Musculoskeletal Science and Practice 50 (2020) 102253

Contents lists available at ScienceDirect

Musculoskeletal Science and Practice

journal homepage: www.elsevier.com/locate/msksp

Original article

Genetic and environmental effects on lumbar posture, flexibility and motion control in healthy adults

Munkh-Erdene Bayartai [a,b,*], Paulo H. Ferreira [a], Evangelos Pappas [a], Marina B. Pinheiro [a], Batlkham Dambadarjaa [b], Enkhchimeg Khuyagbaatar [b], Justin Sullivan [a]

[a] Sydney School of Health Sciences, Faculty of Medicine and Health, The University of Sydney, Sydney, Australia
[b] Department of Physical Therapy, School of Nursing, Mongolian National University of Medical Sciences, Ulaanbaatar, Mongolia

ARTICLE INFO

Keywords:
Environment
Heritability
Lumbar spine
Kinematics

ABSTRACT

Background: Although alterations in posture, flexibility, and motion control of the lumbar spine are associated with low back pain, the underlying interplay between genetic and environmental influences on these traits remains unclear. The aim of this study is to investigate the extent to which genetics and the environment influence lumbar lordosis, flexibility, and motion control.

Design: The present cross-sectional and observational study employed the classic twin design with structural equation models.

Methods: An inertial measurement unit with a wireless movement analysis system, the ViMove (DorsaVi, Melbourne, Australia) was used to measure lumbar lordosis, flexibility, and motion control during range of motion and functional tests. Intraclass correlation was used to determine twin resemblance for the traits. Heritability (genetic influence on trait variation) of lumbar lordosis, flexibility and motion control was estimated from 52 healthy twins, 34 monozygotic and 18 dizygotic using age and sex adjusted univariate genetic models.

Results: A strong heritability estimate was found in lumbar lordosis (77%, 95% confidence interval [CI]: 38%–91%) in standing, followed by lumbar flexibility (67%, 95% CI: 32%–85%) in the sagittal plane. No significant intraclass correlations were found in monozygotic twin pairs for lumbar motion control or in dizygotic twin pairs during the hurdle step and in-line lunge test.

Conclusion: Genetic factors appear to have a substantial influence on lumbar lordosis and lumbar sagittal flexibility. Lumbar motion control may be more influenced by environmental factors.

1. Introduction

Low back pain (LBP) is one of the top five leading causes of disability globally (GBD, 2016 Disease and Injury Incidence and Prevalence Collaborators, 2017). It is estimated that 8 out of 10 people will experience LBP at some stage of their lifetime, with 11–12% of people with LBP being disabled by their condition (Balague et al., 2012). The influence of genetics on LBP has been shown to be substantial. Monozygotic (MZ) twins, who share 100% of their genes, are five times more likely than dizygotic (DZ) twins, who share 50% of their genes, to experience LBP if their co-twin had previously experienced LBP (Junqueira et al., 2014). The classic twin design is commonly used to estimate heritability (genetic influence on trait variation) based on twin resemblance (Mayhew and Meyre, 2017). It is estimated that the heritability for experiencing LBP varies between 21 and 67% (Ferreira et al., 2013). Although it has been shown that genetic factors play a substantial role in LBP, the pathways by which genetics influence LBP remain unclear.

Genetics may influence LBP by altering posture, flexibility, and motion control of the lumbar spine. In a study that followed 402 healthy workers with no serious back pain in the preceding three years, reduced lumbar lordosis and mobility were consistent predictors for developing serious LBP during the follow-up period (Adams et al., 1999). People with LBP exhibit reduced lumbar range of motion (ROM) in all directions (Laird et al., 2014). In addition, altered control of movement is commonly used as a criteria for classifying people into one of three chronic LBP sub-groups, where abnormal tissue loading is associated with decreased or excessive spinal stability (O'Sullivan, 2005). Therefore, measurements of spinal flexibility and motion control are commonly performed in the assessment and management of LBP (Delitto et al., 2012). Although alterations in spinal posture, flexibility and

* Corresponding author. Sydney School of Health Sciences, Cumberland Campus, The University of Sydney, PO Box 170, Lidcombe, NSW, 1825, Australia.
E-mail address: mbay0086@uni.sydney.edu.au (M.-E. Bayartai).

https://doi.org/10.1016/j.msksp.2020.102253
Received 12 June 2020; Received in revised form 27 August 2020; Accepted 29 August 2020
Available online 4 September 2020

motion control are associated with LBP, the underlying genetic and environmental influences on these variables have remained unclear. To date, only one study has examined the genetic and environmental influences on lumbar motion, with the investigations limited to ROM in the sagittal plane (Battie et al., 2008). Heritability, defined as the amount of phenotypic variation due to genetic differences, of total lumbar ROM, lumbar flexion and lumbar extension was estimated to be 47%, 64% and 39%, respectively. In healthy adults, the heritability of lumbar lordosis in standing and lumbar motion control during functional tasks have not yet been investigated.

