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INTRODUCTION

Low back pain (LBP) is the leading cause of disability worldwide [1]. Although only 10% of people who experience LBP become disabled, this proportion of patients consumes the vast majority of LBP health resources [2-4]. The causes of chronic disabling low back pain (CDLBP) are thought to be multifactorial [5] and thus may need to be considered within a multidimensional framework for both adults [6,8] and adolescents [9]. Many of the contributing factors to LBP have been shown to display familial associations, reflecting genetic or shared environmental factors [10-12]. Specifically: spinal structures such as degenerated discs [10] and bone loss [13, 14]; pain sensitivity and development of chronic pain [15, 16]; psychological factors such as depression and anxiety [17], pain catastrophizing [18], distress [19], pain behaviours and coping strategies [20, 21]; lifestyle factors [22, 23], body mass index (BMI) [22, 23] and physical activity levels [24, 25] as well as lumbar range of motion [26] and back muscle endurance [27]. Recently, a familial association has been reported for spinal posture [28] in people with CDLBP. Specifically, hyperlordotic lumbar postures in standing have been shown to be more common in daughters of parents with such postures [28].

Systematic reviews suggest there is no evidence for a causal relationship between CDLBP and different spinal postures in prolonged sitting [29], standing [30, 31] and squatting [32]. A potential reason is a “wash out” effect that occurs when people with different types of CDLBP are analysed homogenously [33]. However, once subgrouped based on pain provocative habitual spinal postures and movement patterns, people with CDLBP can be differentiated from healthy controls [33-35]. Smith et al. (2008) [36] demonstrated that adolescents subgrouped into non-neutral standing postures, had an increased risk for LBP. Similarly, Dolphens et al. (2013) [37] demonstrated that once adolescent boys were subgrouped based on global and lumbopelvic alignment in standing , those with a sway-back posture were almost twice more likely to report LBP compared to those with neutral alignment.

When considering the association between movement and CDLBP, without subgrouping, literature suggests that no clear relationship exists [6, 38]. A few authors have investigated CDLBP subgroups defined by movement [39-42], however, only one approach acknowledges the complex multidimensional nature of CDLBP [6, 43]. Directional patterns of postures and movements associated with LBP outlined by O'Sullivan (2004) [44] form part of the physical component of this multidimensional classification system [45]. Using a combination of subjective information related to aggravating and easing factors, and observation of patient postures and functional movements, this approach has been shown to be reliable and valid [34, 43, 46]. Inter-tester reliability was found to be almost perfect between expert clinicians (k = 0.96, percentage-agreement 97%) and acceptable between postgraduate clinicians (k = 0.61, range 0.47 – 0.80, percentage agreement 70%, range 60 – 84%) [46]. Dankaerts et al. (2009) [34] subsequently demonstrated this classification system was able to discriminate between two subgroups (active extension, flexion) and healthy controls, both clinically and via trunk electromyography and kinematic analysis. A consistent pattern for both posture and movement was found in subjects with CDLBP reporting direction-specific aggravating and easing postures and movements, providing further empirical evidence of the validity of the movement pattern-derived subgroups [34].

The same movement patterns seen in adults [6, 34, 46] have been demonstrated in children [47] and adolescents [48] when subgrouped based on similar methodology. The underlying basis for different movement patterns in people with CDLBP is likely to be complex and multifactorial. Different hypotheses have been suggested, including the potential of a familial link [49]. Although a familial link has been found between parent-daughter dyads for certain standing postures, to date there has been no investigation of familial relationships in subgroups with distinct postural and movement patterns [28]. Therefore, the aim of the study was to perform a preliminary exploration of familial associations of two movement pattern-derived subgroups. This was undertaken within and between members of families with CDLBP.

