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ORIGINAL ARTICLE

Contributions of birthweight, annualised weight gain and BMI to back pain in adults: a population-based co-twin control study of 2754 Australian twins

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Abstract

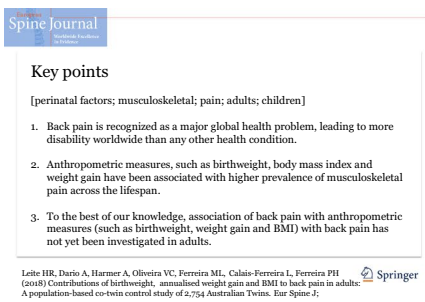
Purpose To investigate associations between anthropometric measures (birthweight, weight gain and current BMI) and back pain; and to determine whether these associations differ between those born with low or full birthweight.

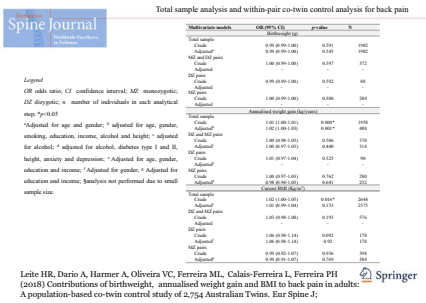
Methods The cross-sectional associations between the lifetime prevalence of back pain and anthropometric measures (birthweight, weight gain and current BMI) among 2754 adult twins were investigated in three stages: total sample; within-pair case–control for monozygotic and dizygotic twins together; and within-pair case–control analysis separated by dizygotic and monozygotic. Results were expressed as odds ratios (OR) and 95% confidence intervals (CI).

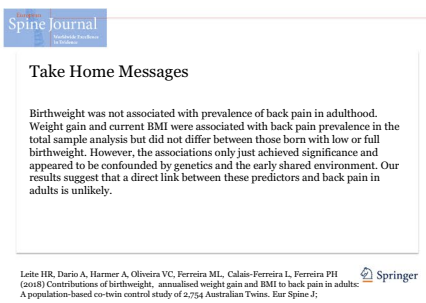
Results Birthweight was not associated with back pain (OR 0.99; 95% CI 0.99–1.00), but a weak association was found between weight gain (OR 1.01; CI 1.00–1.01) or current BMI (OR 1.02; 95% CI 1.00–1.05) and back pain in the total sample analysis. These associations did not remain significant after adjusting for genetics. The associations did not differ between those whose were born with low or full birthweight.

Conclusion Birthweight was not associated with prevalence of back pain in adulthood. Weight gain and current BMI were weakly associated with back pain prevalence in the total sample analysis but did not differ between those born with low or full birthweight. However, the small-magnitude association only just achieved significance and appeared to be confounded by genetics and the early shared environment. Our results suggest that a direct link between these predictors and back pain in adults is unlikely.

Graphical abstract These slides can be retrieved under Electronic Supplementary Material.







Keywords Birthweight · BMI · Twins · Back pain · Genetics

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Introduction

Back pain is defined as pain in the lower and/or upper back area [1]. According to the Australian Institute of Health and Welfare, back pain was the third leading cause of disease in Australia in 2011, and one in every six Australians (16%) reported back pain in 2014–2015, which represents 3.7 million people [2]. Back pain also has a high socioeconomic impact, with more than $1.4 billion spent annually with treatment costs in Australia [3]. The aetiology of back pain remains unknown, and although some contributing factors have been suggested such as psychological, social and genetics [4], others, such as anthropometric measures in early and later life, have been inconsistently related to back pain.

Anthropometric measures, such as birthweight [5, 6], body mass index (BMI) [7, 8] and weight gain [9, 10], have been associated with higher prevalence of musculoskeletal pain across the lifespan. However, the relationship between those variables and back pain is not clear. For example, while several studies showed that a high BMI (e.g. adults) was associated with greater risk of reporting low back pain in adult life [7, 8, 11, 12], other studies failed to show any association [13–15].

