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Parental Bonding in Adolescents With and Without Chronic Pain

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Abstract

Objective Parental responses influence children's pain; however, the specific role of parental bonding in pediatric pain has not been examined. Depressive symptomology is frequently reported in children with chronic pain (CP) and may play a role in the relationship between parental bonding and pain. This study examined the connections between maternal/paternal bonding (perceived care and control) and symptoms of pain and depression in adolescents with CP and in healthy adolescents. **Method** Participants included 116 adolescents (aged 12–17) with CP ($n = 55$) and without ($n = 61$). Adolescents completed the Parental Bonding Instrument separately for their mother and father, as well as measures of depression and pain. **Results** Significant associations between parental bonding and adolescent pain and depression emerged in the pain group, but not in the healthy group. There were no differences in the impact of maternal versus paternal bonding on adolescent pain and depression. Mediation analyses revealed adolescent depression was a mediator of the relationship between maternal care and adolescent pain, and paternal control and adolescent pain in the group with CP. **Conclusions** This study highlights the importance of considering parental bonding and adolescent depression in pediatric CP, suggesting that high paternal control and low maternal care contribute to increased pain in adolescents through heightened adolescent depressive symptoms. The findings emphasize the need for family-based treatment for CP that addresses parent behaviors and adolescent mental health.

Key words: adolescents; chronic and recurrent pain; parenting.

Introduction

Pediatric chronic pain (CP) is a widespread developmental health issue, currently affecting up to 30% of children (Eccleston et al., 2012; Palermo & Chambers, 2005). Many of these children also report symptoms related to emotional well-being including depression (Avagianou, Mouzas, Siomos, & Zafiropoulou, 2010; Carter & Threlkeld, 2012; Laird, Preacher, & Walker, 2015). It is understood that parental responses and behavior, such as solicitousness and criticism, influence pain and functional outcomes in children with CP (Evans et al., 2008; Palermo & Chambers, 2005). However, the role of parental bonding, an attachment concept that reflects elements of parental care and control, has not yet been examined in children's CP or related mental health.

It is well-recognized that healthy attachment positively influences the child's mental and physical development (Ainsworth & Bowlby, 1991). Although hypothesized to play a role in the development and maintenance of CP in childhood, an attachment framework has not yet been empirically explored in pediatric CP. Parental bond, considered within the attachment framework, refers to a parent's behaviors and attitudes toward their child, as perceived by the child (Bretherton, 1992). Parental bond includes

dimensions of "care" and "control" and is related to the child's attachment security. Parents rated as high on "care" are perceived as warm and affectionate, and parents rated as low may be perceived as cold, rejecting, or withdrawn. "Control" is characterized by overprotection and restrictive parental behaviors and attitudes. Parents who are low on "control" enable autonomy and independence (Parker, Tupling, & Brown, 1979). Healthy parental bond comprises high care and low control, and is believed to positively influence the child's development of self-concept, ability to self-soothe, and regulate emotion (Parker et al., 1979). In the acute pain literature, unhealthy bond indices, including high parental control are related to greater pain reaction and negative emotion in young children undergoing immunization (Walsh, McGrath, & Symons, 2008). Adult research via retrospective reports also suggests that parental bonding influences the development of CP. A recent study conducted in Japan reported that adults with CP recalled higher rates of parental control and lower parental care than did healthy adults (Anno et al., 2015). Similarly, Avagianou and colleagues (2010) found that in 65 adult patients with CP, those who reported experiencing low parental care and high control in childhood, especially from mothers, had higher bodily pain ratings than did patients with a healthy parenting bond in childhood. Although suggestive of a link between unhealthy parental bond (i.e., high parental control and low care) and worse pain outcomes in children, these studies are limited by retrospective reports of parent bond, and need to be replicated in pediatric patients.

A potential mechanism for the relationship between unhealthy parent bond and increased offspring pain is the presence of depression. In child and adult samples, parental responses that are controlling or overprotective are linked to increased depression (Avagianou et al., 2010; Cohen, Vowles, & Eccleston, 2010; Davies, Macfarlane, McBeth, Morriss, & Dickens, 2009). High control and low care from mothers seems to be particularly related to depression in adulthood (Avagianou et al., 2010; Lung, Huang, Shu, & Lee, 2004), with evidence that high maternal control is associated with depression in adolescent children independently of care ratings (Martin, Bergen, Roeger, & Allison, 2004). It is thought that attachment dynamics such as parent bond shapes the child's schemas and expectations regarding others, as well as affects their cognitive appraisals of interpersonal events, with dysfunctional attachment leading to distorted expectations and appraisals consistent with depression (Lee & Hankin, 2009). It is plausible that the relationship between dysfunctional parent bond and CP occurs in part through such an increase in maladaptive thought patterns associated with depression. Further empirical work is necessary to demonstrate this theoretical link.

