BMJ Open

Effects of reducing sedentary behaviour on back pain, paraspinal muscle insulin sensitivity and muscle fat fraction and their associations: a secondary analysis of a 6-month randomised controlled trial

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To cite: Norha J, Sjöros T, Garthwaite T, *et al*. Effects of reducing sedentary behaviour on back pain, paraspinal muscle insulin sensitivity and muscle fat fraction and their associations: a secondary analysis of a 6-month randomised controlled trial. *BMJ Open* 2024;**14**:e084305. doi:10.1136/bmjopen-2024-084305

► Prepublication history and additional supplemental material for this paper are available online. To view these files, please visit the journal online (https://doi.org/10.1136/bmjopen-2024-084305).

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Received 15 January 2024
Accepted 09 September 2024

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ABSTRACT

Objectives Sedentary behaviour (SB) is a plausible intervention target for back pain mitigation. Therefore, this study aimed to investigate the effects of a 6-month SB reduction intervention on back pain and related disability outcomes, and paraspinal muscle (ie, erector spinae and transversospinales separately) insulin sensitivity (glucose uptake, GU) and muscle fat fraction (FF).

Methods Sixty-four adults with overweight or obesity and metabolic syndrome were randomised into intervention (n=33) and control (n=31) groups. The intervention group aimed to reduce SB by 1 hour/day (measured with accelerometers) and the control group continued as usual. Back pain intensity and pain-related disability were assessed using 10 cm Visual Analogue Scales and the Oswestry Disability Index (ODI) questionnaire. Paraspinal muscle GU was measured using 18-fluorodeoxyglucose positron emission tomography during hyperinsulinaemic-euglycaemic clamp. FF was measured using MRI.

Results Pain-related disability increased during the intervention in both groups. Back pain intensity increased significantly more in the control group than in the intervention group in which back pain intensity remained unchanged (group×time p=0.030). No statistically significant between-group changes in pain-related disability, ODI or paraspinal GU and FF were observed. In the whole study group, the change in daily steps was associated positively with the change in paraspinal muscle GU.

Conclusion An intervention focusing on SB reduction may be feasible for preventing back pain worsening regardless of paraspinal muscle GU or FF.

Trial registration number NCT03101228.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ The strengths of this study include the use of accelerometers to monitor physical activities and sedentary behaviours throughout the 6-month study.
⇒ Moreover, the imaging modalities (positron emission tomography with hyperinsulinaemic-euglycaemic clamp for muscle-specific insulin resistance and MRI for muscle-specific fat fraction) may be considered the gold-standard measures.
⇒ However, this is a secondary analysis of the whole study, and thus the power calculations were not done for back pain or disability.
⇒ Further, no specific back pain-related eligibility criteria were applied.

INTRODUCTION

Physical activity (PA) is associated with a decreased risk for low back pain.[1 2] Conversely, observational studies suggest an association between high sedentary behaviour (SB) and increased low back pain or pain-related disability.[1 3] A meta-analysis of 16 longitudinal studies reported that higher SB was associated with higher pain-related disability but not with back pain intensity.[1] On the other hand, a meta-analysis of cross-sectional studies found a positive association between non-occupational and occupational SB and back pain.[3] Moreover, we have previously observed cross-sectionally that higher SB is associated with lower pain-related disability.[4] Thus, it is clear that the observational evidence is mixed. However, different study settings (ie, cross-sectional or longitudinal) represent different time frames and the possibility of reverse causality cannot be ruled out.

Previous 3–6-month interventional studies among 50-year-old office workers suggest that reducing SB might improve pain-related disability without affecting back pain intensity.[5 6] However, the mechanisms by which SB modification could affect back pain or disability remain poorly understood.

Insulin resistance and fatty infiltration of the paraspinal muscles are associated with back pain[7–11] and successfully reducing SB improves muscle insulin sensitivity.[12] Moreover, lower levels of PA are associated with higher fat content of the transversospinal muscles.[13] Taken together, these findings make SB a plausible target for an intervention to maintain or improve back health.

Thus, we aimed to investigate the effects of a 6-month SB reduction intervention on back pain, disability and paraspinal muscle fat fraction (FF) and insulin sensitivity (glucose uptake, GU). Additionally, we assessed the back pain- related factors cross-sectionally.

METHODS

This study consists of secondary analyses of a 6-month randomised controlled trial that was conducted at the Turku PET Centre (Turku, Finland) between April 2017 and March 2020 (ClinicalTrials.gov NCT03101228, 5 April 2017).

Participants

As reported earlier,[12 14] volunteers were recruited from the community. Inclusion criteria were age 40–65 years, body mass index (BMI) 25–40 kg/m^2, self-reported physical inactivity (<120 min/week of moderate-to-vigorous PA (MVPA)), accelerometer-measured sedentary time ≥10 hours or ≥60% of accelerometer wear time and metabolic syndrome.[15] Exclusion criteria included diagnosed diabetes or fasting blood glucose ≥7.0 mmol/L, abundant alcohol consumption according to the national guidelines, the use of any tobacco products, diagnosed depressive or bipolar disorder, inability to understand written Finnish, and any condition that would endanger the participant or study procedures (eg, previous exposure to ionising radiation).

