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Research article

The Effect of Structured Exercise Intervention on Intensity and Volume of Total Physical Activity

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Abstract

This study aimed to investigate the effects of a 12-week structured exercise intervention on total physical activity and its subcategories. Twenty-three overweight or obese middle aged men with impaired glucose regulation were randomized into a 12-week Nordic walking group, a power-type resistance training group, and a non-exercise control group. Physical activity was measured with questionnaires before the intervention (1–4 weeks) and during the intervention (1–12 weeks) and was expressed in metabolic equivalents of task. No significant change in the volume of total physical activity between or within the groups was observed ($p > 0.050$). The volume of total leisure-time physical activity (structured exercises + non-structured leisure-time physical activity) increased significantly in the Nordic walking group ($p < 0.050$) but not in the resistance training group ($p > 0.050$) compared to the control group. In both exercise groups increase in the weekly volume of total leisure-time physical activity was inversely associated with the volume of non-leisure-time physical activities. In conclusion, structured exercise intervention did not increase the volume of total physical activity. Albeit, endurance training can increase the volume of high intensity physical activities, however it is associated with compensatory decrease in lower intensity physical activities. To achieve effective personalized exercise program, individuality in compensatory behavior should be recognised.

Key words: Nordic walking, resistance training, energy expenditure, thermogenesis, glucose intolerance.

Introduction

Physical inactivity is the fourth largest risk factor for mortality and a major risk factor for non-communicable diseases (World Health Organization, 2009). The worldwide prevalence of non-communicable diseases, including diabetes, cardiovascular disease, and cancer, is increasing and they are estimated to account for 63% of global deaths (World Health Organization, 2011). Combined evidence from 76 countries suggests that approximately 25% (range 2.6% – 62.3%) of people are currently sedentary (Dumith et al., 2011). Thus, one of the leading targets in the prevention of non-communicable diseases is to enhance the overall intensity and volume of physical activity. The prevention of physical inactivity and related diseases pertains all professionals working in health care, especially exercise physiologists and physiotherapists who have expertise in exercise prescription.

Physical exercises are often prescribed as a first line of defence against inactivity. Such interventions, however may result in compensatory decrease in non-exercise physical activity (King et al., 2007). Non-exercise physical activity has been shown to decrease with cycling (Goran and Poehlman, 1992, Morio et al., 1998, Manthou et al., 2010), walking (Colley et al., 2010), or combined resistance and aerobic training (Meijer et al., 1999) exercise interventions. However, the current evidence regarding the compensatory response is inconsistent (Hollowell et al., 2009, Turner et al., 2010). In previous studies physical activity measurements have accounted only for a small portion of the intervention and focusing mainly on the change in total energy expenditure. Thus, the effect of exercise on non-exercise physical activity during the whole intervention remains to be determined, especially in the sense of intensity and volume. Better understanding of compensatory behavior, could enable more personalized and effective dosage of exercise for different impairments and medical conditions.

The purpose of this study was to measure total physical activity during an exercise intervention and to investigate the effects of a 12-week structured aerobic training (Nordic walking) and power-type resistance training on the intensity and volume of total physical activity and its subcategories. An additional aim was to define the correlates for changes in physical activity.

Methods

Twenty-three (n = 23) male volunteers from a larger (n = 144) randomized controlled trial (ISRCTN97931118), who had completed physical activity questionnaires were included in this study. The inclusion criteria for the trial were: male, 40–65 years of age, body mass index 25.1–34.9 $kg \cdot m^{-2}$, over 12 points in the Finnish diabetes risk test (Lindström and Tuomilehto, 2003), impaired glucose

