

Effects of active commuting to work for 12 months on cardiovascular risk factors and body composition

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Active commuting has the potential to decrease cardiovascular risk by increasing physical activity. We aimed to investigate the effects of active commuting to work for 12 months on body composition and cardiovascular risk factors. Therefore, 73 hospital employees (age: 46 ± 9 years, 36% males), with a predominantly passive way of commuting, were randomly assigned to an intervention group (IG) and a control group (CG) in a 2:1 fashion. The IG was further divided into a public transportation plus active commuting group (IG-PT) and a cycling group (IG-C). Both IGs were prompted to reach 150 min/wk of moderate intensity exercise. Daily self-reported commuting details were verified by GPS tracking. All subjects underwent assessment of body composition, resting blood pressure, glycemic control, and lipid profile at the beginning and end of the study. Data for final analyses were available in 62 subjects. Commuting details indicated that the subjects randomized to IG changed their commuting habits. HbA1c decreased by 0.2% [95%CI: -0.3, -0.2] in IG-PT but was not statistically different between groups ($P = .06$). LDL cholesterol decreased in IG-C by 0.8 mmol/L [-1.1, -0.4] and by 0.6 mmol/L [-1.2, 0.1] in IG-PT which can be considered biologically relevant but did not yield statistical significance. Body composition and blood pressure did not differ between groups. Active commuting to work for 12 months did not change body composition but yielded relevant changes in lipid profile and glycemic control. Health benefits of active commuting should be addressed by healthcare professionals when counseling individuals that seek to improve their cardiovascular risk profile.

KEYWORDS

active commuting, body composition, cardiovascular risk factors, lifestyle modification

1 | INTRODUCTION

In developed countries, cardiovascular diseases (CVD) are the number one cause of death with a projected increase in prevalence,¹ despite the fact that preventive strategies are well

established and easily accessible.² For the majority, the most effective CVD prevention strategy is to increase physical activity (PA).³⁻⁵ Most international guidelines recommend that individuals accumulate at least 30 min/d, 5 d/wk of moderate intensity PA (ie, 150 min/wk in total)^{6,7} but the majority of

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people in developed countries (about 75%) does not meet this target.⁶ Considering the increasingly sedentary lifestyle,⁸ active commuting by means of walking or cycling to and from work is a type of PA that can be integrated into everyday life.⁹ However, active commuting to work has declined in high-income countries in recent decades.¹⁰ Therefore, encouraging population-wide engagement in PA is a key priority for most stakeholders in healthcare systems to address the growing burden of CVD.

Active commuting has previously been associated with lower risk of CVD and all-cause mortality in population-based observational studies.^{3,11,12} PA unfolds its CVD preventive effect via modulation of classic cardiovascular risk factors, including body mass-, blood pressure-, blood glucose-, and lipid-lowering effects. In a recent Danish study, 6 months of bike commuting to work for 5 d/wk improved peripheral insulin sensitivity and body composition in overweight or class 1 obese individuals.¹³ Using the same study design, in which exercise intensity recommendations were monitored in addition to weekly contact between the investigators and participants, lipid profile did not improve.¹⁴

In the present prospective, randomized, and controlled study, we aimed to investigate whether achieving lifestyle changes by introducing active commuting to work verified by wearable GPS trackers, without exercise intensity requirements and thus resembling a real-world intervention, improve CVD risk factor profile and body composition over a time span of 12 months.

2 | METHODS

2.1 | Study approval and registration

The data derived from participants of the GISMO (Geographical Information Support for Healthy Mobility) study. The study was performed in accordance with the Declaration of Helsinki and its current amendments and was approved by the Ethical Committee of the Paris Lodron University Salzburg, Austria (EK-GZ: 43/2016). The study was registered at ClinicalTrials.gov (NCT03098719).

