, et al., 2006). To be eligible for the original studies, participants had to have no chronic illness and no abdominal pain in the month preceding initial study participation. For the current study, eligibility criteria included: 18 years of age or older at follow-up, and at least four years elapsed since initial study enrollment.

Procedure

760 former CAP and 343 former Control participants met eligibility for age and follow-up interval. They were sent letters with a card to return to decline further contact. Among CAP, 6 declined contact, leaving 754 potential participants. Of these, 261 (34%) could not be located, 54 (7%) declined participation, 40 (5%) could not be scheduled, 3 were excluded due to recent onset of chronic disease, 5 were excluded due to missing data, leaving a CAP follow-up sample of 391. CAP participants and non-participants did not differ significantly on gender, age, or baseline pain severity. For the purposes of this study, only participants 18 years of age and older were analyzed due to differences in data collection between adolescent and young adult participants on a pertinent measure, leaving a final CAP sample of 319.

Among Controls, 3 declined contact, leaving 340 potential participants. Of these, 110 (32%) could not be located, 20 (6%) declined participation, 23 (7%) could not be scheduled, 1 was excluded due to missing data leaving a Control sample of 186. Control participants and non-participants did not differ significantly on gender or age. Only participants 18 years of age and older were analyzed for the current study for reasons described previously, leaving a final Control sample of 127.

Participants completed a telephone interview where they answered questions about their health, current chronic pain symptoms, parental pain history, and FGID status. Then, participants were invited to participate in the laboratory portion of the study where they completed heat pain sensitization testing. Procedures for both the telephone interview and laboratory portion were approved by the Vanderbilt Institutional Review Board.

Measures

The Rome III Diagnostic Questionnaire for Functional Gastrointestinal Disorders was utilized in this study to assess the Rome III symptom criteria for three common abdominal pain related FGIDs in adulthood—irritable bowel syndrome, functional dyspepsia, and functional abdominal pain syndrome (Drossman & Dumitrascu, 2006). Participants answered 24 questions regarding their experiences with each symptom over the past 3 months. Participants were classified as having persistent CAP or resolved CAP, depending on whether or not they met criteria for an abdominal pain related FGID at follow-up.

The Persistent Pain Questionnaire (PPQ) assessed history and location of any chronic pain, as well as parental chronic pain history (Bruehl, France, France, Harju, & al'Absi, 2005). Participants were asked to identify locations of both current and lifetime chronic pain based on the standard eight body locations described by the International Association for the Study of Pain. For the purposes of this study, the abdominal pain site was excluded because the Abdominal Pain Index and Rome III questionnaire provided a detailed assessment of current abdominal pain. For each site, participants were asked if they had "ever experienced chronic pain daily or almost daily for 3 months or more." If they responded positively to this question, participants were asked if this experience was in the past 3 months. If "yes," participants were then asked to rate their current pain on a scale of 0-100. The presence of a current non-abdominal pain site was indicated if the participant rated a current non-abdominal pain site at greater than or equal to 30.

Each participant was also asked if their biological mother and biological father, respectively, had ever "experienced chronic pain daily or almost daily for three months or more." Three scores were computed: (1) positive maternal chronic pain history, (2) positive paternal

chronic pain history, and (3) positive chronic pain history in both parents. If the participant did not know the chronic pain history for a parent, a score for dual parent history of chronic pain was not computed.

The Abdominal Pain Index (API) assessed the frequency, duration, and intensity of abdominal pain episodes over the past two weeks (Walker, Smith, Garber, & Van Slyke, 1997). We computed a composite score based on participants' responses to the 4-item index. All items were converted to a 6-point scale ranging from 0 to 5 and all four items were summed to yield a total score, ranging from 0 to 20. Alpha reliability for the API in this study was .67.

The Children's Somatization Inventory (CSI) was designed to evaluate the severity of 35 somatic symptoms over the past two weeks (Walker, Beck, Garber, & Lambert, 2009; Walker, Garber, & Greene, 1991). Participants reported how much each symptom bothered them on a 5-point scale ranging from 0 to 4. The items were summed, yielding a total score ranging from 0 to 140. Alpha reliability for the CSI was .87 in this study.