Measurements

Back pain intensity and pain-related disability were assessed by two questions and 10 cm Visual Analogue Scale (VAS): (1) Have you had back pain within the last month? Mark the intensity of the worst perceived pain during the month on the line below and (2) Has the perceived pain caused you disability at your work or everyday tasks within the last month? Mark the intensity of the greatest extent of disability that you experienced during the month on the line below. A higher value (0–10 cm) indicates higher pain intensity and disability. Additionally, back pain-related disability was assessed using the Oswestry Disability Index (ODI), which provides a value of 0–100% and a higher value represents higher disability.[16]

Paraspinal muscle (ie, erector spinae and transversospinales) FF was assessed using the two-point Dixon MRI method (Philips 3T Ingenuity TF, Philips Healthcare, Amsterdam, The Netherlands and Siemens Magnetom Skyra Fit 3T system, Siemens Healthcare, Erlangen, Germany).[17] Paraspinal muscle GU was measured using ^{18}F-fluorodeoxyglucose positron emission tomography (FDG-PET; GE D690 PET/CT, GE Healthcare, Milwaukee, Wisconsin, USA) during hyperinsulinaemic euglycaemic clamp (HEC) as described previously.[12 18] Both the FF and GU were analysed separately for the transversospinal and erector spinae muscles at the level of L3–4. The measurements were performed using Carimas (V.2.10, https://www.carimas.fi).

PA and SB were measured using accelerometers for 4 weeks during screening (UKK AM30, UKK Terveyspalvelut Oy, Tampere, Finland) to determine the baseline values and throughout the 6-month intervention period (Movesense, Suunto, Vantaa, Finland, with ExSed application, UKK Terveyspalvelut Oy, Tampere, Finland) to monitor and facilitate behaviour change. The accelerometer variables during the intervention period were analysed as means over the whole 6-month period. The participants were advised to wear the device on the right hip during waking hours (except when the device could be exposed to water) and remove it when sleeping at night. Accelerometer wear time of 10–19 hours/day was considered valid, and measurement exceeding 19 hours/day was subtracted from SB as measurement exceeding 19 hours/day likely means that the participant slept with the device on. For example, if the measurement on 1 day was 20.5 hours, 1.5 hours was subtracted from the measured SB, resulting in 19 hours of analysed wear time. The accelerometer data were analysed using 6 s epochs, and the raw acceleration data were analysed using the mean amplitude deviation (to assess sedentariness, light PA (LPA), and MVPA) and angle for posture estimation (to differentiate SB and standing) methods as described previously.[4 18–20]

Height was measured using a wall-mounted stadiometer, and body mass and body fat percentage were measured by air displacement plethysmography (Cosmed USA, Concord, California, USA) after at least 4 hours of fasting. Waist circumference was measured using a measuring tape midway between the lowest rib and the iliac crest. Pain medication use was self-reported by the participants and categorised into using medication or not.

Intervention

After the screening, eligible volunteers were randomised into the intervention and control groups in a 1:1 ratio by a statistician using random permuted block randomisation (block size 44) in SAS (V.9.4 for Windows, SAS Institute). The randomisation was performed for men and women separately.

As described in more detail previously,[18] participants in the intervention group were advised to reduce their daily SB by 1 hour/day for the 6-month study period. Daily SB goals were calculated individually by subtracting 1 hour of SB from the amount during screening. Correspondingly, 1 hour was added to standing, LPA, and MVPA goals distributing the time based on individual preferences. However, a maximum of 20 min was added to MVPA and increasing intentional physical exercise training was discouraged. The ways for replacing SB were discussed

individually and included, for example, using standing desks, taking the stairs instead of the lift and lightly walking. For the control group, the daily SB and PA goals were set equal to the screening values. All participants could monitor their daily SB and PA and the fulfilment of the goals using a mobile phone application (ExSed) connected to the accelerometer.

Patient involvement

Patients were not involved in designing or conducting this study.

Equity, diversity and inclusion

Both the study participants and researchers include self-identified men and women in a relatively balanced fashion. The research group consists of both junior and senior researchers.

Statistical methods

Baseline characteristics are presented as mean (SD) if not stated otherwise. Intervention effects are presented as model-based mean (95% CI). Baseline correlations were analysed using the Spearman rank correlation. The main analyses of intervention effects were performed using linear mixed models for repeated measurements. The outcome of interest was the dependent variable, and independent variables included group, time, sex and group×time in all analyses. Random intercepts for individual effects were also included. Additionally, FF analyses were adjusted for age, and pain questionnaire analyses were adjusted for self-reported regular pain medication status (yes/no) and BMI because this improved the distribution of the residuals. The normal distribution of the residuals was visually inspected, and log10 or square root transformations were performed as necessary. Tukey-Kramer adjustment for multiple comparisons was used. Compound symmetry or unstructured covariance structure was chosen based on the Akaike information criterion. Statistical significance was set at $p<0.05$ (two tailed). The main analyses were performed in SAS (V.9.4 for Windows, SAS Institute) and the correlation analyses were performed using JMP Statistics (V.16, SAS Institute).