The aim of this study is to investigate the extent to which genetics and the environment influence lumbar lordosis, flexibility, and motion control in healthy participants.

2. Methods

The present cross-sectional and observational study employed the classic twin design with structural equation models (Grasby et al., 2017) to investigate genetic and environmental effects on lumbar lordosis, flexibility, as well as motion control and followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines (Supplementary material 1). Twin design studies, which are commonly used to determine heritability of phenotypes, estimate heritability by comparing the similarity of a trait within MZ and DZ pairs. In the classic twin design, the influence of genetics is demonstrated if MZ twin pairs are more similar than DZ twin pairs on the trait of interest (Mayhew and Meyre, 2017).

2.1. Participants

The recruitment period for this study was between December 2018 and March 2019. Participants were recruited from the general population in Ulaanbaatar, Mongolia by advertising with flyers placed on notice boards in universities, health centres and public libraries. Participants were included if they were MZ or DZ twins, aged from 18 to 50 and considered themselves healthy for their age. Both twins of any pair had to agree to participate. After participants were determined to have met the above criteria, the following were screened as exclusion criteria: musculoskeletal conditions affecting daily physical activity that would make the functional tasks required in the study difficult to do; neurological disorders; a history of spinal surgery or any episode of back pain that had required more than four weeks absence from work or normal activities (Qaseem et al., 2017). Informed consent was obtained from all eligible participants. The study was approved by the Ethics Committee, Mongolian National University of Medical Sciences (№ 2018/3-10). Participants were invited to a university laboratory to complete a demographic questionnaire and perform ROM and functional tests for the collection of lumbar kinematic data. A 'peas in a pod' questionnaire (PPQ) was used to determine twin zygosity. The PPQ has been demonstrated to be an excellent proxy indicator of zygosity when genotyping information is not available (Jarrar et al., 2018). The sample size ($n = 52$ twins) was determined using G*Power 3.1 tool (Faul et al., 2009) based on power $\geq 90\%$, a type I error of $\alpha = 0.05$, and the minimal expected differences between MZ and DZ twins (Missitzi et al., 2018) set at $h^2 = 0.39$ as reported in an earlier twin study (Battie et al., 2008).

2.2. Measurements

Participants were asked to perform the following tasks whilst lumbar motion was monitored with the ViMove (DorsaVi, Melbourne, Australia) sensors. The ViMove, an Inertial Measurement Unit (IMU) and wireless movement analysis system, is designed to measure and quantify lumbar movement. In recent years, the popularity of evaluating joint kinematics using the IMU sensors has increased because of its simplicity, efficiency and reduced cost compared with three-dimensional analyses in a biomechanics laboratory. An accelerometer (3 Axis, ±2 to ±24G), gyroscope (3 Axis, ±250°/s to ±1200°/s) and magnetometer (3 Axis, ±1.3G) (Dorsavi, 2017) embedded in the ViMove wireless movement sensors were able to record a ROM value every 50 ms (20 Hz). The ViMove has been validated against the Vicon motion capture system for measurements of lumbar flexion, extension and lateral flexion (Mjosund et al., 2017). The measurement errors for lumbar flexion and extension ROM are low, with root mean squared errors of 1.82° ± 1.00° and 0.71° ± 0.34°, respectively (Mjosund et al., 2017).

2.2.1. IMU sensor placement

To measure lumbar kinematics, the ViMove uses two non-invasive, wireless sensors placed on the lower back using double-sided stickers. The lower sensor was attached directly below a line drawn between both Posterior Superior Iliac Spines. The position of the upper sensor, approximately at the twelfth thoracic segment, was determined by using one of the specific ViMove templates. The template size chosen was based on the individual participant's height (Mjosund et al., 2017). After sensor placement, participants were asked to stand in their usual upright position for sensor calibration and measurement of their posture, specifically the lumbar lordosis angle.