The Functional Disability Inventory (FDI) assessed difficulty in physical and psychosocial functioning due to physical health. Participants reported how much difficultly they would have completing 15 items due to their physical health on a 5-point scale ranging from 0 to 4. The items were summed, yielding a total score between 0 and 60. Alpha reliability for the FDI was .87 in this study.

The Health Service Utilization Questionnaire was developed for this study to assess participants' recent health service utilization, medical diagnoses, and use of prescription medications. For the purposes of this study, 2 specific outcome variables were assessed: (1) use of 4 or more prescription medications, and (2) whether or not the participant had been admitted to an emergency room in the past 3 months.

Education and Work History Report Form asked participants about special circumstances pertaining to education and work history that have impacted the participant's functioning or illness. Specifically, all participants reported whether they ever quit or lost a job because illness interfered with their ability to work.

Pain Threshold and Tolerance were assessed through quantitative sensory testing with a computer controlled Medoc Thermal NeuroSensory Analyzer (TSA-II, Medoc, Inc, Ramat, Israel). As described in prior studies, this device, using a 30 × 30-mm Peltier thermistor probe, applied heat stimuli to the nondominant ventral forearm (Chung & Bruehl, 2008; Dengler-Crish, Bruehl, & Walker, 2011; Fillingim & Edwards, 2005). Participants were trained and then completed practice trials for both pain threshold and pain tolerance tests prior to completing the actual trials in order to achieve consistency. For pain threshold, participants were instructed to indicate, with a mouse click, when the heat first changed from feeling warm to feeling painful. For pain tolerance, participants were told to indicate, with a mouse click, when they could no longer stand the heat pain. For detailed descriptions of the temperature settings, see Dengler-Crish et al. (2011). For this study, mean temperatures for pain threshold and tolerance were computed based on the last 3 of 4 trials.

Pain Sensitization (Wind-up) was assessed with a protocol formerly used in several previous studies (Chung & Bruehl, 2008; Dengler-Crish, et al., 2011). The wind-up assessment trials were designed specifically to assess C-fiber mediated temporal summation through the administration of a standardized oscillating thermal stimulation protocol. For wind-up assessment, 2 sequences of 10 heat pulses each were applied to the ventral forearm with the TSA-II unit using commercially available software (TPS-CoVAS version 3.19, Medoc Inc.). In order to maximize the likelihood of measurable results, we administered two sequences at two

different stimulus intensities (47ºC and 48ºC; (Fillingim & Edwards, 2005). Participants were asked to rate the intensity at the peak of every heat pulse within each sequence on a 0-100 numerical rating scale (0 = "No Pain" and 100 = "Worst Possible Pain"). As indices of wind-up, we calculated the slopes of the lines for each individual by using within-subject regressions fitted to the series of 10 pain ratings at each stimulus intensity (47ºC and 48ºC). See Dengler-Crish et al. (2011) for a more detailed description of these procedures.

Data Analysis

All data analyses were conducted using IBM SPSS version 19.0. We conducted 3 X 3 chi square analyses to examine participants' reports of parental pain history by participant group. Because a similar number of participants in each group responded "Don't Know" regarding their parental pain history, these responses were excluded from the remainder of the analyses. We conducted descriptive statistics to test for the assumption of normality and examine the frequency of elevated symptom severity. Based on this, the measure of somatic symptoms, CSI, and measure of functional disability, FDI, were logarithmically transformed, and we decided to exclude the Healthy Control group because their low rates of symptoms precluded meaningful analyses. ANCOVA analyses for group (2) by parental chronic pain history (2) with age as a covariate examined the association of parental chronic pain history and CAP status to two self-report measures of participants' abdominal pain (API) and somatic symptom severity (CSI) at follow-up. We conducted logistic regressions examining parental chronic pain history as a predictor of dichotomous chronic pain outcomes, with age as a continuous covariate. To examine the relationship between parental chronic pain history and gender, we conducted a 2 x 2 chi-square. ANCOVA analyses for gender (2) by parental chronic pain history (2) with age as a

covariate examined the association of parental chronic pain history and gender on abdominal pain and somatic symptom severity.