The total sample size (n=64) was calculated according to whole-body insulin sensitivity-based power calculations (reported elsewhere).[18] The sample size for the imaging subsample (n=44) was determined based on power calculations for quadriceps femoris insulin sensitivity (reported elsewhere).[12] Assuming an increase of 0.7 (SD 0.55) µmol/100 g/min in the intervention group (10% increase) and an increase of 0.05 µmol/100 g/min in the control group, we calculated that 16 participants per group would be sufficient for detecting a statistically significant between-group change in quadriceps femoris insulin sensitivity ($\alpha=0.05$, $1-\beta=0.9$). To ensure a sufficient sample size despite possible drop-outs and technical challenges, 44 participants were recruited for the imaging subsample. We hypothesise that paraspinal muscle insulin sensitivity would behave similarly to quadriceps femoris and thus, the study would be sufficiently powered to detect statistically significant changes in paraspinal muscle insulin sensitivity.

RESULTS

Baseline characteristics

Of 263 volunteers, 151 were screened, and 64 fulfilled the inclusion criteria. In total, 64 participants were randomised into the intervention (n=33, 39% men) or control (n=31, 45% men) groups (see online supplemental figure 1 for the study flow diagram). Four participants dropped out during the study: one for low back pain (in the control group) and three for personal reasons (two in the control group). Additionally, a subsample of 44 randomised participants (intervention n=23, 39% men; control n=21, 48% men) underwent PET and MRI. The baseline characteristics are presented in table 1.

Baseline correlations

All of the baseline correlation coefficients are presented in online supplemental table 1. Age correlated positively with erector spinae and transversospinal FF ($r_s=0.53$, 0.55, respectively). Erector spinae GU correlated positively with MVPA and step count ($r_s=0.36$ and 0.40, respectively) and negatively with SB ($r_s=-0.31$). Correspondingly, transversospinal GU correlated positively with MVPA and step count ($r_s=0.42$ and 0.40, respectively), but no correlation with SB was found ($p=0.065$). Similarly, both erector spinae and transversospinal FF correlated with MVPA ($r_s=-0.30$ and −0.36, respectively). Increased body adiposity (BMI and body fat percentage) correlated with lower paraspinal muscle GU and higher FF.

Pain-related disability correlated positively with standing time ($r_s=0.27$). Furthermore, the ODI score correlated negatively with MVPA ($r_s=-0.28$) and step count ($r_s=-0.26$). Finally, the ODI score correlated positively with body fat percentage ($r_s=0.33$). Back pain intensity did not correlate with any PA, SB, or paraspinal muscle-related variables.

Intervention effects

Accelerometry

The intervention effects on SB and PA have been reported previously.[18] In comparison to the control group, the intervention group reduced their SB by 40 min/day and subsequently increased their MVPA by 20 min/day, on average over the 6-month intervention period; no statistically significant changes were observed in the control group. LPA increased by 10 min/day in both groups without statistically significant between-group differences. No statistically significant changes in standing time or the number of breaks in SB were observed in either group. Step count increased in both groups but the increase was statistically significantly higher in the intervention group (from 5150 to 6749 steps/day in the control group vs 5326 to 8632 steps/day in the intervention group).

Table 1 Study participant characteristics at the baseline

	Intervention	n	Control	n
Men, n (%)	13 (39)	33	14 (45)	31
Age, years	59 (6)	33	57 (8)	31
Anthropometrics and metabolism				
BMI, kg/m^2	31.5 (4.0)	33	31.7 (4.6)	31
Body fat, %	43.1 (8.0)	33	43.1 (8.0)	31
Waist circumference, cm	111.1 (11.6)	33	110.7 (11.1)	31
fP-Glucose, mmol/L	5.9 (0.5)	33	5.8 (0.4)	31
fP-Insulin, mU/L*	9 (7, 13)	33	11 (7, 17)	30
HbA1c, mmol/l	37.0 (2.8)	33	36.3 (2.7)	31
Transversospinal FF, %*	23.7 (15.6, 33.8)	22	23.8 (19.6, 34.0)	21
Erector spinae FF, %*	17.5 (13.3, 26.8)	22	18.0 (14.4, 23.4)	21
Transversospinal GU, µmol/100 cm^3/min*	2.8 (2.3, 3.2)	23	2.5 (2.0, 3.3)	20
Erector spinae GU, µmol/100 cm^3/min*	2.9 (2.0, 3.3)	23	2.4 (1.9, 3.9)	20
QF GU, µmol/100 cm^3/min*	2.0 (1.4, 2.7)	23	1.9 (1.2, 3.2)	20
Hamstring GU, µmol/100 cm^3/min*	3.0 (2.0, 4.6)	23	2.8 (1.4, 4.0)	20
Whole-body GU, µmol/kg /min*	15.3 (10.7, 21.0)	33	13.9 (9.8, 21.0)	31
Pain and disability				
Regular medication for pain, n (%)	3 (9)	33	4 (13)	31
VAS back pain, 0–10 cm*	0.3 (0.1, 3.5)	33	0.5 (0.1, 3.0)	29
VAS pain-related disability, 0–10 cm*	0.4 (0.1, 2.2)	33	0.7 (0.2, 2.6)	30
Oswestry Disability Index, %*	6.0 (1.0, 13.0)	33	6.7 (2.0, 16.0)	31
Physical activity				
Accelerometry, hour/day	14.5 (1.0)	33	14.6 (1.0)	31
Sedentary time, hour/day	10.0 (0.9)	33	10.1 (1.1)	31
Standing time, hour/day	1.8 (0.6)	33	1.8 (0.6)	31
LPA, hour/day	1.7 (0.4)	33	1.8 (0.5)	31
MVPA, hour/day	0.96 (0.31)	33	0.97 (0.34)	31
Breaks in sedentary time, n/day	28 (8)	33	29 (8)	31
Steps, n/day	5204 (1910)	33	5091 (1760)	31