2.2 | Subject recruiting

The recruiting was carried out within the local hospital, Salzburger Landeskliniken, with approximately 6500 employees. In total, 655 possible participants were contacted via phone calls and 6392 E-mails were sent out with permission of the hospital directorate. A total of 300 employees were initially interested in participating. Of these, 223 were not invited for baseline assessment due to reasons

such as already actively commuting, or known physical or psychological conditions, which would have made the conclusion of the study difficult. A more detailed description of the study design and the recruitment process is provided elsewhere.^{15,16}

2.3 | Study population and group assignment

Seventy-three hospital employees with a predominantly passive way of commuting to work were randomly assigned into an intervention (IG) or a control group (CG) in a 2:1 fashion. The IG was further divided into a public transportation/active commuting (IG-PT) and cycling (IG-C) group, depending on the subject's availability of cycling paths, public transportation, and personal preference. Both IG groups were prompted to reach the WHO recommendation of 150 minutes of moderate intensity PA per week during their commute for 12 months. The CG was asked not to change their passive mode of commuting. Detailed information about medical history and current medication are reported elsewhere.¹⁵ All subjects provided written informed consent before study inclusion.

2.4 | Physical activity assessment

Each subject kept a diary to evaluate distances covered during active commuting. Additionally, subjects were given a wearable device with an integrated GPS tracker and optical heart rate sensor (Polar M200, Polar Electro Oy, Finland) for two weeks at the beginning as well as at the end of the study, to validate diary entries.¹⁷

2.5 | Body composition and cardiovascular risk factor assessment

Data were collected at the beginning and the end of the study. Anthropometric data were obtained by measuring body mass, height, waist, and hip circumferences following standardized procedures.¹⁸ Body mass index (BMI) was calculated according to the formula of Keys.¹⁹ Sum of skinfold thicknesses was measured with Harpenden callipers using the four site skinfold measurement.²⁰ Systolic and diastolic blood pressure (BP) was measured once at rest by the auscultatory method using an adequately sized upper arm cuff and a sphygmomanometer with the participant in a supine position. Venous blood samples were drawn after a 10 hours overnight fast. Fasting glucose, HbA1c, total cholesterol (CHOL), high-density lipoprotein cholesterol (HDL), and triglycerides (TRI) were analyzed in plasma by

TABLE 1 Baseline characteristics of all participants, participants of control group (CG), public transportation plus active commuting group (IG-PT), and cycling group (IG-C)