CHAPTER III

RESULTS

Demographic Characteristics

Participants (n = 446) completed the follow-up study between 4 and 16 years (M = 9.36, SD = 3.33) after enrollment in the original studies (Table 1). At follow-up, the participants were classified into three groups: Healthy Control (n = 127), Recovered CAP (i.e. no longer met Rome III criteria for an abdominal pain related FGID; n = 193), or Persistent CAP (i.e. met current Rome III criteria for an abdominal pain related FGID; n = 126). The CAP groups were significantly older than the Healthy Control group both at baseline [F (2,443) = 6.87; p = .001] and follow-up [F (2,443) = 21.92; p = .000]. Additionally, the Persistent CAP group included a greater proportion of females than the Healthy Control group [X^2 (2, 446) = 10.17; p = .006]. The three groups were mostly Caucasian and did not differ on race and ethnicity.

Table 1. *Demographic characteristics by participant group*

	Participant Group		
Demographics	Healthy Controls (n = 127)	Recovered CAP (n = 193)	Unrecovered CAP (n = 126)
Sex (% Female)	52.0[a]	62.2[a,b]	71.4[b]
Race and Ethnicity (% Caucasian)	95.3	89.6	96.0
Age at Initial Evaluation (years)	11.47 (2.11)[a]	12.20 (2.54)[b]	12.55 (2.35)[b]
Age at Follow-up (years)	20.08 (2.17)[a]	21.97 (3.17)[b]	22.28 (3.19)[b]
Follow-up Interval (years)	8.30 (2.42)[a]	9.80 (3.57)[b]	9.75 (3.54)[b]

Note. Within rows, means with different superscripts differ significantly at p < .01.

Incidence of Parental Chronic Pain History

Maternal chronic pain history, paternal chronic pain history, and a chronic pain history in both parents differed significantly by group, X^2 (4, 446) = 24.62; p < .001, X^2 (4, 446) = 13.48; p = .009, and X^2 (4, 446) = 15.05; p = .005, respectively. The majority of Persistent CAP participants (59.5%) had mothers with a positive chronic pain history in comparison to 40.9% of Recovered CAP participants and 29.1% of Healthy Controls (see Figure 1). Persistent CAP participants reported a greater proportion of fathers with a positive chronic pain history (39.7%) as compared to Recovered CAP participants (28.0%) and Healthy Controls (28.3%). Additionally, a positive chronic pain history for both parents was significantly more common in Persistent CAP participants (28.6%) compared to Healthy Controls (11.8%). The Recovered CAP participants (19.2%) did not differ significantly from either Persistent CAP participants or Healthy Controls.