Adolescence is a particularly important watershed for understanding the relationships between parent bond, depression, and pain, as it is a time of rapid growth and autonomy, and the presence of CP may disrupt this developmental period (Palermo, Valrie, & Karlson, 2014; van Wel, Linssen, & Abma, 2000). Adolescent CP can lead to increased reliance on caregivers, with a corresponding interference in the process of remaining connected yet separate, which is required for a healthy adolescence (Evans, Meldrum, Tsao, Fraynt, & Zeltzer, 2010). Teens with CP may then have reduced opportunities to test their emerging interpersonal and emotion regulation skills required for enduring mental health, which in turn may exacerbate pain. The present study is therefore focused on understanding the relationships among parental bond, depression, and pain during this time of particular sensitivity.

The current study represents the first known research into adolescent self-reported perceptions of maternal *and* paternal bond within a pediatric CP sample. Although the importance of maternal care and control has emerged within the adult literature, the role of father bond is yet to be adequately examined. Another innovation of the study is the use of current adolescent report of parent bond (rather than retrospective reports from adult patients), which provides an opportunity to gain insight into present feelings about parents' behavior, relying less on memory and therefore providing a more accurate characterization of the currently perceived parent bond (Lyddon, Bradford, & Nelson, 1993).

The first aim of the study was to examine the relationships between parental bonding and adolescent pain and depression in adolescents with CP and in healthy control (HC) adolescents. It was hypothesized that there would be significant associations among unhealthy maternal and paternal bonding (high levels of control and low levels of care) and adolescent pain and depression ratings. The second aim of the study was to examine potential differences in maternal and paternal bonding for adolescent pain ratings; given the lack of previous studies addressing this question, associations between maternal/paternal bond and adolescent pain were explored. Finally, the role of adolescent depression was examined as a potential mediator of the relationship between both maternal bonding and adolescent pain ratings, and paternal bonding and adolescent pain ratings.

Method

Participants

The sample for this study was a part of a larger study at the University of California, Los Angeles (UCLA), examining pubertal and gender differences in responses to pain in HCs and those with CP. Previous published papers from the larger data set have

included findings on predictors of laboratory pain and conditioned pain modulation (Evans et al., 2016; Evans, Seidman, Lung, Zeltzer, & Tsao, 2013; Evans, Seidman, Tsao, Lung, Zeltzer, & Naliboff, 2013; Tsao et al., 2013), mother–child pain concordance for pain location (Schwartz, Seidman, Zeltzer, & Tsao, 2013), and menstrual pain in adolescent girls (Payne et al., 2016).

The recruitment and research procedures received UCLA ethics approval. Participants with CP were recruited through a multidisciplinary, tertiary clinic specializing in pediatric CP (approximately 10% were recruited though craigslist postings). Inclusion criteria followed the commonly accepted definition of pain persisting for three months or longer. Each diagnosis of a CP condition was confirmed by a pediatrician specializing in CP. HC participants were recruited through posted advertisements, community events, and referrals from previous participants. Study advertisements were posted on online forums (e.g., Craigslist, local Yahoo groups) as well as at locations where parents and children would be expected to encounter them (e.g., libraries, pediatricians' offices). Previous participants were offered the opportunity to refer their friends/neighbors and earn an additional $25 for each referred family that completed the study.

Eligibility was confirmed by telephone. Parents were asked whether their child met any of the following exclusionary criteria: age outside desired range (8–17 years); daily use of opioids; developmental delay; autism. For the larger study, 364 families (223 control, 141 pain) were screened for eligibility by telephone: eight families [five control (2.2% of group), three pain (2.1% of group)] were excluded as a result of acute injury/illness or developmental delay/autism. Of the 356 families (218 control, 138 pain) invited to participate, 110 [52 control (23.9% of group), 58 pain (42.0% of group)] declined to participate mainly owing to lack of interest or scheduling difficulties. Three families were removed from the control group after enrollment owing to ineligibilities that were discovered after study completion (child age below minimum required and child developmental delay).