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regulation (impaired fasting glucose 5.6–6.9 mmol·L^{-1} and/or impaired glucose tolerance 7.0–11.0 mmol·L^{-1}), no other metabolic diseases, and a successfully passed medical examination. Criteria of exclusion were as follows: previously detected IGR and engagement in any customized diet or exercise program, engagement in very vigorous habitual physical activities, or usage of medication that affects glucose balance. Participants that fulfilled the criteria and gave their written informed consents were initially randomly assigned (1:1:1) to a Nordic walking (NW, n = 48) group, a power type resistance training (RT, n = 49) group, and a non-exercise control (C, n = 47) group. Of that initial sample of subjects, twenty-three subjects returned the completed physical activity questionnaires at least from a 1-week period before the intervention and from a 10-week period during the intervention, and formed the sample for the present study. Altogether, the present study included seven (n =7) subjects from the NW-group, eight (n = 8) from the RT-group, and eight (n = 8) from the C-group. The baseline characteristics of these subjects compared to the other who completed the initial trial have been shown in Table 1. As shown in Table 1, there were no significant differences in the baseline characteristics between those who were included in this study (n = 23) and those who completed the trial but were excluded from this study due to the insufficient physical activity data (n=93). This study was approved by the Coordinating Ethical Committee of the Hospital District of Helsinki Finland. The funding organisations had no role in the collection, analysis, or interpretation of the data of this study, nor did they have the right to approve or disapprove the publication of this manuscript.

A detailed description of the exercise programs has been previously reported (Venojärvi et al., 2013; Wasenius et al., 2014). In brief, both exercise groups completed a 12-week structured physical exercise program, which included 3 training sessions per week (non-consecutive days) each lasting approximately 60 minutes. The NW-group performed progressive endurance training at 50–60% of heart rate reserve (HRR) in weeks 1–4, 60–70% of HRR in weeks 5–8, and 70–80% of HRR in weeks 9–12. At the beginning of each session walking (5 min) and stretching of the main muscle groups were performed to warm-up the body. For cool-down, stretching activities were repeated at the end of each session. The RT-group performed power-type resistance training exercises for main muscle groups with a maximal contraction velocity and an external load from 50% to 80% of the estimated exercise specific maximal strength capacity. The load (from 50% up to 80% of 1-repetition maximum) and number of sets increased (from 1 to 4) while the number of repetition decreased (from 10 to 3) progressively throughout the intervention. Before and after the intervention exercise session cycling or rowing with the ergometer (5 min) and stretches of the main muscle groups were performed to warm-up and cool-down the muscles. The adherence and dose of structured exercise interventions (NW and RT) compared to the other subjects who completed the initial trial (excluded from this study) are shown in Table 2. In the RT-group the intensity was slightly lower for those who were included while there was no significant difference in the NW-group. The dose of the NW-group was significantly greater compared to the RT-group for those who were included, which is consistent with a previous report (Wasenius et al., 2014).

All participants in all three groups were advised not to change their habitual physical activity or lifestyle during the intervention. The C-group participated in the general lectures given to all participants before the intervention, but received no other exercise, diet, or any other kind of intervention.

Body height was determined to the nearest 0.5 cm and body weight (kg) with calibrated weighing scales to the nearest 0.1 kg. BMI was calculated by dividing body weight (kg) with height in meters squared (kg·m^{-2}). Body fat (kg) and fat per cent (%) were measured with electrical bioimpedance (Korea Inbody 3.0, Biospace Co., Seoul, South Korea). Maximum oxygen uptake (VO_{2peak}) was estimated directly during a continuous incremental cycle ergometry until volitional exhaustion or subjective maximum.

Physical activity was measured weekly with specific questionnaires before (4 weeks) and during (12 weeks) the intervention. The questionnaires were completed in sets of 4 weeks. For week 1 participants were instructed to complete the questionnaire accurately in a diary manner and complete the following 3 weekly questionnaires retrospectively while using the first questionnaire as a guiding rule. The questionnaire measured physical activity throughout the day (24 h per day, 7 days a week) and it was divided into the following subcategories of physical activity: occupational (or other daytime) physical activity (OPA), commuting (or transition) physical activity (CPA), leisure-time physical activity (LTPA), miscellaneous home physical activity (MHPA), structured physical exercise activity (SPEA = NW or RT activities), and sleep. The questionnaires asked about the type, frequency (times/week), duration (min), and rating of perceived exertion (RPE) of an activity. The questionnaire included prespecified types of activities (13 in OPA, 11 in CPA, 10 in LTPA, 8 in MHPA), but participants also had the possibility to include their own activities.

