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Parent-offspring association of chronic back pain and the role of physical activity.

Family-linkage data from the HUNT-study

Master's thesis in Human Movement Science
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Abstract:

Chronic musculoskeletal pain (CMP) is an increasing health problem in the western society, and knowledge on the accumulation within families is still uncertain. The main objective of this study was to investigate the association of CMP in the spinal region between parents and their adult offspring, specifically in the neck, shoulder and low back. Additionally the possible modifying role of physical activity (PA) on this association was examined. Data from the population based HUNT study in Norway provided 11247 subjects. Logistic regression was used to calculate the adjusted odds ratio (OR) with a 95% confidence interval (CI). Results showed a moderate increase in OR among all groups, maternal influence ranging from 1.36 (95%CI=1.27 – 1.46) to 1.44 (95% CI = 1.34 – 1.54), and paternal influence from 1.22 (95%CI=1.13 – 1.33) to 1.43 (95%CI=1.28 – 1.59). When stratified on offspring PA a trend was observed showing inactive subjects with a slightly higher OR of back pain than the active subjects, but this difference was not statistically significant. In conclusion there appears to be an association between parental and offspring CMP in the spinal region, and the modifying effect of PA is still uncertain.

Oppsummering:

Kroniske muskelskjelettsmerter er et økende problem i den vestlige verden, og hvorvidt dette akkumulerer innad i familier er fortsatt usikkert. Denne studien har som hovedmål å finne ut av om det er en sammenheng mellom smerte i rygg-regionen hos foreldre og deres voksne barns smerte. Studien ser spesifikt på korsrygg-, skulder- og nakkesmerte. I tillegg til dette undersøkes det om fysisk aktivitet (FA) kan påvirke denne sammenhengen. Dataen er hentet fra helseundersøkelsen i Nord-Trøndelag (HUNT), og disse dataene ga 11247 subjekter. Analysene som ble gjort var justerte logistisk regresjonsanalyser med et konfidensintervall (KI) på 95%. Resultatene viste en moderat sammenheng mellom både mødre og fedre, og deres barn. Odds ratioen (OR) var henholdsvis fra 1.36 (95%KI=1.27 – 1.46) til 1.44 (95% KI = 1.34 – 1.54) og fra 1.22 (95%KI=1.13 – 1.33) til 1.43 (95%KI=1.28 – 1.59). Stratifiserte analyser utført på barnas FA viste en trend mot at inaktive subjekter hadde en høyere OR til smerte enn de aktive, men disse resultatene var ikke signifikante. Konklusjonen var at det er en sammenheng mellom foreldre og barns ryggsmerter, og at det fortsatt er usikkert hvorvidt FA kan modifisere denne effekten.

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Table of Contents

1. Introduction

Chronic musculoskeletal pain (CMP) is one of the greatest health issues in the western society today. The prevalence of CMP is high, and it appears to be increasing [1]. Woolf et. al. wrote in a review in 2003 that musculoskeletal disorders (MD) were the most common cause of severe long term pain and physical disability [2], and in 2012 they wrote another review discussing the need to address the problem of MD [3]. MacBeth and Jones found that 20-33% of the general population will experience shoulder pain (SP) and 51-84% will experience low back pain (LBP) throughout their lifetime[1]. In 2012 Hoy et. al. found that low back pain continued to be a severe problem [4]. CMP causes great economic challenges for the society through paid leave of absence, medical care, and rehabilitation. In Norway 2010, a total of 41% of work related paid sick leave was caused by back pain. In the same report, Ihlebaek estimated the annual cost of MD were 37-44 billion Norwegian kroners [5].

CMP can afflict most joints in the body, and one of the most common locations is back pain [1,2]. LBP can be classified as "specific" or "non-specific" (no clear pathological cause). Woolf et. al. estimates about 90% of LBP cases were non-specific [2]. Specific causes to CMP include arthritis/osteoarthritis, rheumatoid arthritis and fractures. These are seen as specific causes, as they have an established definition and clear cause [6]. The impact of CMP is expected to increase dramatically [1,3,6]. Identifying possible risk factors could help prevent this effect.