2.2.2. ROM and functional tests

Participants were asked to perform lumbar flexion and extension ROM tasks for the measurement of lumbar flexibility, and hurdle step and in-line lunge functional tests for the measurement of lumbar motion control according to the protocol (Dorsavi, 2017) provided by the ViMove software (Table 1). These functional tests assessed using the ViMove are based on the Functional Movement Screen, which is a reliable observation-based grading tool for movement assessment that is used to screen for sports injury risk (Minick et al., 2010).

2.3. Data processing

Kinematic data, including lumbar flexion, extension and lateral flexion ROM values, were recorded using the ViMove software during each ROM and functional test. In the data analyses, all measurements of lumbar motion were extracted using the ViMove software in order to calculate the heritability of lumbar lordosis, motion control and flexibility. First, lumbar lordosis was measured during the calibration process. Second, an individual's lumbar motion control during the functional tests was determined by calculating the coefficient of variation (standard deviation divided by the mean, multiplied by 100) (Steele et al., 2014) of the displacements of the lumbar spine from the baseline (starting position) during each test. This measure was chosen to best represent the level of control of lumbar motion throughout the entire duration of a functional test. The baseline at either end of the streaming graph of each test is provided by the software. The beginning and end of each trial were determined by the closest point to the baseline. For the functional tasks, the spine could potentially move into flexion or extension, or move from flexion to extension during the task, so it was determined that total motion in the sagittal plane would better capture movement variance. Third, an individual's flexibility was determined as the maximum ROM values in the sagittal plane recorded by the ViMove software during the ROM tests. For each test, the average maximum ROM of three trials was used in the analysis.

2.4. Statistical analyses

Descriptive statistics, tests for normality and correlation analyses were performed using SPSS, version 25 (IBM SPSS Statistics for Windows, Armonk, NY, USA). The Shapiro-Wilk test was performed to assess the normality of kinematic data. Independent samples T tests were used to compare participant characteristics, posture, lumbar flexibility and motion control between the MZ and DZ twins. Twin resemblance for the traits was determined by Shrout and Fleiss (type 1,1) intraclass correlation in MZ and DZ twins separately using a one-way random effects

Table 1
ROM and functional tasks performed by the participants.

Tasks	Kinematic data measured	Instructions provided to the participants
Lumbar flexion	Flexion ROM	Participants were asked to stand upright and flex forward as far as comfortable without bending the knees, and hold for 3 s and then return to the starting position.
Lumbar extension	Extension ROM	Participants were asked to stand upright and fold their arms across their chest. They were then asked to bend backward (extension) as far as comfortable without bending the knees, hold for 3 s and return to the starting position.
Lumbar lateral flexion	Right and left lateral flexion ROM	Participants were asked to stand in an upright position with their arms by their side. They were then asked to bend sideways (flex laterally) and slide their arm along the leg as far as comfortable while avoiding any motion in flexion/extension, hold for 3 s and return to the starting position.
Hurdle step	Lumbar flexion/extension ROM, lumbar lateral flexion ROM	Participants were asked to stand and hold a light bar (to standardise arm position) across the back of their shoulders, place their feet shoulder-width apart and their toes aligned directly beneath the hurdle. They were then asked to slowly step over the hurdle with the right leg, touch the ground with their heel, then return to the starting position with the reverse movement. The hurdle was set at the height of the participants' knee joint line.
In-line lunge	Lumbar flexion/extension ROM, lumbar lateral flexion ROM	Participants were given a dowel (to standardise arm position) to hold along the spine. They were asked to grasp the top of the dowel with the left hand and the lower end of the dowel with the right hand. They were then asked to step forward with the right leg, a distance equivalent to the length of their lower leg, with their two feet directly in-line. They then lowered their back knee to touch the ground and immediately returned to the starting position. Participants held the top of the dowel with the hand ipsilateral to the back foot and the other end of the dowel with the contralateral hand.

ROM - range of motion.

model, where each twin pair is considered a random effect (Sham, 2001; Shrout and Fleiss, 1979). In order to compare within pair correlations of MZ and DZ twin pairs for the traits, the classic twin design was used (Hopper et al., 1998). In the classic twin design, the influence of genetics on a trait is expected if higher intraclass correlations were calculated in MZ twin pairs, who are genetically identical, than DZ twin pairs, who share half of their genes. For the traits with higher correlations calculated in MZ twin pairs compared to DZ twin pairs, age and sex adjusted univariate genetic models, which allow to more precisely estimate heritability with variance components (Grasby et al., 2017) were fitted to estimate the heritability of the traits. In the genetic models, additive genetic (A), common shared (C), and non-shared environmental (E) components were considered to estimate the variance in the traits. In order to estimate the proportion of the A, C and E variance components, different models such as ACE, AE and E were computed. Goodness-of-fit statistics were used to find the most parsimonious model, and Akaike's Information Criterion (AIC = χ^2 – 2 d.f., where χ^2 is a chi square test and d.f. is degrees of freedom) was used to assess sub-models (Neale et al., 2016). The AIC is a statistical tool that measures the quality of different statistical models in order to select the best fit model (Akaike, 1974). Lower AIC values indicate models with better fit. Genetic modelling was performed using the 'OpenMx' package (Neale et al., 2016) in R version 3.6.0 (R Core Team, 2019).