	All participants (N = 62)	CG (N = 17)	IG-PT (N = 23)	IG-C (N = 22)	P
Age, years	46 [44, 49]	45 [39, 50]	47 [43, 51]	47 [44, 51]	.666
Men, %	36	29	30	45	
Anthropometrics					
Height (cm)	171 [169, 173]	171 [167, 174]	169 [165, 173]	174 [169, 178]	.197
Body mass (kg)	76.5 [72.3, 80.7]	77.7 [67.3, 88.2]	73.5 [67.2, 79.8]	78.7 [71.9, 85.5]	.542
BMI (kg/m ²)	26.0 [24.9, 27.2]	26.4 [23.6, 29.3]	25.8 [23.9, 27.6]	26.0 [24.2, 27.7]	.897
Waist circumference (mm)	91.1 [87.7, 94.4]	92.6 [84.2, 101.0]	89.1 [84.2, 94.1]	91.9 [86.4, 97.4]	.672
Hip circumference (mm)	103.2 [100.3, 106.1]	101.8 [95.1, 108.6]	103.3 [97.9, 108.8]	104.1 [100.4, 107.8]	.821
Waist/hip ratio	0.88 [0.86, 0.90]	0.91 [0.86, 0.96]	0.86 [0.84, 0.89]	0.88 [0.84, 0.92]	.211
Skinfold (mm)	83.9 [74.7, 93.1]	88.9 [65.5, 112.3]	83.2 [68.0, 98.4]	80.8 [67.6, 94.0]	.783
Blood pressure					
RR _{sys} (mm Hg)	114 [111, 117]	114 [107, 120]	112 [108, 117]	117 [110, 123]	.488
RR _{dia} (mm Hg)	72 [69, 75]	71 [66, 75]	71 [67, 75]	75 [69, 80]	.406
Glucose metabolism					
GLU (mg/dL)	78.0 [74.7, 81.3]	78.2 [71.3, 85.0]	75.3 [79.8, 75.3]	80.7 [74.0, 87.4]	.387
HbA1c (%)	5.3 [5.3, 5.4]	5.4 [5.2, 5.5]	5.3 [5.3, 5.4]	5.4 [5.2, 5.5]	.940
Lipid metabolism					
TRI (mg/dL)	90.9 [79.9, 101.9]	86.0 [59.6, 112.5]	98.0 [80.4, 115.5]	87.2 [69.3, 105.2]	.619
CHOL (mg/dL)	205.3 [196.6, 214.0]	200.2 [180.3, 220.0]	211.8 [196.3, 227.0]	202.4 [189.4, 215.4]	.513
CHOL (mmol/L)	5.3 [5.1, 5.5]	5.2 [4.7, 5.7]	5.5 [5.1, 5.9]	5.2 [4.9, 5.6]	
HDL (mg/dL)	74.3 [68.2, 80.3]	83.7 [65.9, 101.5]	68.4 [60.9, 75.9]	73.1 [65.3, 80.9]	.127
HDL (mmol/L)	1.9 [1.8, 2.1]	2.2 [1.7, 2.6]	1.8 [1.6, 2.0]	1.9 [1.7, 2.1]	
LDL (mg/dL)	113.4 [105.6, 121.2]	101.1 [89.9, 112.3]	123.9 [108.4, 139.4]	112.0 [100.0, 124.3]	.062
LDL (mmol/L)	2.9 [2.7, 3.1]	2.6 [2.3, 2.9]	3.2 [2.8, 3.6]	2.9 [2.6, 3.2]	
LDL/HDL ratio	1.7 [1.5, 1.9]	1.4 [1.1, 1.7]	2.0 [1.6, 2.4]	1.6 [1.4, 1.9]	.050
Cardiovascular risk					
FRS score	2.4 [1.6, 3.1]	3.1 [0.8, 5.3]	2.2 [1.1, 3.2]	2.1 [1.1, 3.0]	.570
HS score	0.50 [0.33, 0.67]	0.57 [0.10, 1.04]	0.50 [0.20, 0.81]	0.46 [0.29, 0.63]	.882

Note: Data are unadjusted mean [95% CI]. Statistics: one-way ANOVA.

Abbreviations: BMI, Body mass index; CHOL, Total cholesterol; Circ, Circumference; FRS score, Framingham Risk Score; GLU, fasting blood glucose; HbA1c, Hemoglobin A1c; HDL, HDL Cholesterol; HS score, HEART Score of the European Society of Cardiology (ESC); LDL, LDL Cholesterol; RR_{dia}, diastolic blood pressure; RR_{sys}, systolic blood pressure; Skinfold, Sum of 3-point skinfold thickness measured with the calliper method; TRI, Triglycerides.

enzymatic methods with a cobas 8000 (Roche Diagnostics). Low-density lipoprotein cholesterol (LDL) was calculated according the Friedewald Equation.²¹

2.6 | Cardiovascular risk scores

Two widely used cardiovascular risk scores were calculated for every subject: the Framingham Risk Score (FRS)²² and the HEART Score (HS) of the European Society of Cardiology (ESC).²³ FRS estimates the 10-year risk of a subject to develop coronary heart disease, while HS was designed to

estimate the 10-year risk of cardiovascular death. The FRS uses the following variables: age, male/female gender, total cholesterol, high-density lipoprotein (HDL) cholesterol, current smoking, and systolic blood pressure. The HS uses age, male/female gender, total cholesterol, current smoking, systolic blood pressure, and country.