The increase of CMP are hypothesized to be due to the predicted ageing of the world's population, and changes in lifestyle factors, such as increased obesity and low physical activity (PA) as a consequence of the urbanization and motorization of the world [1,2]. As a result, finding ways to prevent these conditions become more pressing. Known risk factors are age, increased body weight [7,8], low socioeconomic status [9], psychological disorders (anxiety and depression) [1,2,10], high physical workload [2,11-13], and low physical activity [14-16]. A large population-based study by Yoo et. al. found the effect of elevated BMI to be strongest among women, and suggest metabolic syndrome to be a possible risk factor [7]. Seaman supports this theory, suggesting the systemic pro-inflammatory metabolic consequences as a possible mechanism [8]. Das UN found that exercise has an anti-inflammatory effect [17], and Nilsen et. al. propose this anti-inflammatory effect could be a possible mechanism in preventing LBP [14].

Woolf et. al. states that muscle weakness in back and abdominal muscles are risk factors of LBP [2]. These factors indicate that PA could be a way to prevent back pain. A review by Ferreira et. al. found that there is an overall lack of studies examining PA as a factor in the development or prevention of LBP [18]. More information on whether or not PA can prevent LBP is needed, because risk factors in the form of lifestyle factors are possible to improve. This could reduce the predicted increase in CMP.

To identify the population at risk of CMP, studies have been conducted to investigate whether back pain accumulates within families. These have included twin studies and family studies, all of which examine heritability in the form of a genetic and environmental component [19-26]. Twin studies have shown that the relative influence by genetic or environmental factors might depend on the person's age. Genetic factors have been seen to influence young and middle aged adults to a greater extent [25] than in children and elderly [20,24,27]. Family-studies have found a parent-offspring association of back pain [21-23]. As the incidence of back pain is greater with increasing age, and the influence of heritability is strongest among young to middle aged subjects, studies where the offspring is adult could yield more knowledge about this association.

Previous studies have shown associations between back pain and physical inactivity [14], and thus physically active could influence familial associations in back pain. This study aims to investigate if back pain, defined as low back pain (LBP), shoulder pain (SP) and neck pain (NP), accumulates within families. More specifically, we will examine the association in back pain between parents and their adult offspring. Additionally, we investigate if physical activity (PA) can modify this effect.

2. Methods

2.1 Subjects

Information on parents and their adult offspring was acquired through the HUNT study. The HUNT study is a population-based health study conducted in Nord-Trøndelag, Norway. The study has been carried out in three consecutive surveys: in 1984-1986 (HUNT1), 1995-1998 (HUNT2) and finally in 2006-2008 (HUNT3). In all three surveys, all residents aged 20 years and older were invited to participate. Information on lifestyle and health related factors were collected by questionnaires and clinical examination. The clinical examinations included anthropometric data, blood pressure and a venous blood sample. Questionnaires became more detailed in later surveys, thus information retrieved varies from HUNT1 to HUNT3. More information about selection procedures, participation and questionnaires used in the HUNT studies can be found at http://www.hunt.ntnu.no.

As no information on musculoskeletal pain was obtained at HUNT1, the current study is based on information from HUNT 2 and HUNT3. 93 898 people were invited to participate in HUNT2, of which 65 237 (70%) attended the survey. In HUNT3 93 860 people were invited, and 50 807 (54%) participated. Parental data was gathered using HUNT2 data, and offspring data was found using HUNT3 data. Using the subjects' personal identification number, and information from the Family Registry at Statistics Norway, a linkage between parents and offspring in the HUNT study was established. For the purpose of the present study all offspring with linkage to one parent (either mother or father) was included. This resulted in 11 247 subjects. Each survey was voluntary, and participants signed a written consent. The Regional Committee for Ethics in Medical Research approved the study (ref,no 2011/1455/REK midt).

2.2 Variables

As part of the HUNT study, each participant had been asked questions about musculoskeletal pain. These questions were similar in both HUNT2 and HUNT3, and were conveyed as: "During the last year, have you had pain and/or stiffness in your muscles and limbs that has lasted for at least 3 consecutive months? ". If the subject responded, "yes" to this question, a follow-up question was asked about location. Here participants could report pain in the neck, shoulders and lower back among others. Using answers from these questions, new variables on musculoskeletal pain were computed. If the subject reported having experienced pain the last year, location

of the pain was taken into consideration. This is how variables on NP, SP and LBP were made for both parents and offspring. Subjects reporting no pain the last year were used as a control group.