3. Results

Complete data for all lumbar lordosis, flexibility, and motion control measures were collected for 52 healthy twins, 34 MZ and 18 DZ twins (Supplementary material 2). Participant characteristics are presented in Table 2. Overall, MZ and DZ twins were not different for age, anthropometric measures, nor measures of lumbar lordosis, sagittal flexibility, and motion control. However, the MZ and DZ groups had different mean lumbar flexibility in the frontal plane, meaning that no further analyses were conducted to calculate the heritability of lumbar frontal flexibility as this would violate the assumptions of the classic twin design (Grasby et al., 2017).

Intraclass correlation coefficients for lumbar lordosis and sagittal flexibility measures were higher within the MZ twin pairs than the DZ pairs, indicating that these traits are possibly influenced by genetics. No significant intraclass correlations were found in the MZ or DZ twin pairs for the outcome measures of lumbar motion control (Table 3). Heritability of lumbar lordosis and sagittal flexibility were determined by the age and sex adjusted univariate genetic model (Table 4).

The AE model, which includes additive genetic and unique environmental components, was the best fitting model for both traits because it had the lowest AIC.

Heritability estimates were high for lumbar lordosis in standing (77%, 95% confidence interval [CI]: 38%–91%) and moderate for

Table 2
Participants' characteristics, lumbar lordosis, flexibility and motion control.

Variables	MZ Twins (n = 34)	DZ twins (n = 18)	p value
	Mean ± SD	Mean ± SD	
Characteristics			
Age (years)	26.0 ± 10.8	22.7 ± 5.2	0.14
Weight (kg)	58.6 ± 9.6	61.8 ± 8.7	0.24
Height (m)	1.6 ± 0.9	1.7 ± 0.8	0.24
BMI (kg/m^2)	22.2 ± 2.7	22.7 ± 3.2	0.55
Female (%)	71	83	0.50
Lumbar lordosis (degrees)	31.3 ± 8.5	30.9 ± 9.2	0.85
Lumbar flexibility (degrees)			
Total flexion-extension	93.1 ± 18.1	94.3 ± 14.4	0.80
Flexion	66.2 ± 9.7	66.2 ± 9.1	0.16
Extension	30.8 ± 14.3	28.1 ± 15.9	0.53
Total right and left lateral flexion	52.9 ± 9.1	60.5 ± 7.7	0.004
Right lateral flexion	27.2 ± 5.8	30.4 ± 4.2	0.04
Left lateral flexion	25.8 ± 6.1	30.1 ± 4.5	0.01
Lumbar motion control (degrees)			
Hurdle step, maximum sagittal mobility	19.8 ± 7.1	23.8 ± 6.2	0.05
Hurdle step, maximum frontal mobility	20.0 ± 6.3	20.9 ± 6.5	0.61
In-line lunge, maximum sagittal mobility	18.6 ± 8.9	19.8 ± 10.9	0.66
In-line lunge, maximum frontal mobility	15.4 ± 5.6	13.6 ± 4.9	0.26
Lumbar motion control (Coefficient of variation, %)			
Hurdle step, maximum sagittal mobility	136.9 ± 126.6	126.2 ± 96.5	0.75
Hurdle step, maximum frontal mobility	337.9 ± 187.7	430.5 ± 473.1	0.32
In-line lunge, maximum sagittal mobility	134.6 ± 153.3	91.2 ± 90.8	0.28
In-line lunge, maximum frontal mobility	195.5 ± 136.9	197.5 ± 87.1	0.96

MZ - monozygotic, DZ - dizygotic, p - statistical differences between MZ and DZ twins, SD - standard deviation.

Table 3
Intraclass correlation coefficients for lumbar lordosis, flexibility and motion control among monozygotic and dizygotic twin pairs.