Unless otherwise stated, the results are presented as mean (SD).
*Presented as median (Q1, Q3).
BMI, body mass index; FF, fat fraction; fP-Glucose, fasting plasma glucose; fP-Insulin, fasting plasma insulin; GU, insulin-stimulated glucose uptake; HbA1c, glycated haemoglobin; LPA, light physical activity; MVPA, moderate-to-vigorous physical activity; QF, quadriceps femoris muscle; VAS, Visual Analogue Scale.

Pain and disability questionnaires

The pain and disability questionnaire results are presented in figure 1, and the changes in each participant's back pain by group are presented in figure 2. In the intervention group, back pain did not change whereas it increased statistically significantly in the control group (group×time p=0.030). Pain-related disability increased over time in both groups (time p=0.017), but no statistically significant between-group differences in the changes in pain-related disability or ODI were observed.

Paraspinal muscle FF and GU

Transversospinal FF was higher in the control group throughout the study (p=0.011), but no statistically significant changes were observed in paraspinal muscle FF or GU in either group (figure 3).

Explorative analyses

As previously done,[12 18] when the study group was divided according to the measured changes in SB or daily steps no statistically significant changes in any pain-related outcomes were observed (group x time p>0.05 for all; data are not shown). Furthermore, no statistically significant differences were observed in paraspinal muscle FF or GU when the group was divided according to the measured change in SB (group×time p>0.05 for all; data are not shown). However, with the step-based groups (ie, an increase of >2500 steps/day vs <2500 steps/day increase or