The present study sample included only adolescents aged 12–17 years ($n = 116$) presenting as either healthy ($n = 61$) or meeting inclusion criteria for CP (diagnosed by either a primary care provider or tertiary CP specialist) ($n = 55$). The average age of children was 15 years (range = 12–17 years). Participants were recruited through a multidisciplinary, tertiary pediatric pain clinic (CP) or through craigslist postings or advertisements within the community (HC). Mothers and fathers could be biological or adoptive parents of the child; children were included whether they were living with two parents, a mother, or a father only. Each person in the participating triad was compensated with $50.

Procedure

Mothers and fathers completed written informed consent, and adolescents completed written assent. Families visited the laboratory (families in which a father also attended $n = 30$), where they completed questionnaires and underwent a number of laboratory pain tasks. Only those questionnaires relevant to the current study are discussed herein. The entire session lasted approximately 2 hr.

Measures

Demographics

Mothers completed a questionnaire that was designed for the larger study on gender differences, and was used to attain information relating to child and parents' age, mother's marital status, and child biological sex, and race/ethnicity.

Pain over the Past Month

Bodily pain intensity (over the past month) experienced by participants was assessed using a Numeric Rating Scale (NRS). Children were asked to indicate worst levels of bodily pain intensity over the past month on scale of 0 to 10, with 0 being *no pain* and 10 being the *worst pain possible*. For children who reported having no pain over the past month, a score of 0 was entered for this variable. The NRS is widely used, and has been established as a valid and reliable measure of pain intensity for children (von Baeyer et al., 2009).

Depression

The Revised Children's Anxiety and Depression Scale (RCADS) depression subscale was used to assess major depressive symptomology. This subscale consists of 10 items and is part of the 47-item RCADS. Participants are asked a series of questions relating to feelings of sadness and/or hopelessness and rate themselves as never, often, sometimes, or always. Possible depression subscale scores range from 0 to 30, with higher scores indicating greater levels of depressive symptomology. Factorial, convergent, and discriminant validity and reliability have been demonstrated for this measure (Chorpita, Moffitt, & Gray, 2005).

Parental Bonding

A modified version of the Parental Bonding Instrument (PBI), the Parental Bonding Instrument—Brief Current version, was used to measure how adolescent participants perceive their parents' behaviors and attitudes toward them. The scale measures both control and care over the past 3 months. The questionnaire consists of two identical sets of eight questions, one pertaining to the mother and one pertaining to the

father. Each item was rated on a three-point scale as "never", "sometimes," or "usually." Although originally developed for adults reflecting on their childhood experiences, the brief current measure has been validated for use with adolescents (Klimidis, Minas, Ata, & Stuart, 1992).

Data Analysis

Data were entered into SPSS 21 and descriptive analyses were undertaken to assess normality, missing data, sample size, and characteristics of the sample. Power calculations were conducted using Monte Carlo simulations in Mplus v7.2. For each model, the unstandardized observed effect sizes obtained in the study and product of coefficients approach for mediation effect were used to generate 10,000 simulated data sets. Based on an alpha = .05, analysis revealed that our sample of 53 participants had 21.1% power to detect a true mediation effect for the maternal care model, 14.3% power to detect a true mediation effect for maternal control, and 24.9% power to detect a true mediation effect for paternal control.

Each hypothesis was examined separately for CP and HC groups to understand the factors specific to each group. The first and second hypotheses were tested using partial correlations (controlling for variables found to differ significantly) to explore associations between maternal and parental care/control, adolescent pain, and depression in each group. For Hypothesis 2, significance of the difference between two correlation coefficients was performed on the significant maternal bonding–adolescent pain and paternal bonding–adolescent pain correlations to examine whether maternal or paternal bonding was more strongly related to adolescent pain. This analysis involves Fisher r to z transformation to assess the significance of the difference between the two correlation coefficients. For Hypothesis 3, path models were then estimated using SPSS (Hayes, 2013) to examine adolescent depression as a possible mediator of the relationship between mother and father care and control and the adolescent's pain. Assumptions for mediation were tested and met before conducting these analyses through examination of residuals and collinearity statistics, including correct specification of model in its functional form, no omitted variables, no reverse causality effect, no interaction and homogeneity of error (Cohen, Cohen, West, & Aiken, 2003). Mediation was only tested when preconditions regarding significant associations between the variables of interest were met.