MATERIALS AND METHODS

Study design

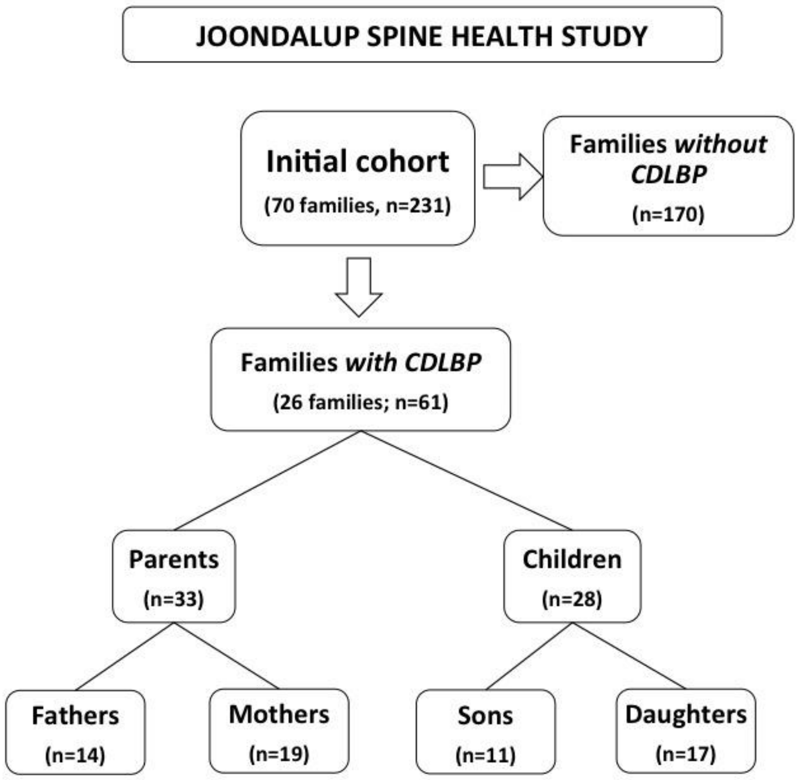
Descriptive study based on data collected in the Joondalup Spinal Health Study (JSHS) [50], a cross-sectional community-based cohort study, conducted between August 2008-May 2009. The JSHS was designed to investigate familial associations in spinal health. The current analysis investigated the familial association of movement pattern-derived subgroups in families with CDLBP.

Study population

Participants in this study represent a subset of the JSHS cohort. Originally, the JSHS recruited 231 participants (70 families consisting of 109 biological parents, 1 non-biological parent and 121 children) within an approximate 10km radius of the study centre in Joondalup, a middle band socio-economic suburb of Perth, Western Australia, with a population of 16,000. To minimise selection bias, potential participants were contacted through random dialling of residential phone numbers based on the Perth electronic telephone directory. Screening for potential eligibility was conducted by operators using a computer-assisted telephone interview [50]. For the purposes of the JSHS, "children" were defined as individuals who lived in the same residence as their parents/guardians and aged between 10-25 years. "Parents" were defined as biological or non-biological parents/guardians, aged up to 65 years. Families with and without LBP were purposely recruited into JSHS. The "pain" families were recruited based on at least one parent and one child in the same family reporting LBP. The complete, original recruitment and inclusion criteria have been described elsewhere [50]. All participants provided written informed consent prior to their participation and ethical approval to conduct this study was granted by institutional Human Research Ethics Committees.

In the current study, chronic LBP was defined by meeting either duration or number of episodes criteria. Specifically, a duration of greater than three months (either continuously or intermittently) such that pain was experienced at least once per week, or more than one episode of LBP over the past year. Disabling LBP was defined as pain impacting on at least three of the following areas: lifting, standing, sitting, sleeping, social interaction, travel, need to take medication or need to see a health professional [50]. Families were excluded from the current study if at least one parent and one child did not experience CDLBP as described above. Data from the one non-biological parent were excluded due to an absence of genetic links with her child. Twenty-six families were included in this study. The distribution of members varied across families, specifically: 7 families with 7 fathers and 7 children; 12 families with 12 mothers and 13 children; 7 families with 7 fathers, 7 mothers and 8 children. Data from 33 parents (14 fathers and 19 mothers) and 28 children (11 sons and 17 daughters) with CDLBP was selected for this study (Fig. 1).

Figure 1 - Flow diagram of the sample selection [50]. Where, 'n' indicates the number of members in the families.



Outcome measures and procedure

Anthropometrics

Height and mass were measured using a stadiometer and an electronic scale respectively. Body mass index (BMI) was subsequently calculated.

Subjective assessment (Questionnaires)

Family members from the initial cohort completed questionnaires which were delivered online through a secure website [50]. LBP pain severity and impact of LBP for each subject was assessed using specific LBP-related items including the Oswestry Disability Questionnaire (ODQ) [51], pain intensity over the past week with the numeric-rating-scale [52] and yes/no questions on interference of LBP with common aggravating activities (sitting, standing, walking, bending, lifting). This information provided an understanding of the participant's LBP behaviour.