Outcome measure	Monozygotic twin pairs		Dizygotic twin pairs	
	rMZ	95% CI	rDZ	95% CI
Lumbar lordosis	0.84	0.61 to 0.94	0.20	−0.47 to 0.73
Lumbar sagittal flexibility	0.73	0.40 to 0.89	0.29	−0.39 to 0.77
Lumbar motion control (during functional tests)				
Hurdle step, lumbar mobility in the sagittal plane	−0.07	−0.51 to 0.41	0.02	−0.62 to 0.67
Hurdle step, lumbar mobility in the frontal plane	0.01	−0.45 to 0.48	0.22	−0.44 to 0.74
In-line lunge, lumbar mobility in the sagittal plane	−0.01	−0.47 to 0.58	−0.26	−0.75 to 0.44
In-line lunge, lumbar mobility in the frontal plane	−0.09	−0.53 to 0.39	−0.32	−0.78 to 0.39

rMZ - intraclass within-pair correlation for MZ twins, rDZ - intraclass within-pair correlation for DZ twins, one-way random effect model was used in the intraclass correlations.

lumbar sagittal flexibility (67%, 95% CI: 32%–85%) (Table 4). Thus, the variance in lumbar sagittal flexibility was moderately influenced by unique environmental factors (33%, 95% CI: 15%–68%), followed by lumbar lordosis in standing (23%, 95% CI: 9%–62%). However, there was no heritability associated with lumbar motion control as no significant correlations were found within the twin pairs.

4. Discussion

In the present study, the heritability estimates for lumbar lordosis, flexibility and motion control were investigated in healthy people. Lumbar lordosis in standing had the highest heritability estimate, followed by lumbar flexibility, whereas no heritability was associated with lumbar motion control. Therefore, environmental contributions play a greater role in determining the variance in lumbar motion control, compared to lumbar flexibility and posture.

The variance in lumbar lordosis was predominantly influenced by genetic factors. One previous study that included participants with disc degeneration found the heritability of lumbar lordosis to be 59% (Stone et al., 2015), which was lower than the heritability estimated by the current study. However, this difference in heritability estimates could be explained by the inclusion of participants with disc degeneration and a much higher mean age of 64 in the earlier study, as posture appears to be influenced by these characteristics (Dreischarf et al., 2014; Lao et al., 2015). A recent systematic review that investigated the association between lumbar lordosis and LBP found that decreased lumbar lordosis was strongly associated with LBP (Chun et al., 2017). Considering that the heritability of LBP has been estimated to be between 27% and 67% depending on LBP severity (Ferreira et al., 2013), the genetic influence on lumbar lordosis may be a pathway through which genetics contributes to LBP. However, future twin studies could help identify the genetic correlation between lumbar lordosis and LBP.

The variance in lumbar sagittal flexibility was moderately influenced by genetics. The heritability estimate for flexibility was slightly higher than the one previous study available to date, where it was determined to be 47% (Battie et al., 2008) in older participants. The heritability estimates from this study may have been affected by the samples age (Dreischarf et al., 2014) and presence of people with disc degeneration, which reduce lumbar mobility (Lao et al., 2015). Nevertheless, the available evidence suggests that lumbar flexibility is moderately influenced by genetics. A previous systematic review has also found that people with LBP had reduced lumbar flexibility compared to those without LBP (Laird et al., 2014). Therefore, the genetic influence on lumbar ROM might be part of the risk factor for LBP. Additionally, reduced lumbar flexion and spinal stiffness have previously been measured in people who were instructed to do seated desk work for a period of 2 h (Beach et al., 2005), implying environmental factors such as occupational activity may also play a role in influencing lumbar sagittal flexibility. Previous studies have also found that lumbar flexibility can be improved by training in both healthy and LBP participants (Kofotolis and Kellis, 2006; Rider and Daly, 1991), implying that training may modify lumbar flexibility in people with and without LBP. In addition, a recent systematic review that included prospective cohort studies demonstrated that people with reduced lateral bending ROM were 144% more likely to develop LBP (Sadler et al., 2017), suggesting that decreased lumbar ROM could be a cause of LBP. However, as the MZ and DZ groups had different mean lumbar flexibility in the frontal plane, we were unable to analyse this further and calculate the heritability of motion in this plane (Grasby et al., 2017). Given the association between lumbar lateral bending and LBP, future studies are needed to determine the extent to which lumbar frontal flexibility is influenced by genetics.