Parental information on leisure time physical activity (LTPA) was obtained from HUNT2 using the following question: ”How much of your leisure time have you been physically active during the last year? (Think of a weekly average for the year. Your commute to work counts as leisure time.)”. The participants should report their weakly average of both “light” (not sweat or out of breath) and “hard” activity (sweat and/or out of breath” given the following response options: “None”, “less than 1”, “1-2”, or “3 or more” hours per week. This information was combined in a summary measure of physical activity with the following four categories (1: No light or hard activity, 2: <1 hour light and no hard activity, 3: ≥3 hours light and/or <1 hour hard activity, 4: ≥1 hour hard and any light activity. Offspring information on physical activity was obtained from HUNT3 using the following question: “How often do you exercise? (On the average). By exercise we mean going for walks, skiing, swimming and working out/sports, etc.”. Answers were given as: “Never”, “less than once a week”, “once a week”, “2-3 times a week”, or “nearly every day”. Participants who reported to exercise once a week or more were also asked about their average intensity and duration of activity. How hard do you exercise (average)? ”. Answer alternatives were: “I take it easy”, “I don’t get out of breath or break a sweat”, “I push myself until I’m out of breath and break into a sweat”, or “I practically exhaust myself”. The third question was formulated as: ”For how long do you exercise each time (average)? ”. Alternative answers were: “Less than 15 minutes”, “15-29 minutes”, “30 min.-1 hour”, or “more than 1 hour”. The information on frequency, intensity, and duration was then combined in a summary score of physical activity according to the following equation: 1/5 x Frequency + 1/3 x Intensity + 3/4 x Duration. The resulting sex-specific score was then dichotomized according to the median, and used to classify offspring in four groups: 1: No activity, 2: Low (< 1 per week), 3: Medium (< median score), 4: High (≥ median score). For the purpose of the stratified analyses, the two first and the two latter categories were collapsed into Inactive and Active, respectively.

The final variables that were constructed combined the active/inactive variables from each parent separately with the offspring variable into: 1)"inactive-inactive", 2)"active-inactive, 3)"inactive-active", and 4)"active-active".

Additional parental factors included body mass index (BMI), psychological wellbeing and PA during work. Data on body height (to nearest centimeter) and body mass (to the nearest kilogram) were used to calculate body mass index (BMI) as mass divided by the square of height (kg/m^2). Psychological wellbeing was assessed from the question: "Thinking about your life at the moment, would you say that you by and large are satisfied with life, or are you mostly dissatisfied?" Answers were given as: 1)"very satisfied", 2)"satisfied", 3)"somewhat satisfied", 4)"neither satisfied nor dissatisfied", 5)"quite dissatisfied", 6)"dissatisfied", and 7)"very dissatisfied". The participants were classified into two groups: 1)"satisfied", and 2)"dissatisfied". The variable representing PA during work (WPA) was based on the question: "is your work physically strenuous?" Answers were given as: "yes, almost always", "very often", "very rarely", or "never or almost never". In addition to the four categories already in place, a new category was added to represent missing data.

2.3 Statistical Analysis

Descriptive statistics were done on parent and offspring age: mean and range, number of participants in each BMI category, mean BMI, number of participants with or without pain the last year, number of participants with LBP, SP and NP, and number of participants in each PA group ("no activity", "low", "medium", and "high").

Odds ratios (ORs) with 95% confidence intervals (CIs) for the association between parental and offspring's pain was computed using bivariate logistic regression analysis. The main analyses were done separately for father-offspring and mother-offspring associations. General pain and pain at certain locations (i.e. NP, SP, or LBP) were analyzed separately. Further analysis of whether the offspring´s PA had an impact on the parent-offspring associations was done by stratifying the data on offspring's PA ("inactive", "active"). Moreover, a product term of parental pain and offspring PA was included in the regression model to assess statistical interaction. We also examined possible effect modification by a variable combining parental and offspring PA (i.e., four category with possible combinations of parental and offspring being "inactive" or "active".

Analyzing different variables independently identified potential confounders. Adjustment for confounders was done using parental data. Variables that were taken into consideration were: age, BMI, PA, WPA, the opposite parents pain, and psychological wellbeing. All of the above showed slight alterations on the results, and were adjusted for in the statistical analysis. Parental PA was not adjusted for in analysis on children's PA relative to parent`s PA, and its modifying effect on parent – offspring pain association.