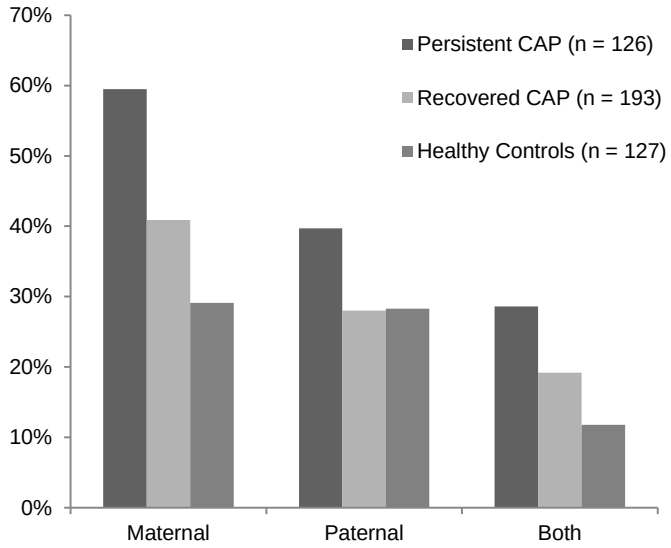
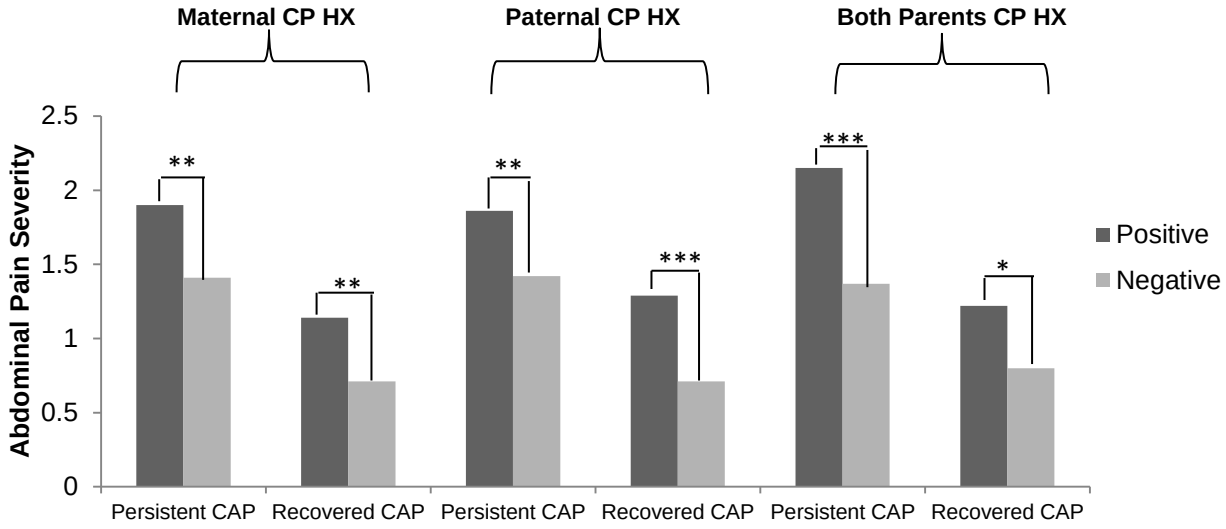


Figure 1. Percent of participants in each group with a positive history of chronic pain in mother, father, and for both parents

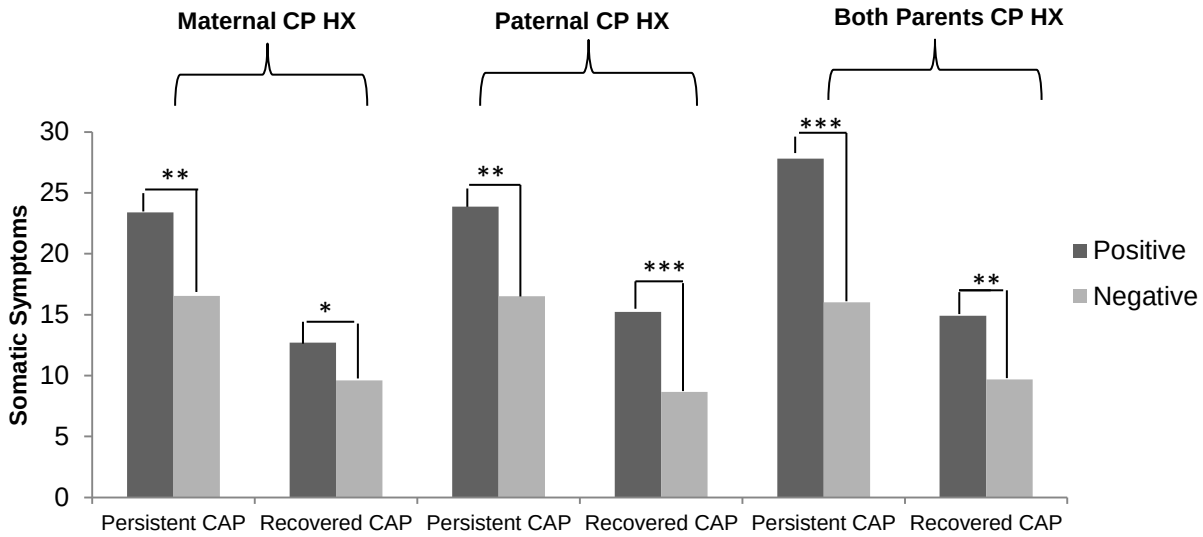
Relation of Parental Chronic Pain History and CAP status to Pain Experience