No heritability was associated with lumbar motion control and thus, individual differences in the trait appear to be due to environmental factors. No previous studies have specifically estimated the heritability of lumbar motion control, and this is preliminary evidence that there is no genetic influence on the control of lumbar motion during a functional task, unlike the findings for lumbar lordosis and ROM. There is evidence that lumbar motion control can be improved by environmental variables, specifically therapeutic exercise. People with chronic LBP appear to benefit from specific lumbar stabilization exercises (Moon et al., 2013). A four-week training designed to strengthen pelvic floor and core muscles has been shown to improve lumbar stability in healthy individuals (Hsu et al., 2018). Although heritability estimation cannot determine whether a trait is modifiable or not, results from our study suggest that environmental influences appear to explain the total variance of lumbar motion control.

The main limitation of the current study was its small sample size

Table 4
Age and sex adjusted univariate genetic analysis for lumbar lordosis and flexibility.

Test	Model	h^2	c^2	e^2	-2LL	Df	ΔLL	Δdf	AIC	W_{AE}/W_i	p
Lumbar lordosis	ACE	0.77 (0.28–0.91)	0 (0.00–0.35)	0.23 (0.09–0.62)	344.16	46.00	–	–	252.16	–	–
	AE	**0.77 (0.38–0.91)**	**-**	**0.23 (0.09–0.62)**	**344.16**	**47.00**	**0.00**	**1.00**	**250.16**	**1.00**	**1.00**
	CE	–	0.36 (0.00–0.66)	0.64 (0.34–1.00)	350.52	47.00	6.36	1.00	256.52	24.04	0.01
	E	–	–	1	353.86	48.00	9.69	2.00	257.86	127.74	0.01
Lumbar sagittal flexibility	ACE	0.67 (0.00–0.85)	0 (0.00–0.70)	0.33 (0.15–0.69)	414.40	46.00	–	–	322.40	–	–
	AE	**0.67 (0.32–0.85)**	**-**	**0.33 (0.15–0.68)**	**414.40**	**47.00**	**0.00**	**1.00**	**320.40**	**1.00**	**1.00**
	CE	–	0.53 (0.20–0.76)	0.47 (0.24–0.80)	416.27	47.00	1.87	1.00	322.27	2.55	0.17
	E	–	–	1	424.94	48.00	10.53	2.00	328.94	71.52	0.01

Notes: A, C and E stand for the additive genetic, common shared and non-shared environmental components of variance. Standardized estimates and 95% confidence intervals (CI) for the proportion of the components were estimated for each trait under h^2, c^2 and e^2, respectively. The models with the lowest Akaike's Information Criterion (AIC) as the best fit are bold faced in the table. W_{AE}/W_i stands for the evidence ratio of Akaike weights, which shows that how many times AE model is more likely to be the best fit model compared to the other models. No p values are presented in ACE as the full model to which other sub-models are compared. LL - Log-likelihood, df - degrees of freedom, ΔLL - difference between the ACE and the sub-models, Δdf - difference in degrees of freedom between the ACE and the sub-models.

and wide confidence intervals in the heritability estimates. The functional tests included in the present study, although able to measure lumbar motion and control during whole body tasks, are not typical of everyday tasks for the general population. The accuracy of the ViMove sensors on the appropriate anatomic landmarks could potentially be affected by skin movement artefact or placement errors. In addition, the present study included young, lean and mostly female participants and, therefore, the findings may not be generalisable across people of all age groups and body types, or to males. These data, however, provide preliminary evidence regarding the relative level of genetic and environmental influences on lumbar lordosis, flexibility and motion control, which could be confirmed through further studies with larger sample sizes, set in different countries with varied environmental contexts.

5. Conclusion

Genetic factors appear to substantially influence lumbar lordosis and sagittal flexibility. However, no heritability was associated with lumbar motion control. Potentially modifiable environmental factors may have a significant influence on lumbar motion control during functional tests and should be further investigated.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.msksp.2020.102253.

References

Adams, M.A., Mannion, A.F., Dolan, P., 1999. Personal risk factors for first-time low back pain. Spine (Phila Pa 1976) 24 (23), 2497–2505.

Akaike, H., 1974. A new look at the statistical model identification. IEEE Trans. Automat. Contr. 19 (6), 716–723. https://doi.org/10.1109/TAC.1974.1100705.

Balague, F., Mannion, A.F., Pellise, F., Cedraschi, C., 2012. Non-specific low back pain. Lancet 379 (9814), 482–491. https://doi.org/10.1016/s0140-6736(11)60610-7.

Battie, M.C.L.E., Videman, T., Burton, K., Kaprio, J., 2008. Heritability of lumbar flexibility and the role of disc degeneration and body weight. J. Appl. Physiol. 104 (2), 379–385. https://doi.org/10.1152/japplphysiol.01009.2007, 1985.