3. Results

3.1 Descriptive analyses:

Descriptive statistics show that mean age among the offspring was 48 years (table 3.1). Mean age among mothers was 61.5 and fathers 61.6 years. 48% of the children, 57% of the mothers, and 51% of the fathers experienced stiffness or pain during the last year. Parental musculoskeletal pain was most common among mothers. Shoulder pain was the most common of pain locations. Among the children 22% experienced shoulder pain. All subject groups had LBP as the most uncommon occurring back pain location. Fathers reported the lowest percentage (7.5%) within this category. Average BMI in all subject groups was within the overweight category (26.8 – 27.2). Lowest mean BMI was observed among fathers, even though this group showed the highest percentage within the overweight group (53%). Within both parental groups the highest percentage reported a low LTPA level (41% of mothers, and 38% of fathers), and 56% of the offspring group reported a high activity level.

3.2 Parent – offspring association analyses:

Logistic regression analyses showed an association between parental and offspring pain regardless of gender (table 3.2). The association remained after adjusting for possible confounders. The offspring OR for overall pain associated with maternal overall pain was 1.37 (CI=1.28 – 1.46), whereas paternal overall pain gave an OR of 1.24 (CI=1.16 – 1.33). Association of SP in both parental groups was the only category that did not show a stronger association than overall pain. Mother-offspring association was greater than father-offspring association for all pain locations except LBP. The odds ratio of offspring developing LBP if either of their parents had experienced back pain were 1.43 (CI = 1.32 – 1.53 for maternal OR, CI 95% = 1.28 – 1.59 for paternal OR).

Table 3.1: Descriptive statistics:

Variable	Children	Mothers	Fathers
Mean Age	47.95	61.49	61.61
Range Age	18-94	23-99	27-100
N BMI			
<18.5	211	145	62
18.5-24.9	11168	7780	5192
24.9-29.9	14565	9397	9221
>30	7398	5530	2824
Mean BMI	27.11	27.21	26.83
N PLY*	12613	13142	8845
N no PLY*	13200	9779	8505
N LBP*	6174	6771	2509
N NP*	6872	7459	4532
N SP*	7285	8065	5172
N PA			
No activity	1530	4918	3112
Low	5893	6812	4813
Medium	7044	4324	3298
High	18198	648	1516

*** PLY = pain last year, LBP = low back pain, NP = neck pain, SP = shoulder pain**

3.3 Analyses of offspring PA`s modifying effect on pain association:

Stratified regression analyses showed offspring PA had a weak modifying effect on pain association (table 3.3). The effect remained after adjusting for possible confounders. Offspring reporting an inactive lifestyle had a higher OR of developing back pain, than the active group, except for father-offspring associations of SP. The OR for SP in inactive children was 1.05 (CI 95% = 0.88 – 1.25) if their fathers had SP, whereas the OR in active children was 1.26 (CI 95% = 1.15 – 1.39). The largest association effect was seen for LBP among inactive children, with and OR of 1.70 (CI 95% = 1.36 – 2.12) if the father reported LBP. Interaction analyses of parental pain and PA gave a P-value of 0.43 for father-offspring overall pain associations, and p= 0.61 for the equivalent analyses for mother-offspring association. Interaction analyses on the separate pain locations showed p-values ranging from 0.06 – 0.89 for father-offspring associations and p= 0.17 – 0.28 for mother-offspring associations. Thus, there were no statistically significant interactions with offspring PA.

Table 3.2: Parent – offspring association analyses:

Variable	Cases/noncases	OR crude	OR adjusted*	95% CI
Pain Mother				
No	4399/4879	1.00	1.00	
Yes	3117/5039	1.46	1.37	1.28 – 1.46
SP** Mother				
No	4399/2923	1.00	1.00	
Yes	1722/1885	1.32	1.36	1.27 – 1.46
NP** Mother				
No	4399/2642	1.00	1.00	
Yes	1625/1735	1.41	1.44	1.34 – 1.54
LBP** Mother				
No	4399/2415	1.00	1.00	
Yes	1457/1450	1.46	1.43	1.32 – 1.53
Pain Father				
No	3748/3506	1.00	1.00	
Yes	2700/3197	1.28	1.24	1.16 – 1.33
SP Father				
No	3748/2007	1.00	1.00	
Yes	1478/1104	1.22	1.22	1.13 – 1.33
NP Father				
No	3748/1742	1.00	1.00	
Yes	1408/957	1.27	1.28	1.17 – 1.39
LBP Father				
No	3748/1626	1.00	1.00	
Yes	1217/783	1.42	1.43	1.28 – 1.59