Results

Descriptive Statistics

A summary of the descriptive information is presented in Table I, including sociodemographic information such as child sex, age, race, and maternal marital status. *T*-tests for continuous data and chi-square tests for categorical data revealed no significant differences between the groups on age or ethnicity; however, there were significant sex and race group differences, with more males in the HC group ($t(114) = -2.21$, $p < .05$), and more African Americans in the HC group compared with the CP group ($t(112) = 2.84$, $p < .05$). In addition, the marital status profiles of the two groups differed, with more CP mothers reporting being married and less being separated/divorced compared with HC mothers ($x^2 = 24.04$, $p < .001$). The CP group had significantly higher pain scores ($t(89) = -5.66$, $p < .001$) and depression scores ($t(114) = -5.51$, $p < .001$) than the HC group.

For each group, paired samples *t*-tests were used to compare the two dimensions of "care" and "control" between mothers and fathers. Mothers were perceived by adolescents with CP to be higher on "care" than fathers ($t(53) = -5.43$, $p < .001$); for the "control" dimension, there was no significant difference between mothers and fathers in the CP group ($t(53) = -.47$, $p = .63$). For the control group, mothers were perceived to be higher on "care" than fathers ($t(57) = -3.83$, $p < .001$), and fathers were perceived to be lower on "control" than mothers ($t(57) = -1.96$, $p < .05$). ANOVAs controlling for sociodemographic group differences (adolescent race and sex) revealed mothers in the CP group had significantly higher care than mothers in the HC group ($F = 3.96$, $p = .049$). No such differences were evident for maternal control ($F = 2.41$, $p = .12$), paternal care ($F = 1.05$, $p = .31$), or paternal control ($F = 0.11$, $p = .75$).

Hypothesis Testing

Relationships Between Parental Bonding and Adolescent Pain

Given the significant differences between the groups on child race and sex, and mothers' marital status, partial correlations were performed controlling for child race and sex (race was first transformed into a dummy variable) and mother marital status (transformed into a dummy variable with married/cohabiting vs. not married or cohabiting). Table II shows partial correlations among the dimensions of "care" and "control," adolescent depression, and adolescent pain over the past month in the HC and CP groups. In the CP group, there were significant correlations between maternal control and pain and between paternal control and pain. Maternal care was significantly negatively related to adolescent pain, such that higher care was related to lower pain, while paternal care was not related to adolescent pain. In the CP group, maternal and paternal controls were both significantly related to adolescent depression. Maternal and paternal care were also significantly related to adolescent depression. In addition, adolescent depression was significantly associated with adolescent pain $r(53) = .36$, $p < .01$. Thus, conditions were met for

Table I. *Demographic Profile of the Healthy Control (HC) and Chronic Pain (CP) Groups*

Variables (*M* and *SD* is included)		HCs ($n = 61$)	Adolescents with CP ($n = 55$)
Age		14.8 (1.77)	15.0 (1.77)
Sex (% females)		47.5	69.1
Mother age		44.6 (6.95)	45.8 (5.94)
Mother's marital status	Married	28	48
	Divorce/separated	15	4
	Single (living alone)	16	2
	Living with a companion	3	1
PBI-BC dimensions (range −4 to 4)	Mother care	2.11 (1.87)	2.69 (1.67)
	Mother control	−0.51 (2.06)	−1.00 (2.06)
	Father care	0.60 (2.43)	1.00 (2.47)
	Father control	−1.26 (1.88)	−1.09 (2.10)
P (Range 1–10)		3.26 (3.18)	7.56 (2.37)
RCADS Depression scores (Range 1–18)		5.4 (4.1)	10.8 (6.3)
Pain that bothers the most (% of pain group)	Head		27.3
	Neck		5.5
	Upper extremity (shoulder, arm, elbow, hand)		5.5
	Chest		1.8
	Abdomen		20.0
	Lower extremity (hip, leg, knee, ankle, foot)		21.8
	Back		14.5
	None (i.e., no pain during the past month)		3.6

Note. PBI-BC = Parental Bonding Instrument—Brief Current version; RCADS = Revised Children's Anxiety and Depression Scale; fathers were not required to participate in the laboratory study so their age is only available for the limited subset who did participate and is not reported in this study; four participants (3 HC, 1 CP) did not have any interaction with their fathers and so did not complete the PBI-BC father subsection.