Beach, T.A., Parkinson, R.J., Stothart, J.P., Callaghan, J.P., 2005. Effects of prolonged sitting on the passive flexion stiffness of the in vivo lumbar spine. Spine J. 5 (2), 145–154. https://doi.org/10.1016/j.spinee.2004.07.036.

Chun, S.W., Lim, C.Y., Kim, K., Hwang, J., Chung, S.G., 2017. The relationships between low back pain and lumbar lordosis: a systematic review and meta-analysis. Spine J. 17 (8), 1180–1191. https://doi.org/10.1016/j.spinee.2017.04.034.

Delitto, A., George, S.Z., Van Dillen, L., Whitman, J.M., Sowa, G., Shekelle, P., et al., 2012. Orthopaedic section of the American physical therapy, A. Low back pain. J. Orthop. Sports Phys. Ther. 42 (4), A1–A57. https://doi.org/10.2519/jospt.2012.42.4.A1.

Dorsavi, 2017. DorsaVi Support Center. Retrieved from. http://support.dorsavi.com/.

Dreischarf, M., Albiol, L., Rohlmann, A., Pries, E., Bashkuev, M., Zander, T., Schmidt, H., 2014. Age-related loss of lumbar spinal lordosis and mobility–a study of 323 asymptomatic volunteers. PLoS One 9 (12), e116186. https://doi.org/10.1371/journal.pone.0116186.

Faul, F., Erdfelder, E., Buchner, A., Lang, A.-G., 2009. Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. Behav. Res. Methods 41 (4), 1149–1160. https://doi.org/10.3758/BRM.41.4.1149.

Ferreira, P.H., Beckenkamp, P., Maher, C.G., Hopper, J.L., Ferreira, M.L., 2013. Nature or nurture in low back pain? Results of a systematic review of studies based on twin samples. Eur. J. Pain 17 (7), 957–971. https://doi.org/10.1002/j.1532-2149.2012.00277.x.

GBD 2016 Disease and Injury Incidence and Prevalence Collaborators, 2017. Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet 390 (10100), 1211–1259. https://doi.org/10.1016/s0140-6736(17)32154-2.

Grasby, K.L., Verweij, K.J.H., Mosing, M.A., Zietsch, B.P., Medland, S.E., 2017. Estimating heritability from twin studies. Methods Mol. Biol. 1666, 171–194. https://doi.org/10.1007/978-1-4939-7274-6_9.

Hopper, J.L., Green, R.M., Nowson, C.A., Young, D., Sherwin, A.J., Kaymakci, B., et al., 1998. Genetic, common environment, and individual specific components of variance for bone mineral density in 10- to 26-year-old females: a twin study. Am. J. Epidemiol. 147 (1), 17–29. https://doi.org/10.1093/oxfordjournals.aje.a009361.

Hsu, S.L., Oda, H., Shirahata, S., Watanabe, M., Sasaki, M., 2018. Effects of core strength training on core stability. J. Phys. Ther. Sci. 30 (8), 1014–1018. https://doi.org/10.1589/jpts.30.1014.

Jarrar, Z.A., Ward, K.J., Mangino, M., Cherkas, L.F., Gill, R., Gillham-Nasenya, I., et al., 2018. Definitive zygosity scores in the peas in the pod questionnaire is a sensitive and accurate assessment of the zygosity of adult twins. Twin Res. Hum. Genet. 21 (2), 146–154. https://doi.org/10.1017/thg.2018.9.

Junqueira, D.R., Ferreira, M.L., Refshauge, K., Maher, C.G., Hopper, J.L., Hancock, M., et al., 2014. Heritability and lifestyle factors in chronic low back pain: results of the Australian twin low back pain study (The AUTBACK study). Eur. J. Pain 18 (10), 1410–1418. https://doi.org/10.1002/ejp.506.

Kofotolis, N., Kellis, E., 2006. Effects of two 4-week proprioceptive neuromuscular facilitation programs on muscle endurance, flexibility, and functional performance in women with chronic low back pain. Phys. Ther. 86 (7), 1001–1012. https://doi.org/10.1093/ptj/86.7.1001.

Laird, R.A., Gilbert, J., Kent, P., Keating, J.L., 2014. Comparing lumbo-pelvic kinematics in people with and without back pain: a systematic review and meta-analysis. BMC Muscoskel. Disord. 15, 229. https://doi.org/10.1186/1471-2474-15-229.