*** Age, BMI, PA, work PA, the opposite parent`s pain, psychological illness.**

**** LBP = low back pain, NP = neck pain, SP = shoulder pain**

Table 3.3: Stratified analyses of children`s PA and parent – offspring pain association:

		Inactive				Active		
Variable	Cases/noncases	OR crude	OR adjusted*	CI 95%	Cases/noncases	OR crude	OR adjusted*	CI 95%
Pain Mother								
No	815/990	1.00	1.00		3507/3795	1.00	1.00	
Yes	624/1127	1.48	1.42	1.23 – 1.64	2440/3826	1.45	1.35	1.26 – 1.45
SP** Mother								
No	4327/1441	1.00	1.00		15168/4615	1.00	1.00	
Yes	1189/466	1.40	1.48	1.28 – 1.71	4071/1388	1.29	1.32	1.22 – 1.43
NP** Mother								
No	4567/1374	1.00	1.00		15773/4239	1.00	1.00	
Yes	1068/414	1.53	1.57	1.35 – 1.82	3938/1292	1.38	1.41	1.30 – 1.53
LBP** Mother								
No	4813/1248	1.00	1.00		16607/3973	1.00	1.00	
Yes	1012/350	1.56	1.56	1.34 – 1.82	3590/1072	1.43	1.38	1.27 – 1.51
Pain Father								
No	704/709	1.00	1.00		2983/2737	1.00	1.00	
Yes	550/724	1.33	1.32	1.13 – 1.55	2116/2410	1.25	1.21	1.11 – 1.31
SP Father								
No	4829/939	1.00	1.00		16726/3057	1.00	1.00	
Yes	1414/241	1.03	1.05	0.88 – 1.25	4621/838	1.27	1.26	1.15 – 1.39
NP Father								
No	5119/822	1.00	1.00		17324/2688	1.00	1.00	
Yes	1267/215	1.25	1.26	1.05 – 1.52	4512/718	1.26	1.26	1.14 – 1.40
LBP Father								
No	5582/478	1.00	1.00		19090/1490	1.00	1.00	
Yes	1226/136	1.64	1.70	1.36 – 2.12	4302/360	1.34	1.33	1.17 – 1.51

*** Age, BMI, PA, work PA, the opposite parent`s pain, psychological illness.**

*** * LBP = low back pain, NP = neck pain, SP = shoulder pain**

3.4 Analysis on children's PA relative to parental PA:

Analysis using the variable combining children and parental PA on parent – offspring pain are shown in table 3.4. Overall, there were no large differences between the different PA groups. However, a somewhat stronger association was observed among inactive offspring with active parents 1.70 (CI 95%= 1.23 – 2.34) and 1.66 (CI 95% =1.20 – 2.28)).

Table 3.4 Stratified analyses on parental PA relative to offspring PA:

	Overall Pain mother			Overall Pain father		
	No	Yes	CI 95%	No	Yes	CI 95%
Inactive O − Inactive P**	1.00	1.36	1.11 – 1.67	1.00	1.25	0.99 – 1.57
Active O − Inactive P**	1.00	1.26	1.14 – 1.39	1.00	1.22	1.10 – 1.38
Inactive O − Active P**	1.00	1.70	1.23 – 2.34	1.00	1.66	1.20 – 2.28
Active O − Active P**	1.00	1.47	1.27 – 1.71	1.00	1.18	1.01 – 1.37

*** Adjusted for age, BMI, work PA, the opposite parent`s pain, psychological illness.**
**** O = offspring, P = parents**

4. Discussion

The main finding of this population-based cross-sectional family linkage study is an association in overall chronic pain between parents and their adult offspring. These associations were largely similar between mothers and fathers and across subgroups of back pain. The strongest parent-offspring associations were found for low back pain. Although no statistically significant interaction was observed, there was a tendency that analyses stratified by offspring LTPA showed somewhat weaker parent-offspring associations if offspring were classified as physically active compared to if they were inactive.