Table 1. Baseline participants characteristics. Data is shown as mean (standard deviation).

	Excluded subjects	Included subjects				
Characteristics	**(n = 93)***	**RT (n = 8)**	**NW (n = 7)**	**C (n = 8)**	**p†**	**p‡**
Age (years)	53.9 (6.4)	55.0 (6.9)	56.6 (8.3)	58.1 (5.1)	.650	.079
BMI (kg·m^{-2})	29.7 (3.4)	33.3 (1.2)	29.9 (3.5)	27.6 (2.4)	.107	.550
BFP (%)	25.3 (5.4)§	24.3 (2.0)	26.1 (3.7)	20.4 (6.6)	.085	.154
VO_{2peak} (ml·kg^{-1}·min^{-1})	30.8 (6.6)||	28.8 (5.9)	28.2 (4.7)	32.9 (8.3)	.529	.432

RT = resistance training group, NW = Nordic walking group, C = control group, BMI= Body mass index, BFP = Body fat percentage VO_{2peak} = peak oxygen uptake. *Subject who completed the randomized controlled trial, but had incomplete physical activity data to be included in this study, †p-value between the included subjects groups, ‡p-value between the excluded subject and the included subjects, §n = 91, || n = 66

Table 2. The physical dose of structured exercise intervention. Data is shown as mean (standard deviation).

Determinants of physical activity	Resistance training group		Nordic walking group	
	Excluded (n = 28)	Included (n = 8)	Excluded (n = 32)	Included (n = 7)
Adherence (%)	65(24)	78 (15)	65 (18)	65 (30)
Absolute intensity (MET)	3.3 (.5)	2.8 (.3)*	5.9 (.4)	5.2 (1.5)†
Relative intensity (%)	43.3 (11.1)	35.8 (6.4)	71.8 (14.0)	67.0 (24.5) †
Volume (METh·wk^{-1})	10.5(1.7)	6.8 (1.8)*	15.5 (2.9)	12.3 (6.3)

MET = metabolic equivalent of task; METh = MET-hours; Relative intensity = percentage of absolute intensity from the individual VO_{2peak} expressed in MET-values. *p-value < 0.050 compared to the excluded in the particular group. †p-value < 0.050 compared to the included resistance training group.

To express the physical activity during the intervention, the mean of physical activity of all individual weeks was calculated. The baseline values were then subtracted from these values to determine the change in physical activity during the intervention. The subcategories were also combined to calculate the physical activity of total LTPA (=LTPA+SPEA), activity time physical activity (ATPA=OPA+CP+LTPA+SPEA), non-LTPA (=total physical activity–total LTPA–SLEEP), and non-exercise physical activity (NEPA = total PA–SPEA–SLEEP).

A metabolic equivalent of task (MET) value was determined for each type of activity according to the reported type of activity and the RPE-value, which was divided into 4 categories (1 = 6–9, 2 = 10–12, 3 = 13–16, and 4 = 17–20). The transformation was based on the MetPro®-physical activity analysis program (Kuntoväline Ltd, Oltermannintie 8, 00620 Helsinki, Finland) that contains previously published MET-values for approximately 2000 different activities. The reliability of the physical activity questionnaire, analysed with the MetPro®-physical activity program, has been acceptable; kappa = 0.48–0.78 (Mälkiä, 1996) and intraclass correlation coefficient = 0.62–0.94 (Sjögren, 2006). The MET-values of the structured NW and RT activities were based on objective measurements and they have been reported previously in detail (Wasenius et al., 2014). The absolute and relative time-weighted average intensities (TWA-MET) and volume (MET-hours) of physical activity were calculated with the MetPro©-program as previously reported (Wasenius et al., 2013; Wasenius et al., 2014). If the reported hours per week were lower or higher than 168 hours, the time was multiplied with 1.0 MET (rest) and added or subtracted from the volume of MHPA subcategory, respectively. For those participants who had missing physical activity data during the intervention (n = 2 for 1 week, and n = 1 for 2 weeks) the data was imputed with data from the previous or following week. The mean measurement period of physical activity was 3.4 (SD 0.8) weeks before the intervention and 10.7 weeks (SD 1.8) during the intervention.