Table II: *Partial Correlations (Controlling for Child Race and Sex) Among Maternal and Paternal Bonding Dimensions of Care and Control, Bodily Pain, and Depression in HC and CP Groups*

Group		Maternal		Paternal	
		Care	*Control*	*Care*	*Control*
CP $N = 55$	Pain	−.29*	.38**	−.19	.28*
	Depression	−.35**	.61**	−.38**	.41**
HC $N = 61$	Pain	.19	−.10	−.02	−.03
	Depression	−.09	.03	−.23	.04

Note. CP = chronic pain group; HC = healthy control group.
*$p < .05$.
**$p < .01$.

further mediation testing for the independent variables of maternal control, maternal care, and paternal control.

In contrast, partial correlations revealed there were no statistically significant associations between parental bonding and adolescent pain or depression in the control group (nor was adolescent depression related to pain); conditions were thus not met for further testing in the HC group.

Associations Between Maternal Versus Paternal Bond and Adolescent Pain

As shown in Table II, and described above, maternal care and control were significantly related to adolescent pain ratings and depression in the group with CP. Similar findings emerged for paternal bonding, except that paternal care was not associated with adolescent pain. To understand potential differences in maternal versus paternal influences on adolescent pain and depression, significance of the difference between two correlation coefficients was performed using the Fisher r-to-z transformation. There were no significant differences between the strength of the maternal care–pain and paternal care–pain associations ($z = -.51$, $p = .61$), or between maternal care–depression and paternal care–depression associations ($z = .17$, $p = .86$). Similarly, there were no significant differences between the strength of the maternal control–pain and paternal control–pain associations ($z = -.48$, $p = .63$), or between maternal control–depression and paternal control–depression associations ($z = 1.41$, $p = .16$). These findings suggest that for adolescents with CP, maternal and paternal bonding indices are similarly influential in adolescent pain and depression.

Adolescent Depression as a Mediator of the Relationship Between Parental Bonding and Adolescent Pain

Bootstrapping analyses using PROCESS were conducted with adolescent depression as a hypothesized mediator between parental bonding and adolescent pain ratings for the group with CP only, as correlations among the variables were not significant in the

control group. Figures 1–3 display the findings for maternal care, maternal control, and paternal control (given the lack of a significant correlation between paternal care and adolescent pain, paternal care was not examined). Statistically significant paths are shown.

For maternal care, the indirect effect was −0.11, with 95% confidence intervals = −0.30 to −0.03, indicating that increased maternal care was related to lower adolescent pain in part through decreased adolescent depression. The ratio of indirect to total effect was 0.34, indicating that adolescent depression accounted for approximately 34% of the variance in the relationship between maternal care and adolescent depression, although because the direct effect was still significant, there was evidence for a partial mediation model.

For maternal control, a direct effect between maternal control and adolescent pain was revealed (0.27, CI = 0.11 to 0.43), and there was support for the relationship between maternal control and adolescent depression (1.84, CI = 1.15 to 2.53); however, there was no evidence for the mediating role of adolescent depression in this relationship (0.09, CI = −0.02, 0.29).

For paternal control, there was no evidence for a direct effect of paternal control on pain (0.14, CI = −0.04, 0.32). However, there was evidence for an indirect effect of paternal control on pain (0.11 with 95% confidence intervals = 0.04 to 0.25), indicating that increased paternal control was related to increased adolescent pain through increased adolescent depression. The ratio of indirect to total effect demonstrated that adolescent depression accounted for approximately 44% of the variance in the paternal control–adolescent pain relationship.

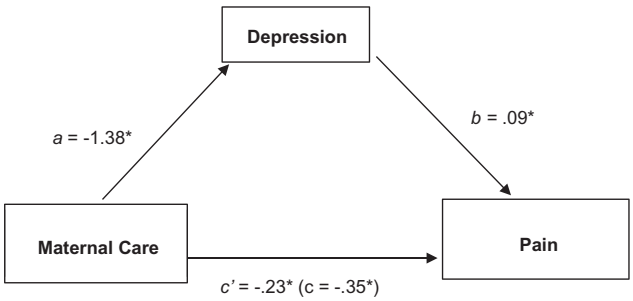

Figure 1. Adolescent depression as a mediator of the relationship between maternal care and adolescent pain.