Self-report measure of symptom severity and disability. A positive history of chronic pain in mothers, fathers, or both parents was associated with higher levels of pain, somatic symptoms, and disability in both recovered and persistent CAP participants (Figures 2, 3, and 4). Specifically, results revealed significant main effects for maternal chronic pain history and group on participants' abdominal pain severity [maternal chronic pain, $F(1,285) = 17.97$, $p < .000$, and group, $F(1,285) = 43.60$, $p < .001$], somatic symptoms [maternal chronic pain, $F(1,285) = 15.41$, $p < .000$, and group, $F(1,285) = 53.85$, $p < .001$], and functional disability [maternal chronic pain, $F(1,285) = 14.05$, $p < .001$, and group, $F(1,285) = 18.62$, $p < .001$]. Similar significant main effects were found for paternal chronic pain history and group on participants' abdominal pain severity [paternal chronic pain, $F(1,250) = 19.16$, $p < .001$, and group, $F(1,250) = 29.85$, $p < .001$], somatic symptoms [paternal chronic pain, $F(1,250) = 25.22$, $p < .001$, and group, $F(1,250) = 39.50$, $p < .001$], and functional disability [paternal chronic pain, $F(1,285) = 10.90$, $p < .01$, and group, $F(1,285) = 15.87$, $p < .001$]. Additionally, significant main effects were found for a chronic pain history for both parents and group on participants' abdominal pain severity [both parents chronic pain, $F(1,244) = 23.98$, $p < .000$, and group, $F(1,244) = 37.58$, $p < .000$], somatic symptoms [both parents chronic pain, $F(1,244) = 27.48$, $p < .000$, and group, $F(1,244) = 41.86$, $p < .000$], and functional disability [both parents chronic pain, $F(1,244) = 23.67$, $p < .000$, and group, $F(1,244) = 17.03$, $p < .000$]. No significant interaction effects were found for group by maternal chronic pain history, group by paternal chronic pain history, or group by chronic pain history for both parents for abdominal pain severity, somatic symptoms, or functional disability.



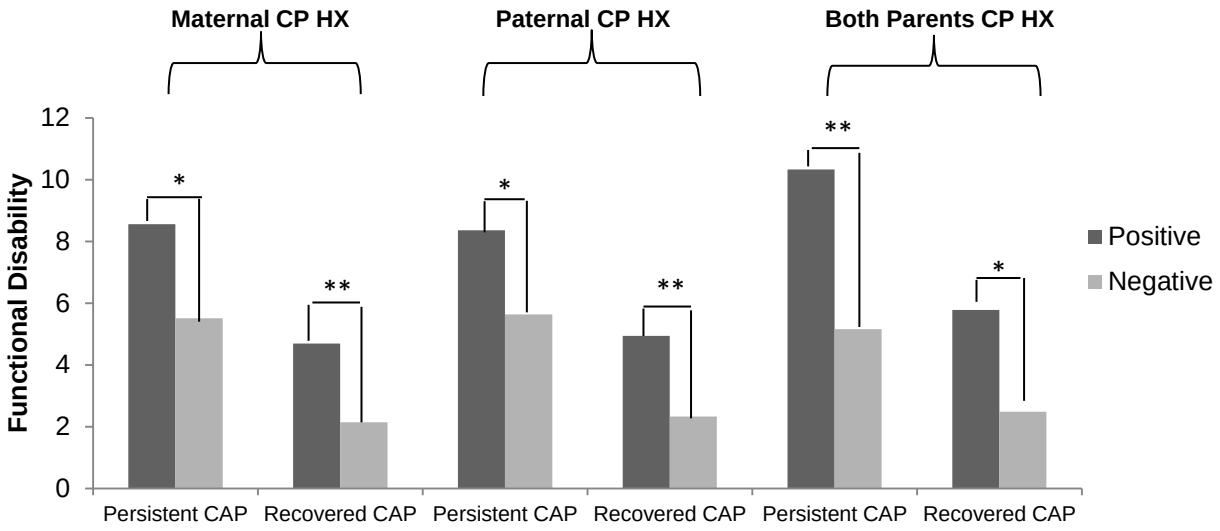
* p < .05, ** p < .01, *** p < .001; Maternal CP HX = Maternal Chronic Pain History; Paternal CP HX = Paternal Chronic Pain History; Both Parents CP HX = Chronic Pain History for Both Parents