Twin studies have been used to provide matching of age, sex, maternal and familial (both genetic and early shared environmental) factors when utilising a co-twin control design to investigate risk factors and intervention strategies for individuals with back pain. When utilising such a strong study design, the association between BMI and back pain disappeared after controlling for genetics and the shared environment in twins discordant for back pain [16]. Similar findings have been observed for birthweight. In a cohort study of adolescent twins, low back pain was associated with birthweight, although the risk was attenuated after adjustment for genetics and the shared environment [17]. Results from these twin studies suggest that familial factors might be confounders in these associations. In fact, previous studies utilising the classic twin design have shown that heritability may explain 40–44% of the variance in liability to symptomatic non-specific back pain [18, 19], suggesting that genetics should be taken into account when studying the direct association between back pain and other risk factors [20].

Twin studies have been previously used to investigate prognosis, prevention and treatment of individuals with musculoskeletal condition, such as back pain [21]. However, the incidence of low birthweight (i.e. < 2500 g) in twin gestations is about 51.7% [22], which highlights birthweight as a potential confounder that needs to be better investigated. Furthermore, because birthweight is associated with changes in body composition across the lifespan [23], it is important to identify whether the association between anthropometric measures (BMI and weight gain) and back pain differs between those born with a low (< 2500 g) or full birthweight (≥ 2500 g) [24].

To the best of our knowledge, association of back pain with anthropometric measures such as birthweight, weight gain and BMI has not yet been investigated in adults. Thus, this study aims to investigate the association between weight gain and current BMI, and back pain in adults, and to determine whether the relationship differs in those who were born with low or full birthweight. By investigating the association between birthweight and back pain with a within-pair analysis (where one twin has back pain while the co-twin does not), we hope to improve the understanding of this health condition by more robustly addressing potential confounding factors such as genetics.

Methods

Design

This was a cross-sectional observational study with an embedded co-twin control design.

Study sample and data collection

Data from 2754 complete and incomplete twin pairs enrolled in this study (> 18 years old) were sourced from Twins Research Australia (TRA). TRA is a not-for-profit organisation with over 70,000 twins who have registered as volunteers for research (http://www.twins.org.au/) [25]. Data used in the present study are from January 2014 to December 2017. Study participants completed a Web-based self-reported questionnaire providing information on demographics, zygosity, anthropometrics and health history. Male and female monozygotic (MZ) and dizygotic (DZ) twins were included. Individuals were excluded if they reported cancer, musculoskeletal (e.g. spine fracture, fibromyalgia and ankylosing spondylitis), neurological or rheumatic conditions. Ethical approval was obtained from the Twins Research Australian Committee and The University of Sydney Human Research Ethics Committee (2018/053).

Assessment of outcome and predictors

The main outcome of this study was lifetime prevalence of back pain. The questionnaire asked: "Has a doctor ever diagnosed you with back pain/back problems?" Participants answering "yes" to this item were categorised as having a history of back pain.

Birthweight, weight gain and current BMI were the main predictors of back pain and were self-reported. Validity of

self-reported birthweight indicates a high degree of reliability among twins [26]. Birthweight was investigated as a continuous or binary variable (low birthweight, < 2500 g; full birthweight, ≥ 2500 g) [24]. BMI was calculated by dividing current body weight (kg) by the square of height (m) and used as a continuous variable. Annualised weight gain was calculated by subtracting birthweight (kg) from current weight (kg), divided by age (years) [27] and treated as a continuous variable.

Statistical analysis

The statistical analyses were conducted in three stages: total sample analysis; within-pair co-twin control analysis for MZ and DZ twins together; and within-pair case–control analysis separated by DZ and MZ (Fig. 1).