Postural and movement pattern assessment

At the time of data collection, participants were asked to wear bike shorts (and singlets for the females) allowing exposure of the lumbar spine, and video footage was taken from a single camera while subjects performed a series of postures and functional movements commonly reported to provoke LBP. These involved: usual posture in standing, forward trunk bending and return, backward trunk bending and return, single leg standing, picking up a stool, usual sitting posture, slump sitting posture, erect upright sitting posture, sit-to-stand to sit and holding a half squat for five seconds. This sequence was performed once, under instruction from a research officer. Images were recorded in the posterior and postero-lateral view [44, 53]. These tasks were based on those used in a study examining movement patterns in an adult population [46]. Previous studies have demonstrated that when these posture and movement patterns are correlated with the person's LBP behaviour, participants can be categorised into subgroups [33, 35, 43, 44].

Subgrouping process

Participants were categorised into one of three movement pattern-derived subgroups using a previously developed framework [44] with evidence for intra-tester reliability [43, 46] and validity [33, 49, 54]. The three subgroups derived from this process were: active extension pattern (AE), flexion pattern (F) and multidirectional pattern (MD) [55]. Definition of these patterns is reported in Table 1.

	Subgroups		
	Flexion Pattern	*Active Extension Pattern*	*Multidirectional Pattern*
Provocative postures and movements	Lumbar flexion related (eg., slump sitting, sustained half squatting, forward bending, lifting, sit to stand associated with a flexed lumbar spine , …)	Lumbar extension related (eg., sitting, standing, forward and backward bending associated with lumbar lordosis, …)	Multi directional related (both flexion and extension) (eg., flexed lumbar spine postures in sitting, +/- bending and extended lumbar spine posture in standing, walking; as well as mixed postures such as, flexed lumbar spine postures in sitting, and extended lumbar spine posture in lifting)
Easing postures and movements	Lumbar extension related	Lumbar flexion related	Neutral spinal posture
Observations	Provocative posture and movements associated with a flexed lumbar spine	Provocative posture and movements associated with lordotic lumbar spine	Provocative posture and movements associated with either flexed or extended lumbar spine

Table 1 Clinical analysis used for the subgrouping of participants in this study. Description of each subgroup; adapted from Astfalck et al. (2010) [35], Dankaerts et al. (2006) [33].

The differentiating factor between MD and AE is the lumbar spine posture in sitting, bending, squatting and lifting. The MD pattern is associated with both flexed and extended lumbar spine postures, and may be classified by a flexed lumbar spine posture in sitting, forward bending, squatting and lifting, whereas the AE pattern is associated with an extended lumbar spine in these positions. The standing posture, however, is similar to both MD and AE groups, associated with an extended spine posture. The differentiating factor between F and MD patterns is that the F group report pain associated with flexed lumbar spine postures in sitting, bending, squatting and lifting, whereas the MD group report pain associated with both flexed and extended lumbar spine postures. This MD pattern may, therefore, manifest as flexion postures associated with sitting, +/- bending and squatting as well as lumbar spine extension postures in standing, walking (single leg standing) +/- bending and squatting. Therefore, in situations where the person does not report pain in standing or walking, but does report pain associated with mixed postures in sitting, bending and lifting (e.g. flexed posture in sitting, and extended posture in lifting) the classification is considered as MD.

It is important to highlight the clinicians were not present during the filming of the tasks, and only had access to subjective data (questionnaires) and the video footage of the tasks. Rather than rating a participant’s performance on specific physical tests, decisions about subgroup categorisation were based on combining information of pain provocative and easing postures and activities (obtained from the ODQ [51]), with the clinician’s analysis of the postures and functional tasks observed on the video footage. Indeed, using a composite set of data more closely aligns with clinical practice, where integration of multiple subjective and objective parameters is undertaken to reach diagnostic and management decisions.

All participants were independently subgrouped by two postgraduate physiotherapists (CL, ES), with any discordance resolved by consensus with two specialist physiotherapists (JPC, POS). The postgraduate physiotherapists had received training in the classification system by JPC and POS, which involved the following steps: 1) all members of the group (CL, ES, JPC, POS) performed an independent analysis of randomly selected videos to categorise subjects into subgroups; 2) subgrouping results were compared between the four members of the group; 3) when discordance occurred, this was resolved by discussing the criteria used to subgroup the relevant subject and a consensus was reached.

