## **CONCISE REPORT**

# Parental pain is not associated with pain in the child: a population based study

G T Jones, A J Silman, G J Macfarlane

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**Background:** Child pain is associated with adverse psychosocial factors. Some studies have shown an association between children's and parental pain. Children may "learn" pain behaviour from their parents.

**Objectives:** To examine whether an association exists between parent and child pain, and, if so, whether this relationship persists after adjusting for psychosocial difficulties in the child.

**Methods:** 1326 schoolchildren took part in a questionnaire based, cross sectional survey. Parents of study participants were sent a postal questionnaire. Occurrence of body pain was ascertained using blank body manikins and, in children, psychosocial factors were assessed using the Strengths and Difficulties Questionnaire. Three child-parent pain relationships were examined: any child pain with any parental pain or with parental widespread pain; and child low back pain with parental low back pain.

**Results:** The risk of child pain associated with parental reporting of pain was minor, and non-significant. Even when both parents reported widespread pain, the relative risk of pain in the child, after adjusting for age and psychosocial difficulties, was 1.2 (95% CI 0.5 to 3.2).

**Conclusions:** Parental pain is not a risk for child pain. Pain behaviour is not learned. Rather, child pain is probably attributable to individual factors and the social environment.

hildhood pain is associated with a number of adverse psychosocial factors. Studies have shown an association between pain in childhood and emotional and conduct problems,¹ a high negative affect score,² and social factors such as loneliness and having difficulty making friends;³ chronic adolescent pain has been shown to be associated with neurotic symptoms and the negative fear of failure.⁴ Further, we have demonstrated that emotional and behavioural problems are not only associated with pain contemporaneously but are also predictive of the onset of childhood pain syndromes.⁵ 6

A number of authors have shown that children whose parents report pain are more likely to report pain than other children, 7-10 and others have demonstrated that for children with chronic conditions, those with parents with a history of chronic pain report higher global impairment and physical disability than other children. 11 Other studies, however, have shown no association between parental pain and child pain. 4 12 13

Although the precise relationship is unclear, the potential association between parental and child pain, in light of recent evidence about the importance of psychosocial factors in the onset of child pain, has led to hypotheses of learned pain behaviour. However, any such association may be due to genetic or shared environment such as shared psychosocial

factors. To our knowledge, there have been no large scale community surveys investigating the parent-child pain relationship in the context of other psychosocial factors. Thus, the aim of the current study was twofold:

- To examine whether children are more likely to report pain if their parents also report pain
- To ascertain whether this relationship, if present, can be explained by adjusting for levels of psychosocial difficulties in the child.

This study examines these aims for any musculoskeletal pain, widespread pain (the "extreme" musculoskeletal pain syndrome), and low back pain (the most common regional pain syndrome).

#### **METHODS**

One thousand five hundred and five children aged 12–15 years, from 39 schools in the north west of England, were asked to take part in the study. The study area includes both urban and rural areas and consists of a population of approximately 1.1 million, with a mix of sociodemographic and economic characteristics. All schools with eligible children within this area were approached and in each participating school between one and three classes were selected by the head teacher for participation in the study. Permission to approach schools was obtained from the appropriate regional directors of education, and parental consent was obtained before the children's participation in the study. Ethical approval for the study was granted by the University of Manchester committee on the ethics of research on human beings.

Information is collected by self completion questionnaires that were completed in the classroom with the researchers present. Subjects were asked: "Thinking back over the past month, have you had any pain which has lasted for 1 day or longer?". Those answering positively were directed to two blank body manikins (anterior and posterior) on which they were asked to indicate the sites of pain. Adverse psychosocial factors were assessed using the Strengths and Difficulties Questionnaire, "4 a well validated instrument that produces a score of "total difficulties", comprising emotional, conduct, and other behavioural problems.