Potential confounders for the total sample analysis included age, sex, BMI, height, smoking habits, alcohol consumption, education, income and comorbidities (anxiety, depression, diabetes mellitus types 1 and 2). The same confounders were investigated in the within-pair twin case–control analyses. All the confounders listed above were treated as categorical variables, except for BMI, height and anxiety and depression. Participant's self-reported mental health (i.e. anxiety and depression) was assessed using the Kessler Psychological Distress Scale (K6) [28]. This is a 5-item questionnaire intended to yield a global measure of distress based on questions about anxiety and depressive

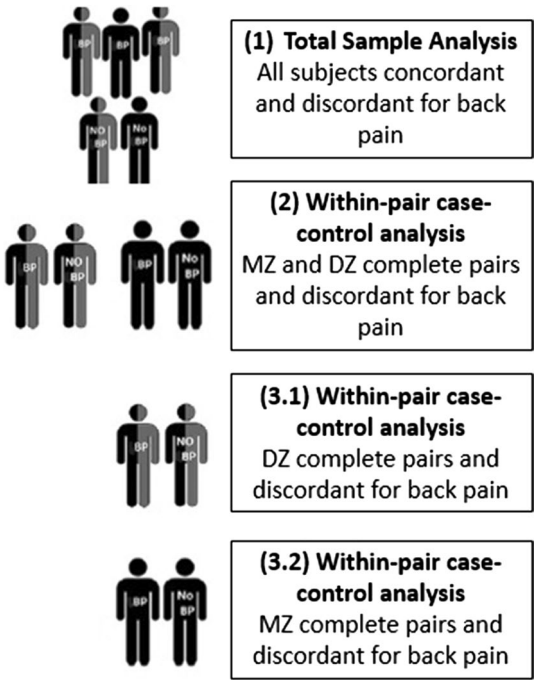

Fig. 1 Statistical analysis design. BP back pain, MZ monozygotic twins, DZ dizygotic twins

symptoms. The total score is composed of a sum of scores in all six domains and ranges from 5 to 30 (< 20—normal; 20–24—mild; 25–29—moderate; and < 30—severe) [28].

Total sample analysis

In the total sample analysis, all participants were included and twins were analysed as individuals rather than pairs. The association between predictors (birthweight, annualised weight gain and current BMI) and outcome (back pain) was assessed using logistic regression analysis. We used a robust estimator for all total sample analyses to account for possible non-independence of data (vce function on STATA). To select potentially confounding variables for inclusion in each of the multivariable models, we performed univariable linear or logistic regression analyses evaluating the association between both explanatory (birthweight, annualised weight gain and current BMI) and outcome (back pain) variables and the confounders, respectively. When birthweight was used as a binary variable (low or full birthweight), we performed logistic analyses. When a p value < 0.20 for the association between the possible confounders and both the explanatory and outcome variables was identified, this covariable was included in the relevant multivariate regression model. The confounders included in the multivariate model for the total sample analysis were retained in the subsequent DZ and MZ analyses, thus aiming to generate comparable results across the three analytical phases.

Within-pair twin case–control analyses

To control for genetics and early shared environmental factors on a potential association between anthropometric measures and the lifetime prevalence of back pain, we performed a within-pair co-twin control analysis on all complete twin pairs who were discordant for low back pain using conditional logistic regression models. We reported exact p values and described the strength of the association in the multivariable models and presented estimates as odds ratios (OR) and 95% confidence intervals (CI). Data analyses were accomplished using STATA software (version 14.0).