Data analysis

Descriptive statistics were based on frequency distributions and medians, IQRs and ranges for categorical and continuous data respectively. Univariate analysis included χ^2 and Fisher exact tests for categorical comparisons, and Mann-Whitney *U* tests for continuous outcomes. Unweighted kappa coefficient was used to assess level of agreement between examiners' subgroups. Spearman's correlation coefficient (rho; *ρ*) was used to determine if correlations existed between familial dyads within movement pattern subgroups. Data were analysed using IBM SPSS version 22.0 (Armonk, NY). P-values <0.05 were considered statistically significant.

RESULTS

Participant characteristics

Table 2 details the characteristics of family members (14 fathers, 19 mothers, 11 sons and 17 daughters). Age and BMI for parents were similar, with the mean BMI for both mothers and fathers reaching the minimum for classification as 'overweight' [56]. Fathers had significantly more years since the first episode of LBP compared to mothers (p=0.019). No differences were observed between sons and daughters.

Inter-observer reliability in clinical subgrouping

Based on independent classification by two postgraduate clinicians, percentage of agreement of subgroups was 98%, *K* = 0.96.

Table 2. Participant baseline characteristics.

	Father n=14	Mother n=19	**Son** n=11	Daughter n=17
Age (median (IQR) years)	49.0 (7.0)	46.0 (7.0)	20.0 (7.0)	18.0 (5.0)
Age of onset of LBP (median (IQR) years)	20.0 (15.0)	30.0 (16.0)	15.0 (4.0)	13.0 (4.0)
Years since onset of LBP (median (IQR) years)	30.0 (14.0) [a]	15.0 (21.0)	4.0 (5.0)	3.0 (3.0)
BMI (median (IQR) kg/m2)	29.1 (4.9)	26.6 (7.1)	23.1 (5.3)	22.9 (4.5)
Episodes of LBP in the past year, N (%)				
1 - 3 episodes	2 (14.3)	2 (10.5)	1 (9.1)	1 (5.9)
4 - 10 episodes	5 (35.7)	4 (21.1)	5 (45.5)	9 (52.9)
> 10 episodes	7 (50.0)	13 (68.4)	5 (45.5)	7 (41.2)
Intensity of low back pain during the last week (median (IQR) for NRS 0-10)	4.0 (4.0)	5.5 (1.8)	5.0 (3.5)	5.0 (4.0)
Number of work or school days missed due to LBP, N (%)				
0 days	10 (71.4)	12 (63.2)	7 (63.6)	11 (64.7)
1 - 2 days	2 (14.3)	2 (10.5)	1 (9.1)	3 (17.6)
3 - 7 days	2 (14.3)	4 (21.1)	2 (18.2)	2 (11.8)
15 - 30 days	0 (0)	1 (5.3)	1 (9.1)	0 (0)
181 - 365 days	0 (0)	0 (0)	0 (0)	1 (1.6)
Impact of LBP, N (%) responding 'yes'				
Seeking health professional advice	7 (50.0)	11 (57.9)	7 (63.6)	12 (70.6)
Using medication for pain	5 (35.7)	11 (57.9)	2 (18.2)	5 (29.4)
Interfering with normal activities	10 (71.4)	11 (57.9)	6 (54.5)	7 (41.2)
Interfering with recreational activities	11 (78.6)	14 (73.7)	6 (54.5)	7 (41.2)
Oswestry Disability Index score (median , (IQR), range)	16.0, (13.0), 28.0	24.0, (18.0), 36.0	12.0, (8.0), 15.6	11.1, (11.1), 22.9
Pain aggravating activities N (%) responding 'yes'				
Sitting	9 (64.3)	12 (63.2)	5 (45.5)	7 (41.2)
Standing	9 (64.3)	11 (57.9)	6 (54.5)	9 (52.9)
Playing sport	9 (64.3)	9 (47.4)	6 (54.5)	5 (29.4)

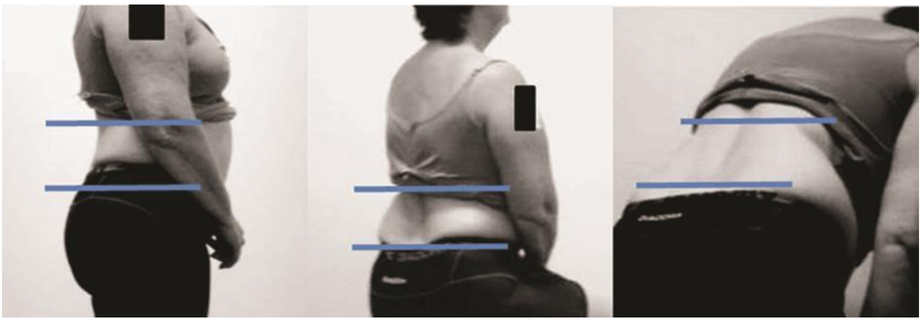
[a] Significant difference between fathers and mothers ($p < 0.05$)
Low back pain (LBP)
Interquartile range (IQR)