Statistical analysis

Within-group comparison was performed with Wilcoxon signed-rank test. To test whether the change in the determinants of physical activity (during the intervention – before the intervention) differed between the groups, the Kruskal-Wallis test was first applied, and then individual groups were compared with Mann-Whitney U-test with Bonferroni correction for post-hoc analyses. Associations between the variables were evaluated with Pearson's correlation coefficient and with linear mixed effect models for repeated measurements to adjust for random participants variation in the intercept or slope. The estimation was based on the restricted maximum likelihood (REML). According to Akaike's Information Criteria (AIC), a model with best-fit was reported. Extreme outliers were removed from the analysis. A p-value <0.050 was considered statistically significant. Statistical analyses were conducted with IBM SPSS Statistics, version 21.0 (IBM Corp., Armonk, New York, USA).

Results

No significant differences were found between the groups in the determinants of physical activity at baseline (Table 3). During the intervention there was no significant between or within the groups change in the volume of total physical activity (Figure 1). The mean volume of total LTPA however increased significantly in the NW-group compared to the C-group (Figure 1).

According to within the group analyses, the mean TWA-MET of LTPA and total LTPA decreased (-0.3 MET, SD 0.4, $p < 0.05$; and -0.6 MET, SD 0.6, $p < 0.05$, respectively) in the RT-group. In the NW-group, in addition to the increase in the volume of total LTPA (Figure 1), the mean volume of physical activities performed with a intensity of ≥50 of VO_{2peak} increased (23.4 MET-hours, SD 20.1, $p < 0.05$) from baseline to intervention. In the C-group only the volume of total LTPA decreased (-11.1 MET-hours, SD 16.9, $p < 0.05$) compared to baseline. No significant between or within the group differences were found for the physical activity determinants of OPA, CPA, MHPA, ATPA, non-LTPA, NEPA or SLEEP.

According to the linear mixed model adjusted for individual variation in the intercept, no significant association was found between the volume of SPEA and change in NEPA in the RT group (Estimate = 0.929, 95% CI, -0.925 – 2.783, p = 0.322) or in the NW-group (Estimate = 0.085, 95% CI, -0.859 – 1.029, p = 0.858). However, one MET-hour increase in the total LTPA was associated with a decrease of -0.24 MET-hour (95% CI, -0.45: -0.02; p = 0.030) in the volume of NLTPA in the RT-group. While in the NW-group one MET-hour increase in the volume of total LTPA resulted in a decrease of -0.56 MET-hour (95% CI, -0.68 – -0.44; $p < 0.001$) in the volume of NLTPA.

No significant correlation was found between the baseline characteristic (age, BMI, fat percentage, VO_{2peak}) and the change in the volume of total physical activity, NEPA, or NLTPA in any of the groups. In the RT-group

Table 3. Baseline intensity, relative intensity, and volume of physical activity in different subcategories. Values are mean (standard deviation).