2.7 | Statistical analyses

All variables were tested for normal distribution using the Kolmogorov-Smirnov test. Statistical comparison of

TABLE 2 Passive and active distances covered during commute to work from control group (CG), public transportation plus active commuting group (IG-PT), and cycling group (IG-C)

Covered distances	CG (N = 17)	IG-PT (N = 23)	IG-C (N = 22)	P
Walking, km/year	110 [19, 202]	305 [203, 407]	65 [12, 119]	<.001
Cycling, km/year	35 [0, 73]	450 [185, 716]	1673 [1159, 2187]	<.001
Public transportation, km/year	273 [0, 681]	1861 [357, 3366]	1248 [0, 2642]	.230
Car, km/year	2371 [30, 4711]	299 [73, 526]	322 [29, 616]	.016

Note: Data are unadjusted mean [95% CI]. Statistics: one-way ANOVA.

baseline values and distances covered during the commute to work between groups was performed by one-way ANOVA. Per-protocol comparison of outcome variables between groups was calculated by one-way ANCOVA using baseline values and gender as covariates and subsequent post hoc testing with Bonferroni correction. A two-way ANCOVA was performed to test whether the intervention was more effective in overweight individuals. Descriptive data are presented as unadjusted means and 95% confidence intervals (CI) or unadjusted means and 95% CIs of the deltas between beginning and end of the study. A *P* value of $\leq .05$ (two-sided) was considered statistically significant. Cohen's *d* was calculated to estimate effect sizes. All statistical analyses were performed with IBM SPSS Statistics version 24 (SPSS, Inc).

3 | RESULTS

Overall, data for final analyses were available in 62 subjects. Thereof, 21 were working as nurses, 9 as physicians, 20 working in administration, and 12 practiced various other professions within the hospital. Baseline characteristics of all participants are presented in Table 1. Passive and active distances covered during participant's commute to work are presented in Table 2. Detailed baseline characteristics including medication, adherence to the study protocol, and study dropouts are outlined elsewhere.^{15,16} Mobility data prove high compliance to the respective activity recommendations for CG and the two intervention groups.¹⁵

3.1 | Body composition

At baseline, 58% of subjects were overweight or obese (CG: 53%; IG-PT: 57%; IG-C: 64%) as expected in a predominant sedentary study population. Changes in body composition variables did not differ between groups (Table 3). No significant dose-response relationship could be observed regarding body composition.²⁴ In a subanalysis where both interventions (IG-PT and IG-C) were pooled, no statistically significant difference between overweight compared to

normal body mass participants with regard to anthropometric, blood pressure, lipid, and glucose profile variables were observed (all *P* > .1).

3.2 | Blood pressure, lipid profile, and glycemic control

Changes in lipid profile, glycemic control and blood pressure did not differ statistically significantly between groups (Table 3). However, LDL decreased in IG-C by 13% (−0.8 [−1.1, −0.4]) and in IG-PT by 8% (−0.6 [−1.2, 0.1]; $p_{\text{Groups}} = 0.395$). HbA1c decreased by 0.2% [95% CI: −0.3, −0.2] in IG-PT and by 0.1% [95% CI: −0.2, 0] in CG, but was not statistically different between groups (*P* = .06; Table 3). No significant physical activity dose-response relationship could be observed regarding HDL.²⁴

3.3 | Cardiovascular risk scores

Changes in FRS and HS scores did not differ significantly between groups (Table 3).

4 | DISCUSSION

In the present study, active commuting to work for 12 months did not change body composition but yielded relevant changes in glycemic control and lipid profile.

HbA1c is the gold standard for monitoring glycemic control, and it has been shown that HbA1c levels starting with 5.5% were associated with increased incidence of ischemic stroke, coronary heart disease, and all-cause death in non-diabetic individuals.²⁵ Physical activity programs improve glycemic control among both diabetic and non-diabetic populations,²⁶ and exercise of more than 150minutes a week was associated with HbA1c reductions of 0.89%.²⁷ In the present study, the mean HbA1c was 5.3% at study entry and we observed a borderline significant difference in HbA1c between groups (*P* = .06) and improvement in IG-PT (−0.2%) compared to CG (*P* = .06). Considering that elevated HbA1c

TABLE 3 Changes in outcomes following 12 months of active commuting of all participants and participants of control group (CG), public transportation plus active commuting group (IG-PT), and cycling group (IG-C)