Figure 2. Abdominal pain severity in CAP patient groups by chronic pain history in their mothers, fathers, and both parents



* p < .05, ** p < .01, *** p < .001; Maternal CP HX = Maternal Chronic Pain History; Paternal CP HX = Paternal Chronic Pain History; Both Parents CP HX = Chronic Pain History for Both Parents

Figure 3. Somatic symptoms in CAP patient groups by chronic pain history in their mothers, fathers, and both parents



* p < .05, ** p < .01; Maternal CP HX = Maternal Chronic Pain History; Paternal CP HX = Paternal Chronic Pain History; Both Parents CP HX = Chronic Pain History for Both Parents

Figure 4. Functional disability in CAP patient groups by chronic pain history in their mothers, fathers, and both parents

Other health related outcomes. The results from logarithmic regression indicated that CAP participants who reported a positive maternal chronic pain history, compared to participants with a negative maternal chronic pain history, were twice more likely to report the presence of non-abdominal chronic pain (OR: 2.10; 95% CI: 1.26-3.51; p = .004), 2.17 times more likely to currently take four or more prescription medications, (OR: 2.17; 95% CI: 0.98-4.78; p = .055), 2.41 times more likely to have visited the emergency room in the past 3 months (OR: 2.41; 95% CI: 1.20-4.83; p = .013), and 2.67 times more likely to have lost a job due to illness (OR: 2.69; 95% CI: 1.27-5.69; p = .010). Moreover, CAP participants who reported a positive paternal chronic pain history, compared to participants with a negative paternal chronic pain history, were 4.51 times more likely to report the presence of non-abdominal chronic pain (OR: 4.51; 95% CI: 2.56-7.96; p < .001), 2.84 times more likely to take four or more prescription

medications (OR: 2.84; 95% CI: 1.24-6.53; p = .014), 2.39 times more likely to have visited the emergency room in the past 3 months (OR: 2.39; 95% CI: 1.19-4.81; p = .014), and 3.02 times more likely to have lost a job due to illness (OR: 3.02; 95% CI: 1.36-6.69; p = .006). Finally, CAP participants who reported a positive chronic pain history for both parents, compared to participants with a negative paternal chronic pain history for one or both parents, were 3.89 times more likely to report the presence of non-abdominal chronic pain (OR: 3,89; 95% CI: 2.17-6.972; p < .001), 3.59 times more likely to take four or more prescription medications (OR: 3.59; 95% CI: 1.58-8.16; p = .002), 3.31 times more likely to have visited the emergency room in the past 3 months (OR: 3.31; 95% CI: 1.63-6.72; p = .001), and 3.45 times more likely to have lost a job due to illness (OR: 3.45; 95% CI: 1.57-7.60; p = .002)