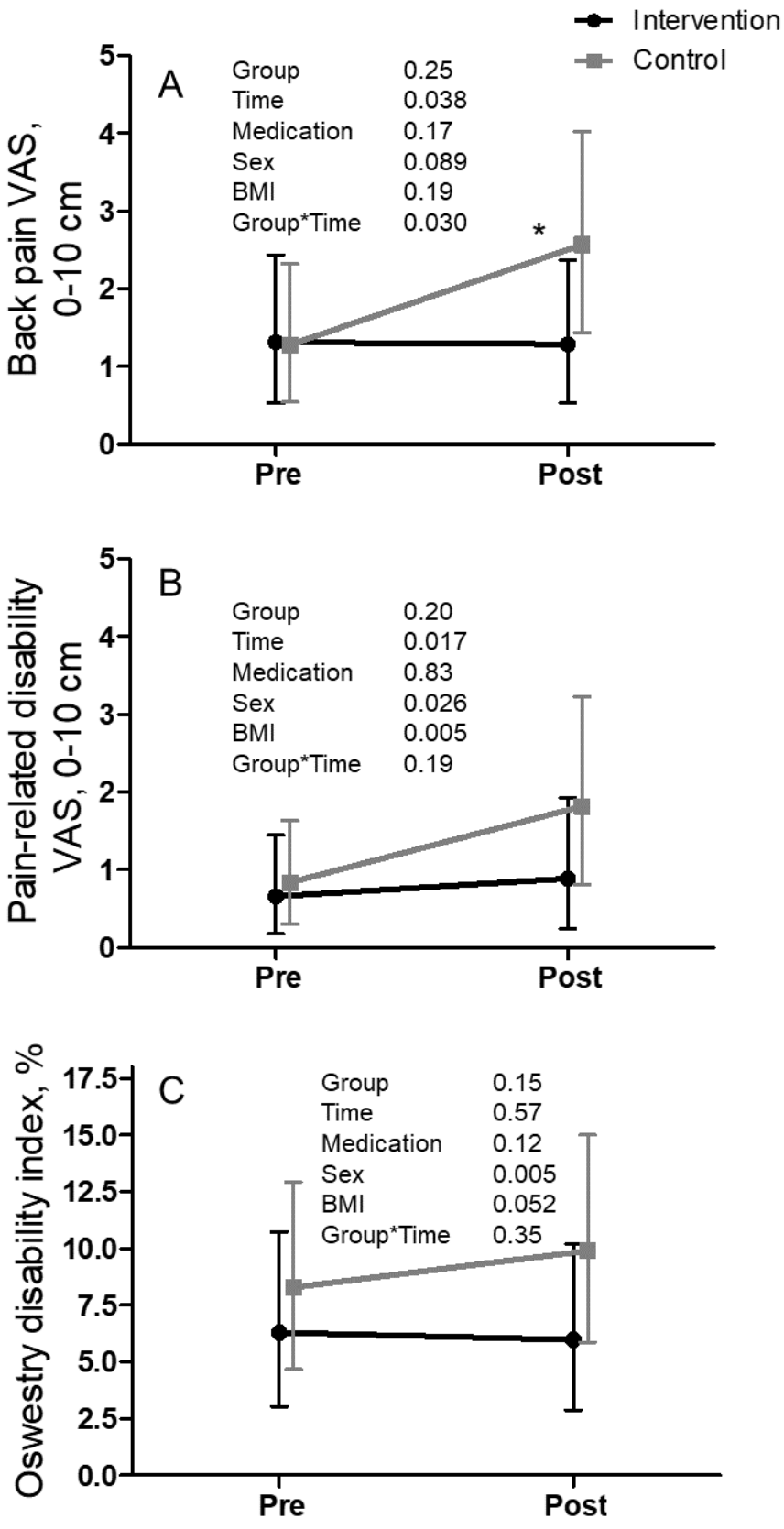

Figure 1 Intervention effects on (A) back pain intensity, (B) pain-related disability and (C) the Oswestry Disability Index. All analyses are adjusted for sex, pain medication status and body mass index (BMI). Black dots represent the intervention group and grey squares represent the control group. The presented estimates are model-based means and 95% CIs. A higher value indicates higher pain intensity or disability on all panels. *Tukey's p=0.026. VAS, Visual Analogue Scale.