	CG (N = 17)	IG-PT (N = 23)	IG-C (N = 22)	<i>P</i> (Effect size)	<i>P</i> _{CG vs IG-PT}	<i>P</i> _{CG vs IG-C}
Anthropometrics						
Body mass (kg)	0.9 [−1.1, 2.8]	−0.4 [−1.5, 0.7]	0.4 [−1.6, 2.4]	.675 (0.014)	.999	.999
BMI (kg/m ²)	0.3 [−0.4, 1.0]	−0.2 [−0.5, 0.2]	0.1 [−0.5, 0.8]	.458 (0.027)	.811	.999
Waist circumference (mm)	−0.3 [−2.0, 1.4]	−1.6 [−2.9, −0.3]	0.5 [−1.9, 2.9]	.105 (0.076)	.955	.970
Hip circumference (mm)	1.5 [−1.6, 4.6]	−1.3 [−2.8, 0.3]	0.1 [−1.7, 1.8]	.162 (0.062)	.174	.697
Waist/hip ratio	0 [−0.1, 0.0]	0 [0, 0]	0 [0, 0]	.328 (0.038)	.999	.999
Skinfold (mm)	−8 [−19, 3]	−9 [−14, −4]	−3 [−10, 3]	.343 (0.037)	.999	.999
Blood pressure						
RR _{sys} (mm Hg)	3 [−3, 9]	3 [−3, 9]	3 [−4, 8]	.861 (0.005)	.999	.999
RR _{dia} (mm Hg)	7 [3, 12]	7 [3, 10]	7 [2, 11]	.971 (0.001)	.999	.999
Glucose metabolism						
GLU (mg/dL)	5.0 [−4.3, 14.3]	8.9 [2.2, 15.6]	5.7 [−2.0, 13.3]	.678 (0.014)	.999	.999
HbA1c (%)	−0.1 [−0.2, 0]	−0.2 [−0.3, −0.2]	−0.2 [−0.2, −0.1]	.058 (0.097)	.060	.999
Lipid metabolism						
TRI (mg/dL)	6.8 [−19.4, 33.0]	9.9 [−9, 28.7]	−4.7 [23.9, 14.5]	.130 (0.069)	.999	.688
CHOL (mg/dL)	−8 [−20.8, 4.6]	−8 [21.4, 5.4]	−18.7 [−28.1, −9.2]	.193 (0.056)	.999	.762
CHOL (mmol/L)	−0.5 [−1.2, 0.3]	−0.5 [−1.2, 0.3]	−1.1 [−1.6, 0.6]	.193 (0.056)		
HDL (mg/dL)	−8.5 [−20.7, 3.6]	−2.7 [7.0, 1.6]	−4.4 [8.7, 0]	.763 (0.009)	.999	.688
HDL (mmol/L)	−0.5 [−1.2, 0.3]	−0.5 [−1.2, 0.3]	−1.1 [−1.6, 0.6]	.763 (0.009)		
LDL (mg/dL)	−1.5 [−10.5, 7.6]	−10.3 [−21.9, 1.4]	−13.3 [−21.2, −5.3]	.395 (0.032)	.999	.543
LDL (mmol/L)	−0.1 [−0.6, 0.4]	−0.6 [−1.2, 0.1]	−0.8 [−1.1, −0.4]	.395 (0.032)		
LDL/HDL ratio	0.1 [−0.1, 0.3]	−0.1 [−0.4, 0.2]	−0.1 [−0.3, 0]	.483 (0.025)	.999	.766
Cardiovascular risk						
FRS score	−0.1 [−1.4, 1.3]	0.4 [−0.3, 1.0]	−0.5 [−1.0, 0.1]	.234 (0.050)	.999	.810
HS score	0.0 [0, 0]	0.0 [0, 0]	0.0 [0, 0]	.342 (0.037)	.507	.707

Note: Data are unadjusted mean (95% CI). Statistics: ANCOVA with baseline values and gender as covariates with post hoc pairwise comparison adjusted for multiple comparisons (Bonferroni correction).