Results

Sample characteristics

Lifetime prevalence of back pain in the total sample of 2754 adults was estimated as 24.6% (95% CI 23.0–26.3) with the prevalence for MZ and DZ twins estimated as 23.9% (95% CI 22.0–25.7%) and 27.1% (95% CI 23.8–30.5), respectively. Among all twins, the mean age was 45 years with 75% and 22.8% of the twins being MZ and male, respectively

Table 1 Anthropometric data and lifestyle factors for participants with and without back pain and for the total sample

Variables	Back pain absent		Back pain present		Total	
	Mean (SD) or %	*n*	Mean (SD) or %	*n*	Mean (SD) or %	*n*
Age (years)	44.0 (15.2)	2075	49.6 (14.0)	679	45.4 (15.1)	2754
Gender (male)	21.9%	455	25.7%	175	22.8%	630
Height (m)	167.4 (11.6)	2042	166.9 (10.6)	671	167.0 (10.8)	2713
Weight (kg)	69.8 (16.5)	2023	73.2 (16.6)	658	70.6 (16.2)	2681
Smoking habits[a]	26.1%	541	31.6%	215	27.5%	756
Alcohol consumption[b]	30.2%	626	32.2%	219	30.7%	845
Education[c]	97.6%	2025	96.17%	653	97.2%	2678
Income[d]	26.9%	560	24.5%	167	26.4%	727
Diabetes						
Type 1	0.77%	16	0.44%	3	0.69%	19
Type 2	1.78%	37	1.77%	12	1.78%	49
Anxiety and depression[e]	8.74 (3.4)	2063	8.69 (3.3)	673	8.72 (3.4)	2736
DZ twins	23.9%	496	27.1%	1847	24.7%	680
MZ twins	76.1%	1579	72.9%	495	75.3%	2074

DZ dizygotic, *MZ* monozygotic, *BMI* body mass index, *SD* standard deviation; *n* number of individuals

[a]Percentage of those who smoke now

[b]Percentage of those who consumed more than four drinks in a day in the last 12 months (≥ 3 day a week)

[c]Percentage of those who completed any post-school qualifications (> 12 years)

[d]Percentage of those who receive annual gross income before tax less than $31,200

[e]Kessler Psychological Distress Scale (K6) total score

Table 2 Study sample characteristics of birthweight, annualised weight gain and current BMI measures for the total sample and cases and controls within a twin pair

Variables	Back pain absent		Back pain present		Total	
	Mean (SD)	*n*	Mean (SD) or %	*n*	Mean (SD) or %	*n*
Birthweight (g)						
Total sample	2355 (682.1)	1487	2335 (653.2)	495	2350 (674.9)	1982
DZ	2494 (723.9)	328	2446 (663.0)	118	2481 (708.0)	446
MZ	2315 (664.9)	1159	2301 (646.9)	377	2312 (680.3)	1536
Low birthweight (g)[a]						
Total sample	57.7%	859	58.2%	288	57.9%	1147
DZ	48.5%	159	49.1%	58	48.7%	217
MZ	60.3%	700	61.0%	230	60.6%	930
Annualised weight gain (kg/year)[b]						
Total sample	68.4 (15.7)	1468	72.4 (17.9)	490	69.4 (16.0)	1958
DZ	69.9 (14.7)	323	74.0 (14.9)	117	71.0 (14.9)	440
MZ	67.9 (15.9)	1145	71.9 (17.9)	373	68.9 (16.2)	1518
Current BMI (kg/m^2)						
Total sample	25.0 (6.1)	1993	26.0 (5.8)	651	25.2 (6.1)	2644
DZ	25.4 (8.1)	482	26.2 (5.0)	180	25.6 (7.4)	662
MZ	24.9 (5.4)	1511	25.9 (6.1)	471	25.1 (5.6)	1982

DZ dizygotic, MZ monozygotic, *BMI* body mass index, SD standard deviation; *n* number of individuals

[a]Low birthweight (< 2500 g)

[b]Annualised weight gain was calculated by subtracting birthweight (kg) from current weight (kg), divided by age (years)

(Table 1). Results for the mean and percentage of birthweight, annualised weight gain and current BMI for twins with and without back pain are described in Table 2.

Birthweight

Birthweight was not associated with lifetime prevalence of back pain in the total sample analysis (OR 0.99; 95% CI 0.99–1.00) or in any of the within-pair twin case–control analyses (Table 3). Furthermore, the results did not change after stratifying analyses by gender (Table 4).