4.1 Association between parental pain and their adult offspring`s pain:

Three previous studies of this nature have been conducted [21-23]. These three all investigate family linkage of chronic musculoskeletal pain, and they use data collected from parents and their adult offspring. In all three studies parents and offspring answered individual questionnaires. Bruehl et. al. found that in order to achieve acceptable validity in family-linkage studies, independent reports from both parents and offspring are necessary [28]. All studies support the theory that CMP accumulates within families [21-23]. Two of these studies were conducted using HUNT2 and HUNT3 data [21, 22]. As expected, these studies showed close to identical results to this one. The current study found a small difference between the results from mother-offspring and father-offspring associations toward back pain. Lier et. al. stratified their analyses by offspring and parental gender, and their results showed that neither modified the parent-offspring association on back pain [21-22]. This is consistent with the results of the present study, although as these studies are based on the same data as the present study, they do not provide any additional support to the findings of the current study. Hocking et. al. conducted their study using data collected in the Scottish Family Health Study and categorizing their chronic pain data through the Chronic Pain Grade[23]. They found age and sex to be the strongest associated covariates, and though Lier et. al found these variables to be of no consequence, they found them to have a stronger association than other confounders (BMI, PA, socioeconomic status) [21-22]. The results of Hocking et. al.´s study suggest that genetic effects on chronic pain are at least as important as the measured environmental factors to the development of chronic pain [23].

This is supported by twin studies investigating the heritability of CMP, although the relative difference is disputed. Nyman et. al. found that genetics influence concurrent LBP and neck/shoulder pain to a greater extent than environmental factors (60% vs 40%) [25]. Fejer et. al. on the other hand show the opposite in their results, where the genetic component was 45% and non-shared environmental factor accounted for 55% [24], but the difference in results from these studies is inconsequential. Both studies show that there could be a genetic component to back pain, whether this is an independent component specific to back pain or if it is due to the genetic component to pain sensitivity is uncertain. Pollard et al, suggest that pain behavior can be learned from other family members [29]. If so, parental behavior could contribute the parental-offspring association observed in the present study. In contrast, Jones et. al. found that pain behavior is not learned, but can be attributed to individual factors and the social environment [30]. Pain perception is a complex process that could be influenced by a number of environmental and genetic factors. A review by Buskila states that it has been suggested that genetic factors can account for a significant amount of the variance in the perception of pain, sensitivity to painful stimuli and development of chronic pain [31]. This could account for some of the association found in our study. Though this study cannot determine the relative contribution of genetic or environmental influence, or possible epigenetic effects on chronic pain [32], it supports the claim that there is an association between parents and offspring experiencing CMP in the spinal region.

Analysis on subgroups based on pain location showed similar results on the association between parental and offspring pain. According to a study by Leboeuf-Yde et. al. it appears that pain in the shoulder, neck and low back can be seen as the same condition [33]. Though they did observe exceptions for lumbar pain, and therefore cannot rule out separate entities for a smaller group of individuals with back pain. This is in agreement with findings from the current study.

4.2 LTPA modifying effect on parent-offspring spinal pain association:

Changes in lifestyle could account for the potential increase in back pain reports [1,2]. One of the theories behind PA being a viable preventive method is that there has been an increase in inactive work environments and lifestyles [2]. Results from this study show a weaker parent-offspring association if the offspring reported being active, but interaction analyses showed that these results were not statistically significant. The

non-significance of these results could be caused by the lack of nuance in the dichotomous PA variables used in the analysis. The PA variables in the dataset combined results from questions on PA intensity, volume and duration into a four-category variable. This variable was then split into a dichotomous variable. People who had answered no or low PA was combined into one group, and medium and high constituted a second group. According to Nilsen et. al. even low levels PA can have a protective effect on LBP [14]. Because subjects who reported low activity levels were included in the inactive group, possible effect of physical activity could have been masked. Descriptive statistics showed that among the offspring the no/low activity groups included few subjects compared to the medium/high activity group. The same analysis showed that 55% of the offspring group reported a high activity level. Thus it suggests that a too high activity level could reduce the potential beneficial effect of PA. In the parental group the opposite was true; more parents reported being inactive than active. This could be due to a generation change; the offspring are generally more active, or it could be that there was a higher reporting rate of activity than the actual activity level of the population group among the offspring.