Abbreviations: BMI, Body mass index; CHOL, Total cholesterol; Circ, Circumference; FRS score, Framingham Risk Score; GLU, Fasting blood glucose; HbA1c, Hemoglobin A1c; HDL, HDL Cholesterol; HS score, HEART Score of the European Society of Cardiology (ESC); LDL, LDL Cholesterol; RR_{dia}, diastolic blood pressure; RR_{sys}, systolic blood pressure; Skinfold, Sum of 3-point skinfold thickness measured with the calliper method; TRI, Triglycerides.

levels of $\geq 5.5\%$ results in an increased CVD risk,²⁵ it is likely that the magnitude of improvement in glycemic control observed in the present study might have lead to long-term benefits regarding CVD health.

Current international recommendations suggest tailoring intervention strategies for the prevention of CVD based on total CVD risk.⁷ Considering the well-established, direct relationship between dyslipidemia and CVD,²⁸ interventions that improve lipid profiles are considered a main pillar in preventive cardiovascular medicine.⁷ In a meta-analysis of studies involving 12 weeks or more of aerobic exercise, LDL fell by 5% and triglyceride levels fell by 3.7% whereas HDL cholesterol increased by 4.6%.²⁹ In a study conducted by de Geus et al, 65 subjects were asked to cycle to work at least

three times a week for 12 months and LDL decreased and HDL increased significantly in this group.³⁰ In contrast, in a controlled study conducted by Gram et al overweight and obese subjects were asked to cycle to work for 6 months and no effects with regard to lipid profile was observed between groups.¹⁴ Our results are in line with the results of Gram et al, as we did not observe a statistically significant difference between groups. However, LDL decreased in IG-C by 13% (−0.8 mmol/L) and by 8% (−0.6 mmol/L) in IG-PT. Considering that long-term exposure to 1 mmol/L lower LDL has been associated with a 55% reduction in CVD risk,³¹ the CVD preventive effect of active commuting to work could be considered relevant in the present study. Regular exercise training has been repeatedly shown to result in an increase

in HDL and decrease in TRI. In the present study, such effects could not be documented, which might be due to the lower CVD risk in our study subjects and the absence of clear dietary recommendations. Also, no recommendations were given with regard to exercise intensity, which has previously been shown in the Copenhagen City Heart Study³² to have an impact on lipid profiles.

Previous population-based, long-term observational studies have reported that compared with car commuters, active commuters had lower baseline BMI and percentage body fat, and lower rates of hypertension.^{10,33-35} The present interventional, randomized, and controlled study could not confirm these effects which is in line with the results of the study conducted by de Geus et al.³⁰ Differences in study design like selection bias in non-randomized studies may contribute to these inconsistent findings. In addition, in contrast to other studies demonstrating weight loss secondary to an active commuting intervention,^{13,36} our study participants had lower BMI, and thus, weight loss and also other concomitant improvements are more challenging to achieve.

This study has some limitations. No a priori sample size calculation was performed since the sample size was defined by the willingness of includable hospital staff to participate in the study. However, great effort was undertaken to recruit as many participants as highlighted by the recruitment process provided elsewhere.¹⁶ Exercise intensity during active commuting was not regularly monitored. Thus, insufficient exercise intensity during active commuting might have attenuated our results. However, the lack of exercise intensity requirements better resembles a real-world intervention. Another limitation is that full nutrition information of study participants was not available. Finally, the study design did not include data about physical activity during leisure-time activities. In conclusion, the present study demonstrates that active commuting to work for 12 months does not change body composition but yields relevant changes in lipid markers and glycemic control. These changes have the potential to impact further prevention strategies such as the necessity of lipid-lowering or antidiabetic medication.

5 | PERSPECTIVE

Previous, mainly observational, studies have reported that active commuting has beneficial effects on body composition and most major CVD risk factors. Our results indicate that active commuting to work for 12 months improves glycemic control and lipid profile whereas other risk factors like body composition were not affected. Still, considering the magnitude of HbA1c and LDL reduction by active commuting in the present study, benefits of active commuting should be addressed by healthcare professionals when counseling individuals that seek to improve their cardiovascular risk profile.

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CONFLICT OF INTEREST

There is no conflict of interest to report.

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