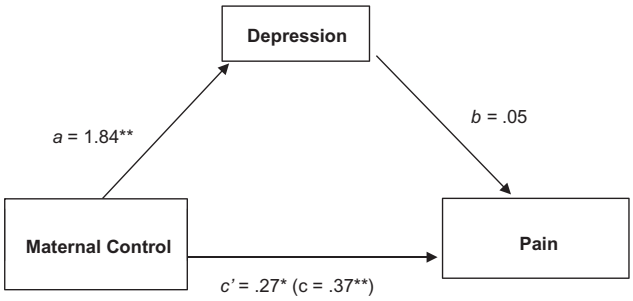

Figure 2. Adolescent depression as a mediator of the relationship between maternal control and adolescent pain.

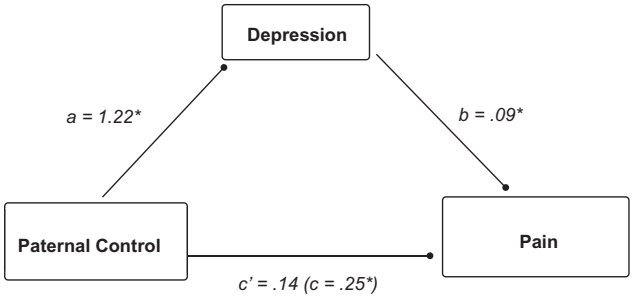

Figure 3. Adolescent depression as a mediator of the relationship between paternal control and adolescent pain.

Discussion

This study is the first report that we are aware of that has tested the mediating role of depression in the relationship between parental bond and pain in adolescents with CP, including the first use of a child self-report parental bonding measure to gain insight into current perceptions of the parent–child bond, and the first direct comparison of maternal and paternal bond in pediatric pain. Our data supported the first hypothesis that unhealthy parental bonding (maternal and paternal low levels of care and high levels of control) is related to increased adolescent pain and depression; however, these relationships did not exist in the health control group. Almost all relationships among parent bonding and adolescent pain and depression were significant in the CP group (with the exception of paternal care–adolescent pain), indicating that reduced parental care and increased parental control are related to worse outcomes in adolescents with CP, but no such relationships were evident in the control group. These findings suggest the particular importance of parental processes in adolescent pain and mental health when a child has CP. However, given that the marital profiles of the two groups of mothers significantly differed, we must be cautious in our interpretations regarding the control group. Despite attempting to control for marital status differences between the two groups of parents, it is possible that we did not find significant parent bond–child depression and pain associations because of greater disruptions in attachment owing to parental separation in the control group. Alternatively, we may not have detected relationships between parent bond and adolescent pain and depression in the control group owing to range restriction, such that the low pain and depression scores in the HC may have reduced correlations.

Regarding the second hypothesis, exploring potential differences between maternal and paternal bond, there did not appear to be any differences in the strength of maternal bond and paternal bond associations, suggesting that maternal and paternal bonds are both important in understanding adolescent depression and pain in the context of pediatric CP. Third, there was support for the mediating role of adolescent depression in the relationship between parental bonding and adolescent pain in the CP group, with depression emerging as a significant mediator of the relationship between maternal care and adolescent pain, and between paternal control and adolescent pain. It is possible that we did not find a significant meditation model for maternal control owing to low power to detect an effect.

Prior retrospective research with adult samples has demonstrated that high parental "control" and low parental "care" are associated with pain in adult patients with CP (Anno et al., 2015; Avagianou et al., 2010). Our findings add to this literature by extending the findings to adolescent patients. Analyses comparing care and control scores between groups suggest that the parental bonding–pain associations seen in the CP group are not necessarily a result of more dysfunctional parenting by the CP group parents compared with HC parents. There were no significant differences between the groups in maternal or paternal control, or paternal care. The only significant difference was in maternal care, with CP adolescents reporting more care in their mothers than HC adolescents. This finding is consistent with the wider literature showing that mothers of children with CP are heavily engaged in providing care for their children (Palermo & Eccleston, 2009). The care subscale of the PBI used here assesses the warmth of such interactions, and it appears that adolescents with CP perceive high level of such warmth. The fact that high maternal care in the CP group was also associated with decreased adolescent depression and pain suggests that it is a protective factor and we must look elsewhere for an explanation for why a parental bond–pain relationship exits in the CP group but not in the HC group. One possibility is that perhaps adolescents with CP are particularly sensitive to what they may perceive as intrusion, lack of autonomy granting or control from their parents, and even normative levels of parental control are associated with child depression and increased pain. Previous research has identified that certain individuals, including those with sensory processing sensitivity, may be particularly sensitive to parental bonding behaviors, including low care in their mothers, compared with individuals without sensitivity (Liss, Timmel, Baxley, & Killingsworth, 2005). Another, related explanation is that there were more girls in the CP than HC group, and girls may be more sensitive to the effects of parental control than boys. However, all paths remained significant even when controlling for child sex. Future studies should explore the possible sensitivity of children with CP to normative parenting behaviors.