Annualised weight gain

Annualised weight gain showed a small-magnitude relationship with lifetime prevalence of back pain in the total sample analysis in the crude (OR 1.01; 95% 1.00–1.01, $p < 0.001$) and adjusted (OR 1.02; 95% CI 1.00–1.03, $p = 0.001$) analysis. However, after adjustment for genetics and early shared environment in the within-pair analyses, the association did not persist (Table 3). Furthermore, the association did not change in the total sample analysis after stratifying the analysis by gender. MZ within-pair case–control analysis among males was not possible due to small sample size (Table 4). The association between annualised weight gain and back pain stratified by birthweight (low vs. full birthweight) showed a weak risk in the low (OR 1.01; 95% CI 1.00–1.02, $p = 0.006$) and full-birthweight group (OR 1.02; 95% CI 1.01–1.03, $p = 0.000$ in the total sample analysis (Table 5). The association was no longer significant after adjusting for genetics and early shared environment.

Current BMI

Current BMI was weakly associated with lifetime prevalence of back pain in the total sample crude analysis (OR 1.02; 95% CI 1.00–1.04, $p = 0.016$). However, the association disappeared after adjustment for confounders in the total sample analysis (OR 1.01; 95% CI 0.99–1.03, $p = 0.153$) (Table 3). After adjusting for genetic and early shared environment factors using data from 195 complete twin pairs discordant for back pain (44 DZ and 151 MZ pairs), BMI did not increase the risk of lifetime prevalence of back pain. When the analysis was stratified by gender, the results remained the same among females (OR 1.04; CI 1.02–1.06, $p = 0.000$), although after adjustment for genetics and shared environment the association disappeared. Furthermore, the association between current BMI and back pain stratified by birthweight (low vs. full birthweight) showed a positive weak risk in the low-birthweight group (OR 1.03; 95% CI 1.00–1.06, $p = 0.022$) compared to the full-birthweight group (OR 1.03;

Table 3 Total sample analysis and within-pair co-twin control analysis for back pain

Multivariate models	OR (95% CI)	*p* value	*N*
Birthweight (g)			
Total sample			
Crude	0.99 (0.99–1.00)	0.591	1982
Adjusted[a]	0.99 (0.99–1.00)	0.345	1982
MZ and DZ pairs			
Crude	1.00 (0.99–1.00)	0.597	372
Adjusted	–	–	–
DZ pairs			
Crude	0.99 (0.99–1.00)	0.592	88
Adjusted	–	–	–
MZ pairs			
Crude	1.00 (0.99–1.00)	0.300	284
Adjusted	–	–	–
Annualised weight gain (kg/years)			
Total sample			
Crude	1.01 (1.00–1.01)	0.000*	1958
Adjusted[b]	1.02 (1.00–1.03)	0.001*	488
DZ and MZ pairs			
Crude	1.00 (0.98–1.03)	0.506	370
Adjusted[c]	1.00 (0.97–1.03)	0.440	314
DZ pairs			
Crude	1.01 (0.97–1.04)	0.525	90
Adjusted[§]	–	–	–
MZ pairs			
Crude	1.00 (0.97–1.03)	0.762	280
Adjusted[d]	0.98 (0.94–1.03)	0.641	232
Current BMI (kg/m^2)			
Total sample			
Crude	1.02 (1.00–1.05)	0.016*	2644
Adjusted[e]	1.01 (0.99–1.04)	0.153	2575
DZ and MZ pairs			
Crude	1.03 (0.98–1.08)	0.193	576
Adjusted	–	–	–
DZ pairs			
Crude	1.06 (0.98–1.14)	0.092	178
Adjusted[f]	1.06 (0.98–1.14)	0.92	178
MZ pairs			
Crude	0.99 (0.92–1.07)	0.936	398
Adjusted[g]	0.99 (0.91–1.07)	0.769	384