Prevalence of subgroups

All participants could be classified, matching one of the two subgroups (AE or MD). Clinical features of these two subgroups are presented in Figure 2a and 2b. Four participants reported pain in sitting and lifting, and no pain in standing. Based on the classification criteria relating to aggravating activities, these participants could be either classified as F or MD pattern. Postural and movement assessment revealed they presented a flexed lumbar spine posture for one of the tasks (i.e. sitting) and an extended lumbar spine posture for the other aggravating task (i.e. squatting). Therefore, these participants were sub-grouped as multidirectional pattern (MD). We did not observe any participants who could be classified into a flexion pattern (F) and therefore analyses are restricted to the AE and MD patterns only. See Table 3 for a detailed description of subgroup membership for participants in relation to their family. Forty (40) subjects were classified as AE (13 males and 27 females) and 21 participants as MD (12 males and 9 females). This distribution is in line with other studies showing the majority of patients with CDLBP to be categorised as AE or MD patterns [34, 35]. The majority of parents were classified as AE (71.4% of fathers and 89.5% of mothers), sons as MD (72.7%) and daughters as AE (58.8%) (Table 4). Significant differences in descriptive characteristics for participants within and between each subgroup were observed (Table 4). Within group comparisons showed a significant difference in median age between sons and daughters in the MD group (p=0.040). Between-group comparisons showed a significant difference in median age between sons (p=0.048), with MD sons being older than AE sons.

Figure 2 - Snapshots of video footage representing two subjects from distinct subgroups, performing a set of standardised postures and movements. **A.** Represents a mother classified as an Active Extension (AE) pattern. **B.** Represents a son classified as a Multidirectional (MD) pattern.

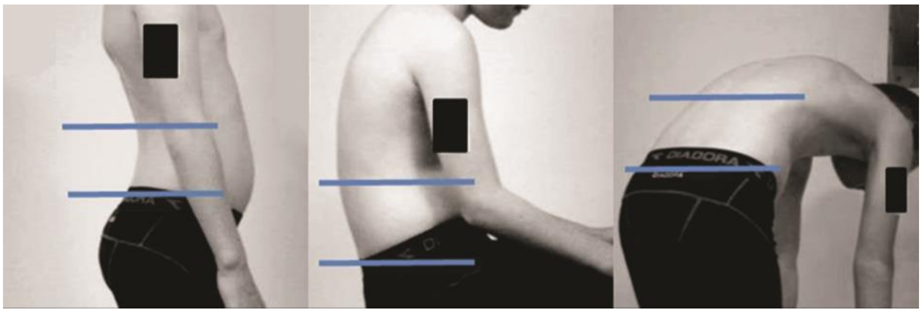
(2.A) Active extension pattern



Postures and activities involving extension of the lumbar spine aggravate symptoms (sitting, standing, walking, bending, lifting). In this example, pain is provoked in standing, sitting and forward bending associated with maintenance of lumbar extension (lordosis) in these tasks.

Provocative postures and activities associated with maintaining extension of the lumbar spine (lordotic standing, sitting and forward bending)

(2.B) Multidirectional pattern



Postures and activities associated with maintaining either flexion or extension of the lumbar spine aggravate symptoms. In this example, pain is provoked in both directions: in standing associated with maintenance of lumbar extension, and in sitting and forward bending associated with sustained lumbar flexion.

Flexion:

Postures and activities involving flexion of the lumbar spine aggravate symptoms

(sitting, forward bending, lifting, travelling).

Provocative postures and activities associated with maintaining flexion of the lumbar spine (lifting, sitting and forward bending).

Extension:

Postures and activities involving extension of the lumbar spine aggravate symptoms (standing, walking)

Provocative postures and activities associated with maintaining extension of the lumbar spine (lordotic lumbar spine in standing and walking).