After participation in the study, two postal questionnaires were sent to the home address of each child, one for completion by each of the child's parents or guardians. "Parental" data collection was from the main male and female adults in the child's household. Thus, although not necessarily the genetic parents of the child, these adults would probably have the most influential effect on learned pain behaviour, if such learning exists. Follow up reminder questionnaires were sent to non-responders after 3 weeks,

Abbreviations: AP, any pain; LBP, low back pain; RR, relative risk; WP, widespread pain

Table 1	Relationship	between pain	in the parent	t and pain in th	e child
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	Child pain			RR* with adverse psychosocial behaviour		
	Yes	Total	RR*	Low	High	Adjusted
Relationship: Parental AP w	rith child AP					
Both parents						
Pain free	125	253	1.0	1.0	1.0	1.0
Any pain	88	147	1.2 (0.9 to 1.6)	1.2 (0.8 to 1.7)	1.2 (0.8 to 1.9)	1.2 (0.9 to 1.6)
Any parent						
Pain free	162	325	1.0	1.0	1.0	1.0
Any pain	321	566	1.1 (0.9 to 1.4)	1.1 (0.8 to 1.4)	1.1 (0.9 to 1.5)	1.1 (0.9 to 1.3)
Mothers						
Pain free	250	506	1.0	1.0	1.0	1.0
Any pain	256	427	1.2 (1.001 to 1.4)	1.1 (0.9 to 1.5)	1.2 (0.9 to 1.5)	1.2 (0.97 to 1.4)
Fathers						
Pain free	222	430	1.0	1.0	1.0	1.0
Any pain	153	286	1.0 (0.8 to 1.3)	1.0 (0.8 to 1.6)	1.0 (0.8 to 1.4)	1.0 (0.8 to 1.3)
Relationship: Parental WP v	with child AP					
Both parents						
Pain free	320	603	1.0	1.0	1.0	1.0
Widespread pain	4	6	1.3 (0.5 to 3.4)	1.5 (0.4 to 5.9)	1.0 (0.3 to 4.2)	1.2 (0.5 to 3.2)
Any parent	-	-	(,	( /	(	(5.5 .5 5)
Pain free	396	732	1.0	1.0	1.0	1.0
Widespread pain	43	86	0.9 (0.7 to 1.3)	0.9 (0.6 to 1.5)	0.8 (0.5 to 1.3)	0.9 (0.6 to 1.2)
Mothers				(	( /	()
Pain free	472	870	1.0	1.0	1.0	1.0
Widespread pain	34	63	1.0 (0.7 to 1.4)	1.1 (0.7 to 1.9)	0.8 (0.5 to 1.4)	0.9 (0.7 to 1.4)
Fathers	•		(6.5)	(0.7 .0)	0.0 (0.0 10 1.1)	0.7 (0.7 10 11-1)
Pain free	362	687	1.0	1.0	1.0	1.0
Widespread pain	13	29	0.9 (0.5 to 1.5)	0.7 (0.3 to 1.8)	0.9 (0.4 to 1.8)	0.8 (0.5 to 1.4)
Relationship: Parental LBP v	with child IRP					
Both parents	VIIII CIIIIG EDI					
Pain free	78	361	1.0	1.0	1.0	1.0
Low back pain	21	70	1.4 (0.9 to 2.3)	1.5 (0.8 to 2.9)	1.3 (0.6 to 2.8)	1.4 (0.9 to 2.3)
Any parent	۷.	, 0	1.4 (0.7 10 2.0)	1.5 (0.0 10 2.7)	1.0 (0.0 10 2.0)	1.4 (0.7 10 2.3)
Pain free	103	452	1.0	1.0	1.0	1.0
Low back pain	111	416	1.2 (0.9 to 1.6)	1.1 (0.7 to 1.6)	1.2 (0.8 to 1.8)	1.1 (0.9 to 1.5)
Mothers	111	410	1.2 (0.7 10 1.0)	1.1 (0.7 10 1.0)	1.2 (0.0 10 1.0)	1.1 (0.7 10 1.3)
Pain free	152	644	1.0	1.0	1.0	1.0
Low back pain	85	289	1.2 (0.95 to 1.6)	1.2 (1.8 to 1.8)	1.2 (0.8 to 1.7)	1.2 (0.9 to 1.6)
Fathers	00	207	1.2 (0.75 10 1.0)	1.2 (1.0 10 1.0)	1.2 (0.0 10 1.7)	1.2 (0.7 10 1.0)
Pain free	114	519	1.0	1.0	1.0	1.0
Low back pain	47	197	1.1 (0.8 to 1.6)	1.2 (0.7 to 2.0)	1.0 (0.6 to 1.6)	1.1 (0.8 to 1.6)

and again after a further 2 weeks, and non-response at the end of this period was assumed to be refusal to participate. Parents were also asked whether they had had pain over the past month which had lasted for 1 day or longer, as in the children's questionnaire.