OR odds ratio; *CI* confidence interval; *MZ* monozygotic; *DZ* dizygotic; *n* number of individuals in each analytical step; *$p < 0.05$

[a]Adjusted for age and gender

[b]Adjusted for age, gender, smoking, education, income, alcohol and height

[c]Adjusted for alcohol

[d]Adjusted for alcohol, diabetes types 1 and 2, height, anxiety and depression

[e]Adjusted for age, gender, education and income

[f]Adjusted for gender

[g]Adjusted for education and income

[§]Analysis not performed due to small sample size

Table 4 Total sample analysis and within-pair co-twin control analysis for back pain by gender

Multivariate models	Male			Female		
	OR (95% CI)	*p* value	*n*	OR (95% CI)	*p* value	*n*
Birthweight (g)						
Total sample						
Crude	1.00 (0.99–1.00)	0.477	327	0.99 (0.99–1.00)	0.239	1655
Adjusted[a]	1.00 (0.99–1.00)	0.658	327	0.99 (0.99–1.00)	0.189	1655
DZ and MZ pairs						
Crude	1.00 (0.99–1.00)	0.477	42	0.99 (0.99–1.00)	0.952	298
Adjusted	–	–	–	–	–	–
DZ pairs						
Crude	0.99 (0.98–1.00)	0.568	4	1.00 (0.99–1.00)	0.893	52
Adjusted	–	–	–	–	–	–
MZ pairs						
Crude	1.01 (0.99–1.00)	0.098	38	0.99 (0.99–1.00)	0.856	246
Adjusted	–	–	–	–	–	–
Annualised weight gain (kg/years)						
Total sample						
Crude	1.02 (1.00–1.03)	0.022*	328	1.01 (1.00–1.02)	0.004*	1630
Adjusted[b]	1.02 (0.99–1.06)	0.05	189	1.01 (1.00–1.03)	0.04*	372
DZ and MZ pairs						
Crude	0.99 (0.95–1.04)	0.962	42	1.01 (0.97–1.04)	0.448	296
Adjusted[§]	–	–	–	–	–	–
DZ pairs						
Crude[§]	–	–	–	1.03 (0.98–1.08)	0.225	54
Adjusted[§]	–	–	–	–	–	–
MZ pairs						
Crude	1.01 (0.96–1.06)	0.558	38	0.99 (0.94–1.04)	0.845	242
Adjusted[c]	1.01 (0.85–1.06)	0.838	34	0.98 (0.93–1.04)	0.702	198
Current BMI (kg/m^2)						
Total sample						
Crude	0.99 (0.97–1.01)	0.824	616	1.04 (1.02–1.06)	0.000*	2028
Adjusted[d]	0.98 (0.96–1.01)	0.323	599	1.03 (1.01–1.05)	0.002*	1976
DZ and MZ pairs						
Crude	0.98 (0.87–1.10)	0.790	114	1.05 (0.98–1.11)	0.135	402
Adjusted	–	–	–	–	–	–
DZ pairs						
Crude	0.89 (0.67–1.19)	0.444	30	1.10 (0.99–1.22)	0.056	88
Adjusted[e]	0.97 (0.69–1.36)	0.887	30	1.10 (0.99–1.23)	0.057	88
MZ pairs						
Crude	1.01 (0.88–1.14)	0.931	84	0.99 (0.90–1.10)	0.871	314
Adjusted[f]	1.01 (0.88–1.14)	0.931	84	0.98 (0.89–1.09)	0.829	302

DZ dizygotic; *n* number of individuals in each analytical step

[a]Adjusted for age

[b]Adjusted for age, smoking, education, income, alcohol and height

[c]Adjusted for alcohol, diabetes types 1 and 2, height, anxiety and depression

[d]Adjusted for age and education

[e]Adjusted for income

[f]Adjusted for education and income

[§]Analysis not performed due to small sample size

Table 5 Total sample analysis and within-pair twin case–control analysis for back pain by low or full birthweight