Lao, L., Daubs, M.D., Scott, T.P., Lord, E.L., Cohen, J.R., Yin, R., et al., 2015. Effect of disc degeneration on lumbar segmental mobility analyzed by kinetic magnetic resonance imaging. Spine (Phila Pa 1976) 40 (5), 316–322. https://doi.org/10.1097/brs.0000000000000738.

Mayhew, A.J., Meyre, D., 2017. Assessing the heritability of complex traits in humans: methodological challenges and opportunities. Curr. Genom. 18 (4), 332–340. https://doi.org/10.2174/1389202918666170307161450.

Minick, K.I., Kiesel, K.B., Burton, L., Taylor, A., Plisky, P., Butler, R.J., 2010. Interrater reliability of the functional movement screen. J. Strength Condit Res. 24 (2), 479–486. https://doi.org/10.1519/JSC.0b013e3181c09c04.

Missitzi, J., Geladas, N., Misitzi, A., Misitzis, L., Classen, J., Klissouras, V., 2018. Heritability of proprioceptive senses. J. Appl. Physiol. 125 (4), 972–982. https://doi.org/10.1152/japplphysiol.00544.2017.

Mjosund, H.L., Boyle, E., Kjaer, P., Mieritz, R.M., Skallgard, T., Kent, P., 2017. Clinically acceptable agreement between the ViMove wireless motion sensor system and the Vicon motion capture system when measuring lumbar region inclination motion in the sagittal and coronal planes. BMC Muscoskel. Disord. 18 (1), 124. https://doi.org/10.1186/s12891-017-1489-1.

Moon, H.J., Choi, K.H., Kim, D.H., Kim, H.J., Cho, Y.K., Lee, K.H., et al., 2013. Effect of lumbar stabilization and dynamic lumbar strengthening exercises in patients with chronic low back pain. Ann. Rehabil. Med. 37 (1), 110–117. https://doi.org/10.5535/arm.2013.37.1.110.

Neale, M.C., Hunter, M.D., Pritikin, J.N., Zahery, M., Brick, T.R., Kirkpatrick, R.M., et al., 2016. OpenMx 2.0: extended structural equation and statistical modeling. Psychometrika 81 (2), 535–549. https://doi.org/10.1007/s11336-014-9435-8.

O'Sullivan, P., 2005. Diagnosis and classification of chronic low back pain disorders: maladaptive movement and motor control impairments as underlying mechanism. Man. Ther. 10 (4), 242–255. https://doi.org/10.1016/j.math.2005.07.001.

Qaseem, A., Wilt, T.J., McLean, R.M., Forciea, M.A., 2017. Noninvasive treatments for acute, subacute, and chronic low back pain: a clinical practice guideline from the American college of physicians. Ann. Intern. Med. 166 (7), 514–530. https://doi.org/10.7326/M16-2367.

R Core Team, 2019. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing. Retrieved from. https://www.R-project.org/.

Rider, R.A., Daly, J., 1991. Effects of flexibility training on enhancing spinal mobility in older women. J. Sports Med. Phys. Fit. 31 (2), 213–217.

Sadler, S.G., Spink, M.J., Ho, A., De Jonge, X.J., Chuter, V.H., 2017. Restriction in lateral bending range of motion, lumbar lordosis, and hamstring flexibility predicts the development of low back pain: a systematic review of prospective cohort studies. BMC Muscoskel. Disord. 18 (1) https://doi.org/10.1186/s12891-017-1534-0, 179-179.

Sham, P., 2001. Statistics in Human Genetics. Arnold ; Oxford University Press, London; New York.

Shrout, P.E., Fleiss, J.L., 1979. Intraclass correlations: uses in assessing rater reliability. Psychol. Bull. 86 (2), 420–428. https://doi.org/10.1037//0033-2909.86.2.420.

Steele, J., Bruce-Low, S., Smith, D., Jessop, D., Osborne, N., 2014. Lumbar kinematic variability during gait in chronic low back pain and associations with pain, disability and isolated lumbar extension strength. Clin. Biomech. (Bristol, Avon) 29 (10), 1131–1138. https://doi.org/10.1016/j.clinbiomech.2014.09.013.

Stone, M.A., Osei-Bordom, D.C., Inman, R.D., Sammon, C., Wolber, L.E., Williams, F.M., 2015. Heritability of spinal curvature and its relationship to disc degeneration and bone mineral density in female adult twins. Eur. Spine J. 24 (11), 2387–2394. https://doi.org/10.1007/s00586-014-3477-6.