#### Analysis

We evaluated the influence of parental pain in predicting child pain. Three parent-child pain relationships were examined:

- The relationship between the reporting of "any" pain (AP) in the parent and AP in the child: to examine the basic premise of an association between pain reporting in parents and children
- The relationship between the reporting of widespread pain (WP) in the parent and AP in the child: to examine the prior hypothesis of a possible dose-risk relationship, where widespread pain in the parent may constitute a greater "exposure"
- The relationship between the reporting of parental low back pain (LBP) and LBP in the child: to examine the relationship for a specific regional pain syndrome.

The association between exposure and outcome was calculated using a Poisson regression model, and the results are expressed as relative risks (RR) with 95% confidence intervals (95% CI), adjusted for the child's age. Statistical

analysis was carried out using Stata (version 7) (Stata Corporation, College Station, Texas).

#### **RESULTS**

One thousand three hundred and twenty six children participated in the study (participation rate 88%). The median age of participants was 13.9 years (interquartile range (IQR) 13.3–14.5) and 611 (46.1%) were male. Information was available from both parents of 681 (51.4%) children and from only one parent of 322 (24.3%) children (in 104 (32.3%) of these latter cases it was confirmed that the family only had a single parent). Only 323 children (24.4%) had data from neither parent. Median time between child and parental questionnaire completion was 17 days (IQR 10–35).

Of 1293 children, 713 (55.1%) children reported pain lasting 1 day or longer in the month before the survey, of whom 329 reported LBP (25.4% of the total sample). In mothers, the 1 month period prevalence of AP, LBP, and WP was 45.8%, 30.9%, and 6.7%, respectively, and in fathers 39.8%, 27.2%, and 4.0%.

Children with high total psychosocial difficulties—that is, children who reported high levels of adverse behaviour—were more likely to report pain than their peers (RR = 1.8; 95% CI 1.5 to 2.3), after adjusting for child age and sex. These children also had a significant increased risk of LBP (RR = 2.4; 95% CI 1.8 to 3.3).

1154 Jones, Silman, Macfarlane

Of children whose parents both reported AP, 59.9% also reported AP, compared with 49.4% of children whose parents were both pain free ( $\chi^2 = 4.08$ ; p<0.05). However, when these results were expressed as an age adjusted risk, the magnitude of the effect was neither large, nor significant. Further, the effect of high psychosocial difficulties had no impact on the relationship between parent and child pain. This (lack of) association was found between all AP-AP relationships examined (table 1), and no significant differences were found between maternal and paternal pain.

Table 1 also shows that when examining the parent-child WP-AP and LBP-LBP relationships, there were no consistent patterns to suggest that parental reporting of pain was associated with an increased risk of child pain. Further, no differences were found between children reporting high versus low levels of psychosocial difficulties. In summary, across the range of relationships examined in the current study, there is no evidence to suggest that the reporting of pain in the parent(s) influences the reporting of pain in the child.

#### **DISCUSSION**

This study demonstrates that there are no consistent associations between pain in the child and pain in the parent. Methodologically, there are several issues to consider. Although we purport to measure contemporaneous pain reporting in parents and children, this was not actually the case. Owing to the nature of data collection there was a delay in the collection of the parental data: median delay 17 days (IQR 10-35). However, studies have shown that 83% of people with WP satisfy the International Association for the Study of Pain definition of chronic pain—that is, pain persisting for at least 3 months,15 and it is likely, therefore, that many of the parents reporting WP would have had had symptoms for some time. Further, it is unlikely that any association between parent-child pain comes about through short (minor) episodes of adult pain. Although we have been unable to look specifically at chronic disabling pain in the parents, our analysis shows that there was no relationship between WP in the parent and pain in the child.

Secondly, the sample size is a problem. Although we studied a relatively large number of children, the need for responses from a parent-child pair or triplet led to small numbers, particularly in analysis relating to WP and this might have reduced the likelihood of observing a parent-child association, if one does exist. However, our study provides >90% power to detect a 50% increase in risk of child AP, associated with parental chronic WP. Thus, our inability to demonstrate a significant association between parent and child pain is not due to the small sample size.

Thirdly, we measured behavioural and emotional factors in children. Had we demonstrated any parent-child pain relationships it might have been more pertinent to measure aspects of family social function to identify subgroups of parents-children who exhibited a pain association. However, the fact that we did not detect an association overall suggests that we probably would not have found one in a subgroup.