Multivariate models	Low birthweight			Full birthweight		
	OR (95% CI)	*p* value	*n*	OR (95% CI)	*p* value	*n*
Annualised weight gain (kg/year)						
Total sample						
Crude	1.01 (1.00–1.02)	0.006*	1134	1.02 (1.01–1.03)	0.000*	821
Adjusted[a]	1.00 (0.98–1.03)	0.596	263	1.02 (1.01–1.04)	0.002	208
DZ and MZ pairs						
Crude	1.00 (0.96–1.04)	0.771	176	1.02 (0.98–1.07)	0.259	94
Adjusted[b]	0.97 (0.92–1.02)	0.343	148	1.02 (0.97–1.07)	0.376	84
DZ pairs						
Crude	1.02 (0.93–1.11)	0.623	26	1.02 (0.97–1.07)	0.427	34
Adjusted[§]	–	–	–	–	–	–
MZ pairs						
Crude	1.00 (0.96–1.04)	0.913	150	1.04 (0.95–1.13)	0.384	60
Adjusted[c]	0.97 (0.90–1.04)	0.422	124	1.02 (0.92–1.14)	0.624	52
Current adult BMI (kg/m^2)						
Total sample						
Crude	1.03 (1.00–1.06)	0.022*	1121	1.03 (0.97–1.08)	0.334	817
Adjusted[d]	1.01 (0.96–1.07)	0.593	290	1.05 (1.00–1.10)	0.026	233
DZ and MZ pairs						
Crude	0.99 (0.89–1.11)	0.960	174	1.06 (0.93–1.21)	0.348	94
Adjusted	–	–	–	–	–	–
DZ pairs						
Crude	1.12 (0.76–1.65)	0.553	26	1.09 (0.92–1.29)	0.296	34
Adjusted[e]	1.11 (0.75–1.63)	0.582	26	1.09 (0.92–1.63)	0.582	26
MZ pairs						
Crude	0.98 (0.80–1.10)	0.806	148	1.01 (0.81–1.26)	0.840	60
Adjusted[f]	0.97 (0.86–1.10)	0.725	144	1.02 (0.82–1.27)	0.848	58

OR odds ratio; *CI* confidence interval; *MZ* monozygotic; *$p < 0.05$

DZ—dizygotic; n—number of individuals in each analytical step

[a]Adjusted for age, smoking, education, income, alcohol and height

[b]Adjusted for alcohol

[c]Adjusted for alcohol, diabetes types 1 and 2, height, anxiety and depression

[d]Adjusted for age and education

[e]Adjusted for income

[f]Adjusted for education and income

[§]Analysis not performed due to small sample size

95% CI 0.97–1.08, $p = 0.334$) in the total sample analysis (Table 5). However, after adjusting for confounders in the total sample and within-pair case–control analysis the association disappeared. Stratification by gender for the association between current BMI and back pain by birthweight category was not possible due to small sample size.

Discussion

To the best of our knowledge, this is the first study investigating the associations between birthweight, annualised weight gain and BMI (exposures) and back pain (outcome) using twin design. We demonstrate that lifetime prevalence of back pain was not associated with birthweight, neither in the total sample analysis nor after adjustment for genetic and early shared factors in both MZ and DZ adult twin pairs discordant for low back pain. Annualised weight gain and BMI showed small-magnitude associations with back pain in the total sample analysis; however, the associations disappeared after adjusting for genetics and the early shared environment. These results indicate that a direct link between back pain and anthropometric measures earlier (birthweight) and later (annualised weight gain and adult BMI) in life is unlikely.

Birthweight and back pain

Ours results did not reveal any association between birthweight and lifetime prevalence of back pain either in the total sample analysis or in any of the co-twin control analyses. These results were in agreement with previous cross-sectional studies in which birthweight was not associated with musculoskeletal pain in later life after adjustment for confounders [5, 6, 29, 30].