Laboratory pain experience. Pain threshold did not differ by group [$F(1,156) = .131$, $p > .50$], maternal chronic pain history [$F(1,156) = .039$, $p > .50$], paternal chronic pain history [$F(1,138) = .843$, $p > .10$], or chronic pain history for both parents [$F(1,134) = 1.64$, $p > .10$]. Similarly, pain tolerance did not differ by group [$F(1,156) = .182$, $p > .10$], maternal chronic pain history [$F(1,156) = .182$, $p > .50$], paternal chronic pain history [$F(1,138) = 1.36$, $p > .10$], or chronic pain history for both parents [$F(1,134) = .253$, $p > .50$]. Finally, wind-up did not significantly differ by group [47°, $F(1,147) = 1.38$, $p > .10$, and 48°, $F(1,147) = 1.81$, $p > .10$], maternal chronic pain history [47°, $F(1,147) = 1.17$, $p > .10$, and 48°, $F(1,147) = .411$, $p > .50$], paternal chronic pain history [47°, $F(1,128) = .091$, $p > .50$, and 48°, $F(1,128) = .167$, $p > .50$], or chronic pain history for both parents [47°, $F(1,125) = 2.56$, $p > .10$, and 48°, $F(1,125) = 2.07$, $p > .10$]. No significant interaction effects emerged regarding the relationship of these laboratory pain measures to maternal chronic pain history, paternal chronic pain history, or chronic pain history for both parents and group (all p-values > .05).

Exploration of Gender Effects

Results of chi square analyses revealed no significant associations of participant gender with maternal chronic pain history, paternal chronic pain history, or chronic pain history for both parents. Additionally, the data did not support an interaction between maternal chronic pain history, paternal chronic pain history, or chronic pain history for both parents and gender for current abdominal pain symptoms, somatic symptoms, functional disability, or laboratory measures of pain experience. Positive maternal chronic pain histories, paternal chronic pain histories, or a positive chronic pain history for both parents corresponded with greater symptoms and self-reported pain experience in both girls and boys. Significant main effects for gender were found for abdominal pain severity [$F(1,285) = 31.80$, $p < .001$], somatic symptoms [$F(1,285) = 25.64$, $p < .001$], and functional disability [$F(1,285) = 11.57$, $p < .01$]. However, main effects for gender were not statistically significant for laboratory measures of pain experience (i.e. pain threshold, pain tolerance, wind-up; all p-values > .05). Additionally, no gender by parent chronic pain history interactions were found in predicting the presence of current non-abdominal chronic pain (p-values > .05). CAP patients who reported a positive parent chronic pain history were more likely to have current non-abdominal chronic pain regardless of gender.

CHAPTER IV

DISCUSSION

This investigation sought to examine the relation between parent chronic pain history and persistence of CAP in pediatric patients. A recent meta-analysis found a significant small-to-medium effect size for the relation between parental somatic symptoms to functional abdominal pain (FAP) in their children (van der Veek et al., 2011), but none of the reviewed studies examined whether this association continued over time. The current study found at long-term follow-up, that young adults with persistent CAP had a greater likelihood of having had a mother, father, or both parents with chronic pain. However, young adults who no longer had a clinically significant FGID at follow-up did not differ significantly from Healthy Controls. Thus, parental chronic pain history may serve as an important predictor of chronic pain persistence in CAP patients.

While persistent and recovered CAP patients differed in likelihood of parental chronic pain history, participants in both CAP groups had more severe somatic symptoms, abdominal pain, and disability if one of their parents had a history of chronic pain. Notably, CAP patients who recovered and no longer had a clinically significant FGID at follow-up still reported significantly higher levels of subclinical symptoms and disability if one of their parents had a history of chronic pain than if neither parent had such a history. This is consistent with studies that have found an association between parent pain history and pain symptoms in non-clinical samples of adults (Bruehl, et al., 2005; Lester, Lefebvre, & Keefe, 1994). Additionally, regardless of persistence or recovery, former CAP patients who reported a positive parental

chronic pain history were more likely than former CAP patients with a negative parental chronic pain history to have other clinically significant non-abdominal chronic pain, greater health service utilization, and job loss due to illness. These outcomes all have great public health significance and suggest that the implications of the relation between parent and child chronic pain may extend beyond the family system and into the public sphere.