199

Associations between parents and children subgroups

Overall 46.6% of all parent-child dyads were classified as the same subgroup. Percentage agreement in movement pattern-derived subgroups between parent-child dyads were 46.6%, 42.8% and 56.3% for father-child, mother-child and parent-child respectively. The dyads parent-son and parent-daughter relate to the potential association between a parent (irrespective of gender) and their son and daughter separately. The dyad parent-child relates to the potential association between the parent and their child irrespective of gender. For the correlation analysis, the offspring or parents were collapsed into a single group for the dyads involving 'child' or 'parent', respectively. Non-parametric Spearman's Rho was used to examine the strength of association between parent's and child's subgroups (Table 5). Of the nine dyads (parent-child subgroup relationships) investigated (father-son, father-daughter, father-child, mother-son, mother-daughter, mother-child, parent-son, parent-daughter, parent-child), none were found to have a statistically significant association. Mothers-sons was the only dyad presenting moderately high association of subgroups with Rho=-0.730, p=0.062. However, this association was not statistically significant due to the small number of cases. The proportion of agreement beyond that expected by chance ranged from p = 0.143 for mother-son to p = 0.476 for mother-child relationships.

Table 3- This table describes each family and its family members (F= Father, M= Mother, S= Son, D= Daughter), with their respective aggravating activities (obtained from the ODQ) and the subgroup they belong to (AE or MD). The aggravating activities are presented in hierarchical order (1-4, where 1 is most provocative, and 4 is least provocative) in terms of how provocative each task is for the participant. This information was obtained based on the score provided by the participant to each task in the ODQ.

Families	Family membership	Aggravating activities (from ODQ)				Subgroup membership	
		Lift	Walk	Sit	Stand	AE	MD
1	F	1		2	2	X	
	D	1		1			X

	M	1				X	
2	F	1		1	2	X	
	S	1		1	1		X
3	M	1			1	X	
	D	2		1	1	X	
4	M	1		2	2	X	
	S	1			1		X
5	M	2	2	1			X
	S	2	2	1	1	X	
6	M	4	2	3	1	X	
	D	1	1		1	X	
7	F	1					X
	S			1	2		X
8	F	1			1	X	
	D	1			1		X
9	M	1	3	2	3	X	
	S	2		1			X
10	F	2	2	1	1		X
	D		1	1	2	X	
11	F	1				X	
	M	1	3	2	1	X	
	D	1		2	2	X	
	D	2		1		X	
12	M	1			1	X	
	S	1		1	1		X
13	F	1			1	X	
	S	1			1		X
14	F	2			1		X
	S	1		1	1		X
15	M	1			1	X	
	F	2	2	1	2	X	
	D	2	2	1		X	
16	M			1		X	
	F	2	2	1	1	X	
	D	1		3	2		X
17	M	2	1	1	2	X	
	D	1		1			X
	D	2			1		X
18	M	1		1		X	
	F	1		2	1		X
	D	2		1	2	X	
19	M	1	3	2	1	X	
	F	2		2	1	X	
	D	1			1		X
20	F	1		1		X	
	D	2		1	2	X	

21	**M**	**2**	**2**	**1**		**X**	
	D	**3**		**2**	**1**	**X**	
22	**M**	**1**	**2**	**3**	**3**	**X**	
	D	**1**		**2**	**3**		**X**
23	**F**			**1**		**X**	
	M	**2**		**2**	**1**	**X**	
	S				**1**	**X**	
24	**M**	**1**		**1**	**1**	**X**	
	D			**1**		**1**	
25	**M**	**1**		**1**	**1**	**X**	
	S	**1**		**1**			**X**
26	**M**	**1**	**1**	**1**	**1**		**X**
	S	**1**	**2**		**2**		**X**