A study investigating the association between birthweight and low back pain in a cohort of adolescent twins [17] showed a stronger association between birthweight and back pain in males (than females). However, this association was no longer significant after adjustment for genetics and early-life environment [13]. Genetics and early environment are thus potential confounders that need to be taken into account when investigating the association between back pain and risk factors [18]. The lack of association between birthweight and lifetime prevalence of back pain was therefore somewhat anticipated in the present study. It is already known that approximately 50% of twins are born with low birthweight [22]; hence, it is important to highlight that the back pain prevalence in our twin cohort (25%) is similar to that in the general population which ranges from to 19.3 to 48% [31–33]. Furthermore, the birthweight between those twins reporting back pain or not did not vary substantially in the present study.

Annualised weight gain and BMI

Our results showed that lifetime prevalence of back pain was associated with annualised weight gain and BMI in the total sample analysis (i.e. when not controlling for familial factors). When stratified by gender in the total sample analysis, the associations did not persist among males. The associations were small and weak and were in agreement with previous cross-sectional studies that investigated low back pain and BMI.

Because high BMI and back pain result from complex interactions and traits influenced by genetics and environment, it is crucial to adjust the analysis for familial factors. Thus, after adjustment for genetics and environment using a robust co-twin control analysis, the significant association between the anthropometric measures and back pain did not remain significant. Although there is no study looking at current BMI and back pain association, our findings are in agreement with previous cross-sectional and longitudinal studies investigating the association between body composition (e.g. BMI and body fat distribution) and low back pain using a co-twin control design in adults [16, 20, 34]. Taken together, these studies indicate that genetics and early shared environment are potential confounders when looking at the association between anthropometric measures and low back pain.

An adverse intrauterine environment, as reflected by low birthweight, is associated with more subcutaneous and abdominal fat and less lean body mass in adulthood [35]. Furthermore, twin pairs tend to present with differences in body composition across the lifespan depending on their weight at birth. Thus, we decided to investigate whether the associations between anthropometric measures and back pain differ between those who were born with a low or full birthweight [23]. Regarding annualised weight gain and back pain, our results showed a similar association in the low-birthweight group compared to the full-birthweight group. For the BMI and back pain relationship, there was a positive association in the low-birthweight group, although the OR values did not differ between the groups (i.e. low vs. full birthweight).

Strengths and limitations

Life-course research has drawn attention to early-life risk factors such as birthweight that influence people's health trajectory and increase the odds of future pain conditions. However, early exposures are rarely investigated in the back pain field. However, our results are subject to some limitations: (1) this is a cross-sectional study which limits inference on causation between birthweight and back pain; (2) other confounders were not available to be included (e.g. physical activity engagement); (3) the present data might be influenced by the definition of the main outcome ("back pain present"), considering that "back pain" may vary in terms of severity, frequency, time and period; and (4) our respondents were typically females of higher education and income status and hence may not necessarily reflect risk for all twins with/without back pain. We also acknowledge that (5) self-reported weight and height may have suffered from reporting bias in our study [36] and (6) the smaller sample size especially in the DZ co-twin control analyses may have affected the power to detect a significant association between anthropometric related measures and back pain. Future studies in this field should address these limitations.

Conclusion

Birthweight was not associated with back pain in our study, neither in the total sample analysis nor after adjustment for genetics and early shared environment in the within-pair analyses. Annualised weight gain and current BMI showed small-magnitude associations with back pain prevalence in the total sample analysis and did not differ in those born with low or full birthweight. However, the associations were

weak and appeared to be confounded by genetics and early shared environmental factors. Our results suggest that a direct link between these predictors and back pain in adults is uncertain. We believe that positive associations reported in previous studies might be related to potential perinatal and residual confounding. Considering the life-course approach on the back pain development, research should focus on other potential modifiable risk factors to progress this field.

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Compliance with ethical standards

Conflict of interest None.

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