



## Original article

# An exploration of familial associations of two movement pattern-derived subgroups of chronic disabling low back pain; a cross-sectional cohort study



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## ABSTRACT

**Background:** Altered movement patterns with pain have been demonstrated in children, adolescents and adults with chronic disabling low back pain (CDLBP). A previously developed classification system has identified different subgroups including active extension and multidirectional patterns in patients with CDLBP. While familial associations have been identified for certain spinal postures in standing, it is unknown whether a familial relationship might exist between movement pattern-derived subgroups in families with CDLBP.

**Objectives:** This study explored whether familial associations in movement pattern-derived subgroups within and between members of families with CDLBP existed.

**Design:** Cross-sectional cohort study.

**Method:** 33 parents and 28 children with CDLBP were classified into two subgroups based on clinical analysis of video footage of postures and functional movements, combined with aggravating factors obtained from Oswestry Disability Questionnaire. Prevalence of subgroups within family members was determined, associations between parent and child's subgroup membership was evaluated using Fisher's exact test, and spearman's correlation coefficient was used to determine the strength of association between familial dyads.

**Results:** The majority of parents were classified as active extenders, sons predominately multidirectional and daughters were evenly distributed between the two subgroups. No significant association was found when comparing subgroups in nine parent–child relationships.

**Conclusions:** The exploration of a small cohort of family dyads in this study demonstrated that children's movement pattern-derived subgroups could not be explained by their parents' subgroup membership. These results cannot be generalised to the CLBP population due to this study's small sample. Larger sample studies are needed to further elucidate this issue.

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## 1. Introduction

Low back pain (LBP) is the leading cause of disability worldwide (Buchbinder et al., 2013). Although only 10% of people who experience LBP become disabled, this proportion of patients consumes the vast majority of LBP health resources (Linton and Ryberg, 2000; Walker et al., 2004; Becker et al., 2010). The causes of chronic disabling low back pain (CDLBP) are thought to be multifactorial (Gatchel et al., 2007) and thus may need to be considered within a

multidimensional framework for both adults (O'Sullivan, 2005a, 2012; O'Sullivan et al., 2014) and adolescents (Beales et al., 2012). Many of the contributing factors to LBP have been shown to display familial associations, reflecting genetic or shared environmental factors (Leboeuf-Yde, 2004; El-Metwally et al., 2008; Ferreira et al., 2013). Specifically: spinal structures such as degenerated discs (Ferreira et al., 2013) and bone loss (Makovey et al., 2007; Zhai et al., 2009); pain sensitivity and development of chronic pain (Hocking et al., 2010; Buchheit et al., 2012); psychological factors such as depression and anxiety (Nomura et al., 2002), pain catastrophizing (Welkom et al., 2013), distress (Caes et al., 2011), pain behaviours and coping strategies (Guite et al., 2011; Lynch-Jordan et al., 2013); lifestyle factors (Davison and Birch, 2001; Farajian et al., 2014), body mass index (BMI) (Davison and Birch, 2001; Farajian et al., 2014) and physical activity levels (den Hoed et al., 2013; Aaltonen et al., 2013) as well as lumbar range of motion (Battie et al., 1985) and back muscle endurance (Campbell et al., 2011). Recently, a familial association has been reported for spinal posture (Seah et al., 2011) in people with CDLBP. Specifically, hyperlordotic lumbar postures in standing have been shown to be more common in daughters of parents with such postures (Seah et al., 2011).

Systematic reviews suggest there is no evidence for a causal relationship between CDLBP and different spinal postures in prolonged sitting (Roffey et al., 2010a), standing (Christensen and Hartvigsen, 2008; Roffey et al., 2010b) and squatting (Roffey et al., 2010c). A potential reason is a “wash out” effect that occurs when people with different types of CDLBP are analysed homogeneously (Dankaerts et al., 2006a). However, once subgrouped based on pain provocative habitual spinal postures and movement patterns, people with CDLBP can be differentiated from healthy controls (Dankaerts et al., 2006a, 2009; Astfalck et al., 2010a). Smith et al. (2008) demonstrated that adolescents subgrouped into non-neutral standing postures, had an increased risk for LBP. Similarly, Dolphens et al. (2013) 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 (O'Sullivan, 2005a; Wai et al., 2010). A few authors have investigated CDLBP subgroups defined by movement (Sahrmann, 2002; Luomajoki et al., 2008; Kim et al., 2013; Kim and Yoo, 2015), however, only one approach acknowledges the complex multidimensional nature of CDLBP (O'Sullivan, 2005a; Vibe Fersum et al., 2009). Directional patterns of postures and movements associated with LBP outlined by O'Sullivan (2004) form part of the physical component of this multidimensional classification system (O'Sullivan et al., 2015). 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 (Dankaerts et al., 2006b, 2009; Vibe Fersum et al., 2009). 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%) (Dankaerts et al., 2006b). Dankaerts et al. (2009) 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 (Dankaerts et al., 2009).

The same movement patterns seen in adults (O'Sullivan, 2005a; Dankaerts et al., 2006b; Dankaerts et al., 2009) have been demonstrated in children (O'Sullivan et al., 2011a) and adolescents (Astfalck et al., 2010b) 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 (Dankaerts and O'Sullivan, 2011). 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 (Seah et al., 2011). 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.

## 2. Materials and methods

### 2.1. Study design

Descriptive study based on data collected in the Joondalup Spinal Health Study (JSHS) (Briggs et al., 2010), 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.

### 2.2. 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 10 km 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 (Briggs et al., 2010). 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 and 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 (Briggs et al., 2010). 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 (Briggs et al., 2010). 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

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