Table 4. Participant baseline characteristics by subgroup

Classification	Characteristic	Father	Mother	Son	Daughter
		Father *N=10 (71.4%)* Median (IQR) [min-max]	Mother *N=17 (89.5%)* Median (IQR) [min-max]	Son *N=3 (27.3%)* Median [min-max][*]	Daughter *N=10 (58.8%)* Median (IQR) [min-max]
AE	Age (years)	48.5 (7.3) [43.0-67.0]	47.0 (26.2) [38.0-56.0]	14.0 [13.0-15.0][b]	17.0 (4.8) [12.0-24.0]
	Age of onset of LBP (years)	25.0 (13.5) [18.0-37.0]	30.0 (19.5) [12.0-50.0]	12.0 [10.0-13.0]	13.0 (4.5) [9.0-20.0]
	Years since onset of LBP (years)	30.0 (13.3) [10.0-46.0]	18.0 (20.5) [1.0-35.0]	1.0 [1.0-5.0]	3.5 (2.5) [2.0-7.0]
	BMI (kg/m²)	29.7 (18.7) [22.9-38.1]	26.6 (6.3) [20.1-49.2]	19.1 [19.1-22.1]	23.7 (6.9) [14.4-34.1]
	Oswestry score (%)	14.0 (12.0), [2.0-24.0]	24.0 (20.0) [4.0-40.0]	15.6 [6.7-22.2]	12.7 (10.0) [6.7-28.9]
		Father *N=4 (28.6%)* Median (IQR) [min-max]	Mother *N=2 (10.5%)* Median [min-max][*]	Son *N=8 (72.7%)* Median (IQR) [min-max]	Daughter *N=7 (41.2%)* Median (IQR) [min-max]
MD	Age (years)	47.0 (7.0) [44.0-52.0]	38.5 [33.0-44.0]	20.0 (5.5) [13.0-25.0][b]	18.0 (5.0) [16.0-21.0]
	Age of onset of LBP (years)	18.5 (17.3) [13.0-35.0]	33.0 [30.0-36.0]	15.5 (5.5) [11.0-20.0]	15.5 (6.0) [12.0-19.0]
	Years since onset of LBP (years)	29.5 (14.3) [14.0-32.0]	5.5 [3.0-8.0]	4.5 (3.5) [1.0-10.0]	2.0 (5.0) [1.0-9.0]
	BMI (kg/m²)	28.2 (6.1) [26.0-33.1]	25.6 [25.6-38.4]	23.8 (7.1) [20.7-34.8][a]	21.4 (2.5) [19.9-27.2][a]
	Oswestry score (%)	22.0 (17.0) [10.0-30.0]	17.8 [15.6-20.0]	10.0 (8.0) [6.7-16.0]	10.0 (15.6) [6.0-24.4]

[*]Median [min-max] if n≤3
[a] Within groups: son-daughter $p<0.05$
[b]Between groups: sons $p<0.05$
Active extension (AE)
Multidirectional (MD)
Years (Y))

Table 5. Familial associations in subgroups in nine *family dyads* (parent-child relationships: mother-son, mother-daughter, mother-child; father-son, father-daughter, father-child; parent-son, parent-daughter, parent-child).

Familial dyad	Relationships (*n*)	Covariate	AE (*n*)	MD (*n*)	*ρ*	p-value
Mother-Son	7	*Mother*	5	2	-0.730	0.062
		Son	3	4		
Mother-Daughter*	14	*Mother*	14	0	-	-
		Daughter	8	6		
Mother-Child	21	*Mother*	19	2	-0.309	0.172
		Child	11	10		
Father-Son	5	*Father*	3	2	0.408	0.495
		Son	1	4		
Father-Daughter	10	*Father*	8	2	-0.408	0.242
		Daughter	6	4		
Father-Child	15	*Father*	11	4	-0.111	0.693
		Child	7	8		
Parent-Son	*11*	*Parent*	7	4	-0.386	0.241
		Child	3	8		
Parent-Daughter	*17*	*Parent*	15	2	-0.306	0.233
		Child	10	7		
Parent-Child	28	*Parent*	22	6	-0.250	0.516
		Child	13	15		

******Mothers in single group, restricting ability to test association.*

26 families (14 fathers, 19 mothers, 28 children)
Families distribution: 12 families (12/19 mothers, 13/28 children), 7 families (7/14 fathers, 7/28 children), 7 families (7/14 fathers, 7/19 mothers, 8/28 children)
Total mothers: *19 (AE group=17, MD group=2) – (21/28 children)*
Total fathers: *14 (AE group=10, MD group= 4) – (15/28 children)*

DISCUSSION

To our knowledge, this is the first study to explore associations of subgroups of postures and functional movements commonly reported to provoke LBP in a sample of families with CDLBP. It is important to highlight however, that the small sample size is a major limiting factor of this study. Therefore, the results from this study should only be considered as exploratory and a framework for future studies more adequately powered to address the research question.

The lack of parent-child dyad associations in subgroups may infer an influence of other environmental/experiential factors on the development of movement patterns in this cohort. This fits with the current understanding on movement development and behaviour, involving factors other than family [57]. Individuals develop movement uniquely, as a result of the interaction between genetics, maturation, and life experiences [57]. Individual life experiences are environmentally dependent including not only familial, but also societal and cultural influences [57]. Contributors to movement learning and development are multidimensional, including gender [58-60], BMI[60], back muscle endurance[60], TV time [60], emotional state [60-63], chronic pain [34, 64, 65], socio-cultural aspects and beliefs [66, 67]. Although genetics and familial environment can potentially influence, and be influenced by, many of these factors; the movement expression of such influences was not found to be associated within the families in this study. A future twin-study would be able to explore familial versus environmental contributions to movement patterns acquisition more definitively.