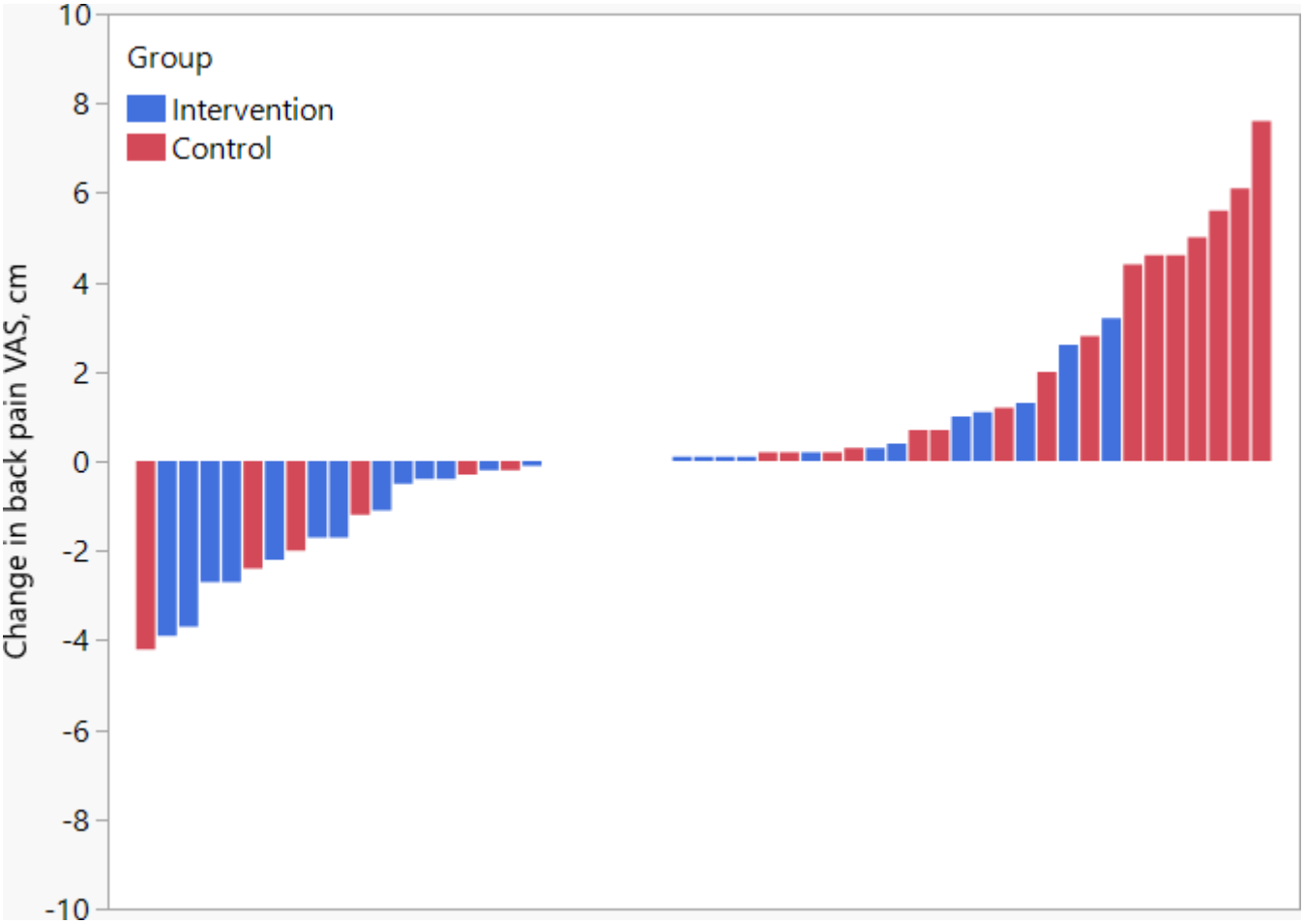

Figure 2 Changes in each participant's back pain during the intervention. Blue bars represent participants in the intervention group and red bars represent participants in the control group. Of the six participants with no changes in back pain, four were in the intervention and two in the control group. A higher value indicates higher pain intensity. VAS, Visual Analogue Scale.

decrease), the changes in erector spinae and transversospinal GU were statistically significantly different between groups in favour of the more active group (group×time p=0.033) (online supplemental figure 2).

In the whole study group, the changes in BMI, body fat percentage and body mass correlated positively with the change in ODI (r_s=0.37, 0.26 and 0.35, respectively; online supplemental table 2). None of the changes in PA or SB correlated with the changes in pain-related outcomes. The change in BMI correlated negatively with the change in erector spinae and transversospinal GU (r_s=−0.34 and −0.40, respectively). In line with the analyses based on high versus low step count increase, the changes in steps correlated positively with the changes in paraspinal muscle GU (r_s=0.39 and 0.41 for the transversospinales and erector spinae, respectively) but not with the changes in FF.

DISCUSSION

In the present study, we show that an intervention aimed at reducing SB by 1 hour/day for 6 months may prevent the worsening of back pain intensity which was observed in the control group. However, the change in back pain intensity was not associated with changes in paraspinal muscle (ie, erector spinae or transversospinales) FF, GU or the changes in PA, SB, pain-related disability or ODI score. Additionally, no intervention effects on paraspinal muscle FF or GU were observed, although increases in daily steps were associated with improved paraspinal muscle GU.

Pain and physical behaviours

In this study, back pain intensity increased by about twofold in the control group, on average. Although the baseline median back pain was relatively low among all participants (median 0.3 cm and 0.5 cm on the VAS in the intervention and control groups, respectively), the change in the control group represents a substantial relative change in pain intensity.[21] Considering this, preventing back pain from worsening with an SB reduction-focused intervention could be clinically meaningful, even if no improvements in pain intensity are achieved. However, we did not observe any intervention effects on pain-related disability or ODI score, meaning that the changes in back pain intensity were unrelated to functional outcomes. This might be explained by the relatively low pain intensity that might not be severe enough to cause disability.