Determinants of physical activity (PA)		Resistance training (n = 8)	Nordic walking (n = 7)	Control (n = 8)
Absolute intensity	Occupational PA (MET)	2.4 (.9)	2.0 (.6)	2.6 (1.3)
	Commuting PA (MET)	2.6 (2.0)	2.4 (1.3)	2.0 (.9)
	Leisure-time PA (MET)	4.7 (1.2)	5.0 (.9)	5.1 (.9)
	Total leisure-time PA (MET)	4.7 (1.2)	5.0 (.9)	5.1 (.9)
	Miscellaneous home PA (MET)	1.8 (.7)	1.5 (.2)	1.6 (.3)
	Activity time PA (MET)	2.6 (.8)	2.2 (.7)	2.7 (.8)
Relative intensity	Occupational PA (%)	30.5 (13.3)	24.8 (6.7)	29.7 (14.3)
	Commuting PA (%)	30.3 (17.2)	28.8 (12.4)	22.3 (5.7)
	Leisure-time PA (%)	58.6 (13.5)	63.6 (17.5)	57.6 (10.9)
	Total leisure-time PA (%)	58.6 (13.5)	63.6 (17.5)	57.6 (10.9)
	Miscellaneous home PA (%)	23.5 (11.1)	18.3 (2.1)	17.9 (3.6)
	Activity time PA (%)	33.0 (10.3)	27.8 (7.6)	31.3 (9.5)
Volume	Occupational PA (METh·wk^{-1})	100.9 (31.5)	95.0 (68.0)	97.7 (38.1)
	Commuting PA (METh·wk^{-1})	18.6 (21.2)	10.7 (6.6)	10.1 (5.8)
	Leisure-time PA (METh·wk^{-1})	34.2 (27.1)	23.0 (24.8)	29.2 (12.7)
	Total leisure-time PA (METh·wk^{-1})	34.2 (27.1)	23.0 (24.8)	29.2 (12.7)
	Miscellaneous home PA (METh·wk^{-1})	101.5 (48.8)	97.3 (47.1)	104.2 (10.7)
	Sleep (METh·wk^{-1})	44.7 (7.5)	49.7 (6.0)	43.7 (6.4)
	Activity time PA (METh·wk^{-1})	153.6 (58.8)	128.8 (77.8)	137.1 (40.7)
	Non-exercise PA (METh·wk^{-1})	255.1 (83.1)	226.1 (63.5)	241.3 (43.5)
	Non-leisure-time PA (METh·wk^{-1})	221.0 (70.2)	203.1 (56.3)	212.0 (43.9)
	≥50% VO_{2peak} PA (METh·wk^{-1})	99.4 (104.7)	32.0 (25.8)	73.6 (45.9)
	Total PA (METh·wk^{-1})	299.8 (82.4)	275.9 (59.3)	285.0 (49.2)

MET = metabolic equivalent of task; METh = MET-hours; Relative intensity = percentage of absolute intensity from the individual VO_{2peak} expressed in MET-values; VO_{2peak} = peak oxygen uptake. There were no significant differences between the groups.

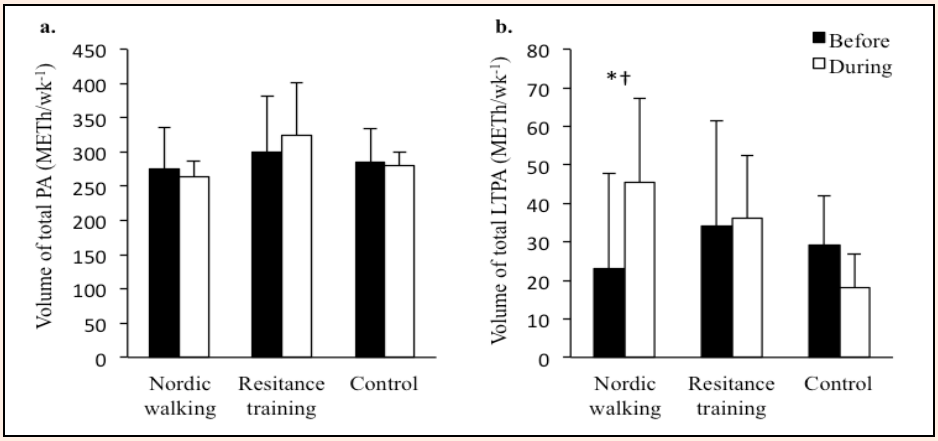

Figure 1. Changes in the volume of total physical activity (a.) and total leisure-time physical activity (b.) with a 12-week structured exercise intervention. PA = physical activity; LTPA = leisure-time physical activity; METh = MET-hours. *p-value is <0.05 compared to the change in control group, †p-value is <0.05 compared to before value.

the changes in absolute and relative TWA-MET of total LTPA were significantly correlated with baseline BMI (r = 0.742, p = 0.035 and r = 0.795, p = 0.018, respectively). No significant correlations were found in the NW-group nor in the C-group between the baseline characteristics and the change in intensity or volume of total LTPA.