The investigation of the prevalence of movement pattern-derived subgroups in family members with CDLBP demonstrated that the proportion of parents classified as AE was greater than MD. This was substantially different to previous studies using a similar classification procedure. A considerably lower proportion of AE (8% of adults) was previously reported [43]. Similarly, Dankaerts et al. (2009) [34] reported lower prevalence of AE amongst adults (24% of adult males and 67% of adult females). These findings may reflect differences in subgrouping process, sample sizes, as well as sampling methods as both studies utilised clinical cohorts with higher disability levels, compared to this study, which used random sampling of a community-based cohort. These differences may also reflect variance in BMI and age between study samples. As BMI and age are known to influence movement and posture [28, 68], one might suggest that the older mean age and a higher mean BMI for both females and males adults in our study sample compared to Dankaerts et al. (2009) [34] might have contributed to the observed variance. However, due to insufficient number of participants this association was not assessed in the present study and requires further research to be confirmed. Future studies involving larger sample sizes could consider analyses of the influence of different age groups (e.g. 10-16yo and 17-25yo) in the subgrouping process. In children, sons were predominantly classified as MD while daughters presented a more even distribution across both groups. These findings are consistent with another study using random population sampling, which found a gender difference in subgrouping, with 78.6% of boys classified as MD and 71.4% of girls classified as AE [48]. The large discrepancy of patterns seen between adults and children might be explained by different stages and rates of development or different study samples. People might change their movement behaviour according to different factors (e.g. lifestyle, health issues, and environment) across different stages of their life. Therefore, future studies with a larger population, including multiple age groups, tracked across the lifespan would enable this to be determined.

Clinical implications

Assessment of postural and movement patterns associated with LBP is common in clinical practice. Our results support that subgrouping can be performed reliably by clinicians based on video of postures and functional movements linked to pain aggravating factors; as previously reported [43, 46, 48].

The findings of this study highlight that the underlying basis for postural and movement patterns in this particular cohort of participants with CDLBP is likely to be complex and multifactorial, consistent with a contemporary understanding of the correlates of movement behaviour. In this study, while some parents and their children presented with a remarkable likeness in the way they postured and moved, with 46.6% of all parent-child relationships similarly classified, others did not (Fig. 3). This likely indicates the potential interaction between genetic, familial, cultural and societal influences as well as individual responses to pain in this cohort, providing insight to the importance for clinicians to work within a multidimensional framework.

Figure 3 - Snapshots of video footage representing two families in sitting and squatting.

(3.A) Represents a parent-child dyad from one family displaying the same subgroup (MD).

(3.B) Represents a parent-child dyad from one family displaying different subgroups (father AE and son MD).

Limitations and recommendations

A major limitation of this study was the small sample size. A *post hoc* sample size calculation showed that a sample of 24 dyads (parent-child subgroup relationships) would be required to calculate a correlation coefficient of 0.7 with 90% power (alpha=0.05) . (G*Power 3.1.7). This information provides perspective on the analysis of this data (n= 9 dyads), and limits this study to an exploration of familial associations relevant to this sample.

The small numbers of participants in each group could have affected the ability to identify potentially important associations, or indeed contributed to spurious findings. Should this question be of further interest, future research should therefore, either include larger samples (a minimum of 24 family dyads) or utilize twin samples in order to decrease variance in genetics.

The method of assessment was based on visual analysis and individual clinical judgement, which while reliable and time efficient for a population study, resulted in categorical data excluding the possibility of exploring associations of postural and movement patterns using quantitative data.

Standardised movement-testing limited the ability to explore specific functional deficits reported by individuals. Also, as the video footage was pre-recorded in the original cohort study; there was no potential to gain more clinical information regarding pain response to adjustments in posture and movement, to help determine clear directions of pain provocation.

CONCLUSION

The results of this study provided an exploratory analysis of familial associations of two movement pattern-derived subgroups within and between members of a small number of families with CDLBP. In the population utilised in this study, movement pattern subgroups differ between parent-child dyads with CLBP. Children's subgroup membership cannot be consistently explained by their parents' movement pattern subgroups, suggesting these patterns may be influenced by multidimensional factors. Given the small sample size, the results reflect findings of this particular cohort and therefore cannot be generalised. This preliminary study can be used as a guide for future research in this area.

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