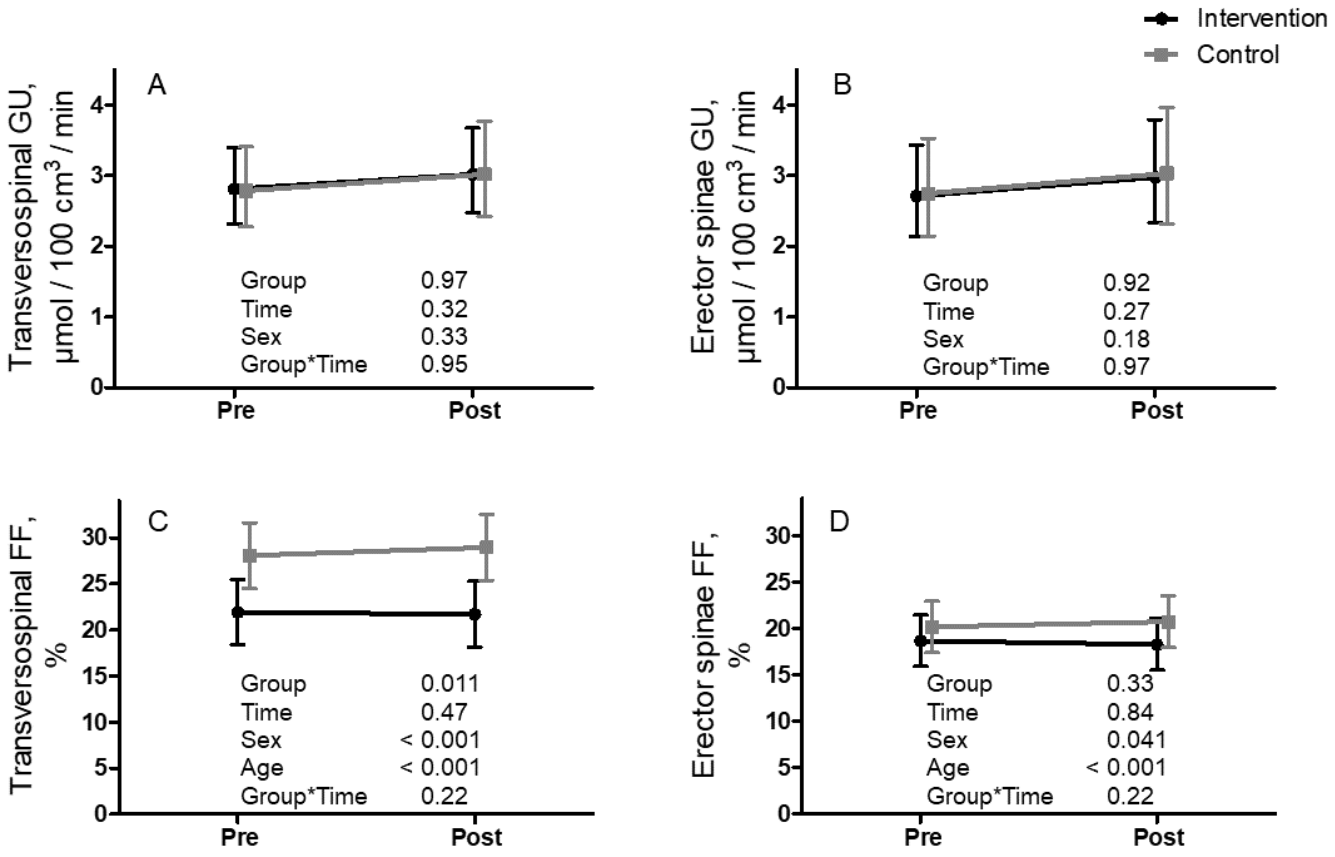

Figure 3 Intervention effects on (A) transversospinal muscle glucose uptake (GU), (B) erector spinae GU, (C) transversospinal muscle fat fraction (FF) and (D) erector spinae FF. GU analyses (A and B) are adjusted for sex, and FF analyses (C, D) are additionally adjusted for age. Black dots represent the intervention group and grey squares represent the control group. The presented estimates are model-based means and 95% CIs.

The reason for back pain intensity increase in the control group remains elusive. One explanation for the increase could be related to the open-label nature of this study. Although not formally documented, many control participants were disappointed to be included in the control group instead of the intervention group. These negative emotions may have affected pain intensity.[22] This phenomenon, in conjunction with the possible benefits from the increased PA in the intervention group, could explain the difference between groups. The fact that the explorative analyses with SB or step-based post hoc group divisions showed no between-group differences in pain-related outcomes further emphasises that the sole allocation to either intervention or control group may have affected the perception of pain. However, the cross-sectional correlations in this study show that a higher amount of MVPA and a higher step count are associated with better function, measured with the ODI (online supplemental table 1). Moreover, both the cross-sectional correlations and the correlations of changes during the study suggest that maintaining a healthier body composition could decrease disability, as body fat percentage correlated positively with the ODI score (see online supplemental tables 1 and 2).

Contrary to our results, a previous 6-month randomised controlled trial involving adults with low back pain (mean ODI score about 24%) observed a statistically significant improvement in ODI with an intervention that resulted in an average SB reduction of 1.4 hours/day.[5] Moreover, back pain intensity measured using VAS did not differ between groups in the study.[5] The study sample was comparable to ours in terms of age, SB and BMI, but two-thirds of the participants met the PA guidelines, whereas in our study, this was an exclusion criterion. Additionally, an inclusion criterion in the previous study was long-standing low back pain, while we did not consider pain history in the inclusion or exclusion criteria. Further, the aforementioned study did not aim to change only SB but also included behavioural counselling in the self-management of pain. Furthermore, the reduction in SB was notably higher in the previous study compared with ours (1.4 vs 0.7 hours/day).[5] These factors can explain the differences in the findings. Additionally, the intensity of longstanding pain might not always be related to the disability.[23]

It should be acknowledged that back pain is often a recurring and varying, and sometimes a long-standing complaint.[24] For this reason, future studies should assess pain and disability more frequently than only at baseline

and at the end of the intervention, as in the current study. However, the 6-month duration of this study should be sufficient to reveal the effects of an SB reduction intervention, as in a previous study the ODI score tended to decrease up until 3 months before plateauing.[5]

As reported earlier (although with n=72 from the screening data), standing time correlated positively with pain-related disability at baseline in our study.[4] However, no correlation between the change in standing time and the change in pain-related disability was observed. Related to this finding, a recent randomised controlled trial observed that, within 3 months, increasing occupational standing may increase multisite musculoskeletal pain, but in the longer term (12 months), the increase in pain was no longer present.[25] Thus, as cross-sectional correlations represent shorter rather than longer term, it seems that standing may exacerbate pain acutely, but habitual standing may not be detrimental.