Wider findings regarding the relative strength in relationships between maternal versus paternal bond and pain or depression outcomes in children are mixed. For example, maternal care and control have been implicated in adult offspring pain (Avagianou et al., 2010; Lung et al., 2004), while another adult retrospective study found that compared with maternal bond, paternal bond was more strongly related to pain (Anno et al., 2015). Here, we found no differences in the strength of maternal versus paternal correlations. Perhaps the disparate findings may be related to cultural differences, as the Anno study was conducted in Japan, which has distinct cultural parenting norms, where fathers tend to have a more authoritarian role within the family than fathers in Western countries (Uji, Sakamoto, Adachi, & Kitamura, 2012). Given that mothers and fathers are often highly involved in the day-to-day care of children in Western countries, it is perhaps not surprising that both maternal and paternal bond were similarly associated with adolescent pain and depression in the present study.

Our findings regarding the mediating role of adolescent depression align with the adult literature that has found that maternal care and control are positively related to bodily pain and depression (Avagianou et al., 2010). In addition to replicating these associations in a pediatric sample, our study also provides novel data about the role of adolescent depression as a process variable in the relationship between parent bond and pain, thus highlighting developments for possible therapeutic interventions. Overall, the findings indicate that in families dealing with adolescent CP, decreased maternal care and increased paternal control are associated with greater adolescent depression, which in turn is associated with greater adolescent pain. Although our cross-sectional data preclude causal statements, this scenario is supported by links made in previous research. Low parental care has been associated with a range of mental health issues including adolescents' difficulties coping with stress (Kraaij et al., 2003) and later depression in adulthood (Parker, 1983; Sato et al., 1998). In turn, depression is a known risk factor for CP (Mallen, Peat, Thomas, Dunn, & Croft, 2007). The present findings confirm these associations within the one study, and point to the existence of this process early in the individual's life.

Despite the promising implications of this study, a number of limitations were present. The design is cross-sectional and therefore relationships between the variables cannot be interpreted as causal in nature

and no definite conclusions can be made regarding whether depression places individuals at greater risk for developing CP, or if the long-term experience of CP leads to the development of depression. Similarly, it is not known whether parental bonding behaviors lead to heightened adolescent pain, or whether the adolescent's pain contributes to controlling parenting by fathers and less care behavior in mothers. Longitudinal studies can more clearly ascertain these causal relationships. In addition, while there are advantages to self-report measures, the possibility exists that current mood states may have impacted responses and there is a need to replicate these findings with additional measures of bonding. Wider assessments using observations or parent-reported bond are required to verify and provide context to the findings for child-reported bond. An additional consideration for future research is the inclusion of measures of parental distress, as it is known that parental depression impacts bonding. While emphasizing the need to further explore the roles of both fathers and mothers in adolescent CP, in the present study we were unable to control for time spent with each parent, and it is possible that the findings were impacted by children with CP in this study spending a disproportionate amount of time with mothers, owing to the nature of their condition as well as the fact that control mothers were less likely to be living with the child's father. Overall, there is greater need to ensure groups are matched on important sociodemographic variables, including family income, race/ethnicity, marital status of parents, and time spent with each parent. Future studies should examine the role of time spent with fathers versus mothers and how opportunities to be exposed to parental behaviors may impact findings.

Conclusions

Understanding the psychosocial factors involved in pediatric CP is vital to ensure that appropriate and effective treatments are developed. This study highlights the importance of considering parental bonding behaviors and adolescent depression in pediatric CP. In particular, high paternal control and low maternal care appear to contribute to increased pain in adolescents partly through heightened adolescent depressive symptomology. Given that adolescent depression did not fully mediate the relationship, and that there was only evidence for an indirect effect of paternal control on adolescent pain through adolescent depression, treatment strategies would be best served to take a multipronged approach that includes parents as well as children to address pediatric pain. It is possible that coaching families regarding healthy levels of care and control, combined with targeting adolescent depressive symptoms, would help to decrease adolescent pain. The particular role of depression as a mediator of the parent bond–adolescent pain association highlights the importance of frequent screening and treatment of depressive symptoms in this group.

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