Discussion

The main finding of this study was that a 12-week structured NW or RT intervention did not increase the volume of total physical activity. The volume of total LTPA, however increased in the NW-group compared to the non-exercise control group. When a model was built around the weekly change in the physical activity, we found a clear pattern of compensatory behaviour. In the NW-group a 1 MET-hour increase in the volume of total LTPA was associated with a 0.56 MET-hour decrease in the NLTPA. This type of association was also found in the RT-group. Interestingly no association was found between the volume of structured exercise and change in the volume of NEPA. These findings would suggest that of the intervention induced increase in the volume of total LTPA, approximately 50% in the NW group and 25% in the RT-group is compensated by a decrease in the volume of NLTPA. The findings also suggest an individuality of compensatory behaviour that may not be fully covered by group means.

The results of this study suggest that the NW intervention increased the volume of high intensity physical activities without affecting the volume of total physical activity. Consistently to our study, it has been reported that an 8-week walking intervention increased time spent on vigorous activities without altering the total energy expenditure in obese middle-aged women (Colley et al., 2010). These findings would indicate that lower intensity habitual physical activities are more strongly affected by compensatory behavior than habitual high-intensity physical activities that are similar to intervention activities. Thus, it is possible that structured NW exercises do not compete with habitual non-structured exercises. Actually, NW but not RT intervention could, at least in some individuals, result in increased non-structured exercise activity. To support this, we found a tendency ($p > 0.050$) for increased volume of non-structured LTPA in the NW-group and a significant decrease in the C-group. In the RT-group, no change in total physical activity or non-exercise physical activity with resistance training intervention was found, which is consistent with a previous study (Rangan et al., 2011). The current understanding of compensatory behavior is, however a controversial and highly debated an issue (Gomersall et al., 2013). The direct comparison of previous studies is also difficult due to differences in the samples and intervention characteristics and applied physical activity methods.

There are multiple possible regulatory pathways for compensatory behavior, including what types of activities were replaced (low or high intensity), degree of post-exercise fatigue, reward behaviour, and possibly genetic susceptibility (King et al., 2007). In the present study we investigated whether the baseline characteristics were associated with compensation. No such association was found for any of the measured characteristic including VO_{2peak}, which has previously been inversely associated with compensatory behaviour (Colley et al., 2010). In the RT-group, however, a higher BMI at baseline was associated with a higher intensity of total LTPA. The mean intensity of RT of participants with greater BMI tended to be higher compared to the participants with lower BMI (data not shown). In addition, the intensity of RT was lower than that of the LTPA, which resulted in decreased intensity of total LTPA in the RT group. Thus, the effect of intensity of RT on the intensity of total LTPA would be less in a higher BMI group, which would explain the found associations.

In the present study, structured exercise accounted only for a small portion of the total volume of physical activity, whereas the intensity of structured exercises of all subcategories was highest in the NW-group and the second highest in the RT-group. These findings imply that a change in the volume of total physical activity alone may be insufficient to determine whether the exercise intervention was beneficial health wise. It is not necessary to increase the volume of total physical activity to induce positive changes in health related variables, if a sufficient change in the intensity profile is achieved. Physiologically, changes in intensity are more likely than changes in volume to alter body homeostasis and thus induce positive adaptive responses to exercise. Based on the present study, exercise interventions, especially NW, seem to be more prone to induce changes in the daily intensity profile of physical activity rather than in the volume profile. It is clear that increasing current understanding about the factors that drive individual compensatory behaviour is an important future field of research. This would enable more specific prescription of exercise as a treatment.