Paraspinal muscle FF and GU

We did not observe any intervention effects on either erector spinae or transversospinal FF. This finding is consistent with a recent systematic review of six intervention studies, concluding that paraspinal FF cannot be reduced even with exercise training.[26] This demonstrates that even though paraspinal muscle FF is strongly associated with back pain,[7–9] successful back pain prevention or treatment can be independent of FF. This may be explained in part by the effect of time, that is, age, which correlated with paraspinal FF (r_s=0.55 and 0.53 for transversospinal and erector spinae FF; see online supplemental table 1) and was a significant contributor in the linear models investigating paraspinal FF ($p<0.001$ for both muscle groups) in this study. In accordance, the effectiveness of back pain rehabilitation is not related to specific strength or mobility goals of the rehabilitation,[27] emphasising other than structural aspects in treating experienced pain and disability. Therefore, back pain risk factors (such as paraspinal muscle FF) should not be the direct targets of rehabilitation as much as the psychological and cognitive aspects of pain perception and the individual preferences for physical exercise.[28] However, as lower paraspinal muscle FF associated with higher amounts of MVPA and lower body adiposity, the results suggest that maintaining healthy body composition and MVPA levels might help prevent fat infiltration of the paraspinal muscles.

We have previously reported the intervention effects on hamstrings and quadriceps femoris GU[12] which seem to have responded similarly to the intervention as the paraspinal muscles. The main analyses revealed no intervention effects on any of these muscles. However, the secondary analyses of the present study and the previously published study show the association between increased PA (eg, steps) and improved muscle GU.[12] Additionally, the paraspinal and thigh muscle GU have statistically significant moderate-to-strong correlations of 0.69–0.84 (see online supplemental table 1). Furthermore, paraspinal muscle GU was not cross-sectionally associated with any pain-related outcomes, nor was the change in paraspinal muscle GU correlated with the changes in any pain-related outcomes (online supplemental table 2). Moreover, as paraspinal muscle GU but not FF was associated with steps, the results suggest that GU can improve despite no changes in FF. Finally, as observed before with whole-body GU,[18] the change in BMI correlated negatively with paraspinal muscle GU, indicating lower insulin sensitivity with increasing BMI.

Clinical implications

The present study highlights that being in an SB reduction intervention which elicits changes to PA, standing and SB might work as a protective strategy against back pain. Furthermore, as observed before with strength or mobility goals for rehabilitation,[27] the possible improvements in pain or disability seem to not be related to paraspinal muscle GU or FF.

Strengths and weaknesses

The strengths of this study include the robust measurement of PA, standing and SB with accelerometers during the whole 6-month study. Moreover, the accelerometer data were analysed using validated algorithms.[19 20] Furthermore, the ODI is a validated questionnaire,[16] and VAS is commonly used for pain assessment, and it is associated with functional outcomes.[29] However, a weakness in this study is the use of non-validated questions with the VAS. Another key strength is the muscle-specific GU assessment with the HEC protocol combined with FDG-PET imaging.[30] Further, the two-point Dixon is a highly reproducible method for FF assessment.[31]

One limitation of the present study is the sample size. For the GU assessments, the sample size was likely adequate,[12] but as the pain-related outcomes were not the primary outcomes of the whole trial, the statistical power might have been inadequate. Additionally, the study sample was not chosen based on pain status which may have increased heterogeneity in the sample and thus decreased the statistical power.

Conclusion

An intervention that reduces SB by mainly replacing it with PA may prevent increases in back pain intensity in adults with metabolic syndrome and physical inactivity. Replacing the SB by walking over 6 months may contribute to improved paraspinal muscle insulin sensitivity, and these factors warrant continued investigation in the context of pain and disability.

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Acknowledgements This study was conducted within the Centre of Excellence in Cardiovascular and Metabolic Research, supported by the Research Council of Finland, the University of Turku, Turku University Hospital and Åbo Akademi University. We would like to thank the staff of the Department of Clinical Physiology and Nuclear Medicine at Turku University Hospital and Turku PET Centre for assistance in the data collection.

Contributors IH, KK, TVasankari and TS conception and design of the study. TS, SL, TG, KL and NH acquisition of data. TS, JN, HV-Y, HS, TG, SL, TVerho, VS, JH, EL and IH analysis and interpretation of data. JN drafted the manuscript and all authors edited and revised the manuscript. IH acted as the guarantor.

Funding This study was funded by the Research Council of Finland (324243), the Finnish Cultural Foundation, the Juho Vainio Foundation, the Hospital District of Southwest Finland, the Yrjö Jahnsson Foundation, the Turku University Foundation, the Finnish Diabetes Research Foundation, Turku University Hospital Foundation and the Päivikki and Sakari Sohlberg foundation (220068).

Disclaimer The funding bodies did not take part in designing the study, collecting, analysing or interpreting the data or preparing the manuscript.

Competing interests The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: TS received a speaker fee from Pihlajalinna Plc, Tampere, Finland. The other authors report no conflicts of interest. The results are presented clearly and honestly without fabrication, falsification or inappropriate data manipulation.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and was approved by the Ethics Committee of the Hospital District of Southwest Finland, 16/1801/2017. Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available on reasonable request. Data are available on reasonable request from the corresponding author.

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