To our knowledge there are no previous studies that have investigated whether or not PA can have a modifiable effect on parent-offspring association on back pain. Hartvigsen and Christensen found higher levels of PA to be protective for LBP and that the effect was more pronounced in people with LBP lasting longer than 30 days [15]. In addition studies done by Nilsen et. al, Holth et. al, and Hagen et. al. show that inactive subjects had a higher risk of developing CMP than active subjects [11,14,16]. These results are consistent with the trend found in the current study, but they do not give any additional information as to the effect on a heritable component to back pain. It is still unknown whether or not PA modifies the effect because of parental influence on behavior, and thus affects the environmental factor of back pain.

According to Vik et. al. lifestyle factors such as BMI and PA level track across generations [34]. With this in mind an attempt to answer the questions above was conducted by analyzing the relative influence of parental and offspring PA on the parent-offspring association to CMP. Because many subgroups resulted in very few subjects, this analysis was conducted on overall pain. The results of the current study show the strongest parent-offspring association in overall pain when offspring were

inactive and parents were active. This could be because the offspring have reduced their activity level as a response to back pain. Another possible explanation could be that the offspring activity level is independent of parental influence, and that offspring activity level affects their risk of back pain. We are not aware of any similar studies, and are therefore unable to compare result.

4.3 Study strength and limitations:

The strengths of the current study include the population-based nature of the data, a large number of parent-offspring trios, and the ability to link family members using the Family registry at Statistics Norway. Another strength is the independent reports given by both the adult offspring and their parents. The questionnaires used enabled us to adjust for parental characteristics associated with CMP. These included age [1,2], BMI [7], work related PA [11-13], and psychological wellbeing [10]. Another confounder that could have been adjusted for was socioeconomic status. Previous studies have shown an association between socioeconomic status and CMP [9], but analyses of potential confounders in the current study found socioeconomic status to have a weak effect on the parent-offspring association on CMP. It should be noted that residual confounding due to unknown factors cannot be ruled out, as this is an observational study.

Limitations to this study include no questions on pain intensity, and that the reported back pain could be the result of a specific cause or trauma such as a fracture or prolapse due to the nature of the questions asked. Another limitation is that there is no information on pain variance over time. There is no information on how the pain has developed, if it was constant, or if there had been any pain previous to the HUNT study, or in the time afterwards. Nevertheless the questions on chronic musculoskeletal pain used in the HUNT study have been shown to have acceptable reliability [35]. The stratified analyses on PA included very crude variables on PA due to their dichotomous nature, despite questions on PA in the HUNT studies including intensity, duration and volume. This could be one of the reasons as to the weak effect found in this study. Another explanation could be that the questions did not include the specific type of activity subjects performed. According to Sullivan et. al. aerobic exercise, pilates and yoga have shown significantly beneficial effects in rehabilitation of chronic pain [36]. This could indicate that not all forms of PA have a preventive effect on CMP. In the final analyses on the relative effect between parental and

offspring PA on the parent – offspring association on back pain, there were few subjects in the subgroups. Only results from the overall pain group showed significant results. The overall pain variable includes pain from the entire body, and thus represents CMP in general, not only in the spinal region. Furthermore the subjects participating in the HUNT3 study have been shown to be a healthier population group, with a higher socioeconomic status, and lower mortality than non-participants [37]. This indicates that participants represent a more health – conscious sample than the general population, which could result in an underestimation of the results. Though the same study by Langhammer et. al. found participants under the age of 80 to report more musculoskeletal pain than non-participants. However, Rothman have argued that representativeness is not a prerequisite for valid associations of biological phenomena [38], and unless there is a systematic difference between participants and non-participants in parent-offspring pain association, then it is unlikely the selection bias affects our results.

Finally, previous studies have been conducted on the subject using HUNT-data, but studies using other study populations in this age group are sparse. Thus future studies should be conducted using different sample groups. This would enable us to either confirm or disprove the results found in the current study. Analyses on the effect of PA on parental – offspring association on CMP showed interesting results that also should be investigated further as there are so few studies on the preventive effect of PA on CMP [18].

In conclusion, the current study found an association between parental and offspring CMP pain that could be explained by both genetic and environmental factors. There was observed a trend towards active offspring having a weaker association with parental pain, than inactive subjects, though this needs further research to confirm. The relative activity level between parents and offspring showed inactive offspring to have a greater association with their parental back pain, independent of parental activity level. The findings of the current study contribute to the understanding of CMP tracking across generations. It also shows that further research should be conducted on PA as a preventive factor both on the general population and population groups with a family history of chronic pain.

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