To substantiate the effect of structured exercise on habitual physical activity or measured outcome, it is vital to analyse the change in physical activity throughout the intervention period. In this study we achieved this goal, which could be viewed as a major strength. After all, the ability of the exercise intervention to induce positive changes in health related outcomes is dependent on the intervention's ability to increase the dose of regular daily physical activity (Kesäniemi et al., 2001).

There are limitations in this study that should be taken into account when interpreting the findings. In this study a specific physical activity questionnaire was applied throughout the intervention, whereas in previous studies, different combinations of more objective measurements have been used, including heart rate, indirect calorimeter, accelerometers, and doubly labelled water (Goran and Poehlman, 1992; Hollowell et al. 2009; Morio et al., 1998). Although these methods are arguably accurate in measuring the amount of energy expenditure (International Organization For Standardization, 2004), they are in fact insufficient in providing information about the intensity, type of activity, or sociological context (Butte et al., 2012). Information regarding all aspects of behavior of physical activity can be gathered with questionnaires (Haskell, 2012). Moreover, physical activity questionnaires have been shown to be suitably reliable (Helmerhorst et al., 2012) and they are also cost–effective in continuous monitoring of physical activity. Although the criterion validity of the questionnaires against the objective monitors has been low (Helmerhorst et al., 2012, Prince et al., 2008), it does not automatically indicate inferiority of the questionnaires. According to Haskell (2012), it is possible that the questionnaires and wearable monitors detect different features of physical activity, thus making them complementary partners rather than excluding rivals. In addition, as previously reported (Hollowell et al., 2009) the large number of incomplete data related to accelerometer measurements indicates that objective measures may not be feasible in measuring total physical activity throughout the 12-week exercise intervention. Accelerometers would also require some type of diary or log to collect information about the physical activity subcategories. The accuracy of the physical activity measurements, as applied here, can be estimated to be 10–20% (International Organization For Standardization, 2004).

Another limitation is the small sample size, which may have been underpowered to detect significant changes. The power calculations were not performed, since the change in physical activity was not the primary aim of the initial randomized controlled trial, which is a common approach in clinical trials. Due to the small and most likely a selective group of participants, our findings may well be specific for this particular group. Albeit, there were no significant difference in the baseline characteris-

tics between those included in the analysis and those excluded. Furthermore, small sample size exercise trials are still conducted widely. It can be argued that similar type of large inter-individual variation in non-structured physical activity exist in those studies as in the present study. Albeit, such studies may be sufficiently powered to detect changes in outcomes (e.g. biomarker), they may not be that for the physical activity. Therefore, such trials may be unpowered to induce systematic difference in the change in physical activity, which could partly explain the large inter-individual variability in response to equal dose of prescribed exercise (Bouchard and Rankinen 2001). Our data clearly shows that the physical activity response to structured exercise intervention is neither stable nor similar between individuals. This large inter-individual variation in our sample suggests that future exercise studies should aim for more robust methods to quantify and to analyse the actual change in physical activity induced by the training. Due to the limitations the results should be interpreted with caution and in the future larger more powerful sample sizes may be required.

Conclusion

Structured NW or RT intervention does not increase the volume of total physical activity in overweight or obese men with impaired glucose regulation. The NW intervention may increase the volume of higher intensity activities but it induces a compensatory decrease in other physical activities. Whether the increase induced by the NW is sufficient, to provide health benefits, remains to be determined. The present evidence also indicates that there is a large inter-individual variation in compensatory behavior, which should be recognized as a possible confounder of individual responses to structured exercise.

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Key points

- Structured NW or RT training does not increase the volume of total physical activity.
- NW intervention can increase the volume of higher intensity activities.
- The increased in volume of LTPA induced by the structured NW and RT interventions was associated with the decreased volume of NLTPA.

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