Contrary to our hypothesis, gender did not seem to play a large role in the association between parent and child chronic pain in the current study. A positive chronic pain history in mothers or fathers corresponded with greater symptoms and disability in both daughters and sons. Studies in the pediatric chronic pain literature typically examine the association between mother and child symptoms (Helgeland, Van Roy, Sandvik, Markestad, & Kristensen, 2011; Kashikar-Zuck et al., 2008; Saunders, Korff, Leresche, & Mancl, 2007), but evidence from the current study suggests that the association between father and child symptoms is equally as important. This association could be explained by both genetic and environmental factors. Chronic pain could be linked to some combination of recessive alleles where both parents have to contribute the same genetic material in order for a child to inherit a specific vulnerability towards pain sensitivity. Additionally, in homes where the child interacts with both parents regularly, mothers and fathers both have opportunities to model positive or negative pain behaviors for their children.

The present study was not designed to address the mechanisms underlying the link between parents' chronic pain history and children's pain persistence, but the current body of literature supports several plausible explanations for this association. In terms of biological mechanisms, a study of chronic low back pain patients found that a positive parental chronic pain history was associated with endogenous opioid analgesic dysfunction, suggesting that

dysfunction in this pain response system could be heritable (Bruehl & Chung, 2006). In terms of social learning mechanisms, children may learn pain behaviors through parental modeling or reinforcement of pain behaviors. One experimental study found that children whose parents attended to their symptom complaints during a laboratory task experienced more gastrointestinal symptoms than children whose parents distracted them during the same task (Walker, et al., 2006). Parents who have experienced chronic pain themselves may be more likely to attend to their child's pain complaints, thereby reinforcing these pain behaviors. However, few studies have directly assessed this notion in an experimental context and this hypothesis needs further empirical support (van der Veek, et al., 2011).

This is the first study we know of to examine the relationship between parental chronic pain history and the persistence of pediatric chronic pain in a pediatric population. A major strength of this study was the examination of both clinical and subclinical abdominal pain symptoms in relation to parental chronic pain history. We found that even among former CAP patients who no longer had a clinically significant FGID at follow-up, a positive parental history of chronic pain was associated with subclinical abdominal pain at follow-up. Another unique finding from the current study was the association between parental chronic pain history and health service utilization by their children in young adulthood. Additional research is needed on parent health service utilization and disability in order to examine possible contributions of specific parental health service seeking behaviors and impairment to child health service utilization and disability.

There are several limitations to the current study. Parental chronic pain histories were obtained solely by child self-report. A study by Bruehl and colleagues (2005) comparing offspring and parent report of parental chronic pain history, found high sensitivity values

indicating offspring generally tended to accurately report positive parental chronic pain histories. While this evidence suggests child self-report is a valid way to measure parental chronic pain history, future studies should obtain both child and parent reports of parental chronic pain history. Additionally, despite the limitations of self-report data, a child report of a positive parental chronic pain history suggests that either significant pain behaviors are being modeled or the parent is exhibiting some type of behavior that the child has interpreted to mean the parent had chronic pain. It is possible that children's reports of parental chronic pain are biased by their own pain experiences, but this still has significant implications for the association between children's and parents' pain. In the current study, a significant association between parents' and children's pain was found for self-report measures, but not for laboratory measures of pain sensitivity. In contrast, previous studies have found a significant association between familial or parental chronic pain history and the experimental pain responses of their children (Bruehl & Chung, 2006; Fillingim, et al., 2000). The differences in populations could have contributed to the differences in findings between the current study and the two prior studies. These prior studies were conducted in adult participants with chronic low back pain and healthy young adults.

The results from the present study have implications for the development of interventions. Because parental chronic pain history was significantly associated with child pain persistence and both clinical and subclinical symptom complaints, assessing parental chronic pain history at the time of children's initial evaluation for CAP could help identify those who are at greater risk for long-term chronic pain persistence. Additionally, while more studies are needed to establish that parental modeling influences child pain behaviors, the association between parental chronic pain history and child health service utilization and disability in this

study suggests that parental modeling may play a role in the development of children's pain behaviors. If this is the case, then interventions designed to reduce the number of pain behaviors exhibited by parents may help decrease the use of health services and increase daily functioning in both the parent and child.

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