Previous studies report conflicting results for the presence (or absence) of a parent-child pain relationship.7-10 12 13 Our study, after rigorous examination of several relationships, shows that such an association does not exist. Previous work from this cohort has shown the importance of psychosocial factors as both a risk marker for,1 and predictor of,5 of musculoskeletal pain symptoms. This study adds to these studies by demonstrating that these factors are in no way influenced by the occurrence of parental pain.

In summary, the results of this study provide no evidence to support the hypothesis of learned pain behaviour in children. Instead, there is growing evidence that the cause of bodily pain in children is related primarily to individual constitutional factors, psychological status, and their social environment.

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### Authors' affiliations

G T Jones, A J Silman, Arthritis Research Campaign Epidemiology Unit, School of Epidemiology and Health Sciences, University of Manchester, UK

G J Macfarlane, Unit of Chronic Disease Epidemiology, School of Epidemiology and Health Sciences, University of Manchester, UK

Correspondence to: Dr G Jones, ARC Epidemiology Unit, School of Epidemiology and Health Sciences, University of Manchester Medical School, Oxford Road, Manchester, M13 9PT, UK; gareth.jones@ man ac uk

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#### **REFERENCES**

- 1 Watson KD, Papageorgiou AC, Jones GT, Taylor S, Symmons DP, Silman AJ, et al. Low back pain in schoolchildren: the role of mechanical and psychosocial factors. Arch Dis Child 2003;88:12-17.
- 2 Balague F, Skovron ML, Nordin M, Dutoit G, Pol LR, Waldburger M. Low back pain in schoolchildren. A study of familial and psychosocial factors. Spine 1995:**20**:1265–70.
- 3 Brattberg G. The incidence of back pain and headache among Swedish school children. Qual Life Res 1994;3(suppl 1):S27–31.
- 4 Merlijn VPBM, Hunfeld JAM, van der Wouden JC, Haze-broek-Kampschreur AAJM, Koes BW, Passchier J. Psychosocial factors associated
- with chronic pain in adolescents. Pain 2003;101:33–43.

  5 Jones GT, Watson KD, Silman AJ, Symmons DP, Macfarlane GJ. Predictors of low back pain in British schoolchildren: a population-based prospective cohort study. Pediatrics 2003;111:822-8.
- 6 Jones GT, Silman AJ, Macfarlane GJ. Widespread body pain: predicting its onset amongst children. Arthritis and Rheumatism 2003;48:2615–21.
- Szpalski M, Gunzburg R, Balague F, Nordin M, Melot C. A 2-year prospective longitudinal study of low back pain in primary school children. *Eur Spine J* 2002;11:459-64
- 8 Balague F, Nordin M, Skovron ML, Dutoit G, Yee A, Waldburger M. Non-specific low-back pain among schoolchildren: a field survey with analysis of some associated factors. J Spinal Disord 1994;7:374–9.
- 9 Mikail SF, von Baeyet CL. Pain, somatic focus, and emotional adjustment in children of chronic headache sufferers and controls. Soc Sci Med 1990:31:51-9
- Schanberg LE, Anthony KK, Gil KM, Lefebvre JC, Kredich DW, Macharoni LM. Family pain history predicts child health status in children with chronic rheumatic disease. *Pediatrics* 2001;**108**:E47–53.
- Schanberg LE, Keefe FJ, Lefebvre JC, Kredich DW, Gil KM. Social context of pain in children with juvenile primary fibromyalgia syndrome: parental pain history and family environment. Clin J Pain 1998;14:107-15.
- Kovacs FM, Gestoso M, Gil del Real MT, Lopez J, Mufraggi N, Mendez JI. Risk factors for non-specific low back pain in schoolchildren and parents: a population based study. *Pain* 2003;103:259–68.
- Borge AlH, Nordhagen R. Recurrent pain symptoms in children and parents. Acta Pædiatr 2000;89:1479–83.

  Goodman R. The Strengths and Difficulties Questionnaire: a research note.
- J Child Psychol Psychiatry 1997;38:581–3.

  15 Macfarlane GJ, McBeth J, Silman AJ. Widespread body pain and mortality:
- prospective population based study. BMJ 2001;323:662-5.