Title: **Effect of short-term reduced physical activity on cardiovascular risk factors in active lean and overweight middle-aged men**

**Running title:** Reduced activity in overweight and lean men

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**Abstract**

**Objectives:** An experimental reduction in physical activity is a useful tool for exploring the health benefits of physical activity. This study investigated whether similarly-active overweight men show a more pronounced response to reduced physical activity than their lean counterparts because of their athrogenic phenotype (i.e., greater abdominal adiposity). **Methods:** From 115 active men aged 45-64 years, we recruited nine active lean (waist circumference <84 cm) and nine active central overweight men (waist circumference >94cm). Fasting blood samples and responses to an oral glucose tolerance test (OGTT) were measured at baseline and following one week of reduced physical activity to simulate sedentary levels (removal of structured exercise and reduced habitual physical activity). **Results:** Glucose and insulin areas under the curve (AUC), CRP, ALT, TAG were all higher in the overweight group and remained so throughout ($P < 0.05$). Insulin and glucose AUC responses to an OGTT, as well as fasting triglyceride (TAG) concentrations, increased in both groups as a result of the intervention ($P < 0.05$). There was no change in interleukin-6, C-reactive protein (CRP), Tumour Necrosis Factor-α, soluble intracellular adhesion molecule 1, or alanine transaminase (ALT). **Conclusion**: One-week of reduced activity similarly-impaired glucose control and increased fasting TAG in both lean and overweight men. Importantly, in spite of very similar (high) levels of habitual physical activity, central overweight men displayed a poorer profile for various inflammatory and metabolic outcomes (CRP, ALT, TAG, glucose AUC and insulin AUC).

**Keywords:** Overweight, Abdominal adiposity, Physical Activity, Inflammation, Exercise

**List of abbreviations**

ANOVA: Analysis of variance

AUC: Area under the curve

ALT: Alanine Transaminase

BMI: Body mass index

CRP: C-reactive protein

DEXA: dual energy X-ray absorptiometry

HDL: High density lipoprotein

IL-6: Interleukin-6

LDL: low density lipoprotein

METs: Metabolic equivalents

OGTT: Oral glucose tolerance test

PAL: Physical activity level

sICAM-1: soluble intercellular adhesion molecule-1

TAG: Triglyceride

TEE: Total energy expenditure

TNF-α: Tumour necrosis factor- α

WBC: White blood cells

WHO: World Health Organisation

## Introduction

Exercise intervention studies generally support the notion that regular exercise lowers markers of inflammation and improves metabolic function; cf. [1]. Such studies demonstrate the impact of a *positive* change in behaviour (i.e., increased physical activity). An alternative experimental model that arguably more closely reflects the shift towards a more sedentary society is to examine the effects of *reduced* physical activity in active individuals [2].

Even in young men, removal of structured exercise through short-term detraining leads to large changes in measures of glucose control [3] and lipid metabolism [4]. Furthermore, Gill *et al* [4] found an increase in tumour necrosis factor-α (TNF-α) over a one-week detraining period in young men. However, this study reported no change in Interleukin-6 (IL-6) and we recently reported that the removal of structured exercise in active middle-aged men had no impact on various markers of inflammation, including IL-6 [5]. Importantly, these studies sought to remove *structured exercise* in a classical view of 'detraining'. However, even in regular exercisers, structured exercise represents only a small proportion of total physical activity energy expenditure [6, 7]. Arguably, a more powerful approach that better simulates true sedentary behaviour is to reduce *total* physical activity. Olsen *et al*. [8] and Krogh-Madsen *et al*. [9] achieved this by asking young, active (but untrained) subjects to reduce their daily step count (and hence *total* physical activity thermogenesis) over a two-week period. In these studies, various measures of insulin sensitivity deteriorated (e.g., plasma insulin area under the curve following an oral glucose tolerance test increased by 79%; [8]). Whilst there was no increase in markers of inflammation for this population, it is unclear whether this is also true for middle-aged participants who tend to have elevated circulating concentrations of inflammatory markers [10].

Central adiposity is a risk factor for elevated markers of inflammation [11] and metabolic dysfunction [12]. It has been proposed that regular exercise is anti-inflammatory [13] and there has been some debate over whether it is possible to be 'fat but fit' [14]. It is conceivable that highly active individuals with increased central adiposity are protected against an increase in markers of inflammation and metabolic dysfunction because of their high physical activity [15, 16]. In this context, we postulated that central overweight and lean middle-aged men with similar high levels of physical activity would have similar metabolic and inflammatory profiles; but, that a reduction of *total* physical activity to sedentary levels in central overweight active middle-aged men would lead to a rapid loss of metabolic and inflammatory homeostasis in comparison to their lean counterparts because of their greater central adiposity (i.e., the loss of physical activity would lead to a relatively greater disturbance because of their pro-atherogenic central adiposity phenotype).

## Materials and Methods

### *Experimental Design*

In order to examine the interaction between adiposity and the impact of reduced physical activity in middle-aged men, we set out to recruit two groups of similar (highly) active middle-aged men; one lean group and one central overweight group. As discussed in more detail below, in order to be included in the study, men had to meet two separate criteria (i.e., being highly active as well as having a carefully defined level of central adiposity).

### *Subjects*

Subjects were recruited via local advertisement following local National Health Service ethics approval and after subjects had given written informed consent. The sample size was determined using previous data which indicated a standard deviation of 0.51 pg/ml for fasting IL−6 [17]. Calculations showed that with 80% power and 5% alpha, 11 subjects were required in each group to detect a clinically relevant change in IL−6 of 0.6 pg/ml. Due to the low proportion of eligible subjects for the rigid criteria that were employed and high exclusion rate (described below), recruitment was terminated after 14 months when actual subject numbers were n = 9 men in each group. Subjects were 45-64 years old (Table 1). Exclusion criteria included smoking, current use of medication and/or presence of any co-morbidity (e.g., type 2 diabetes).

After advertisement, 115 men considered themselves eligible and volunteered to take part (Figure 1). Initial eligibility was via a questionnaire to determine whether subjects were likely to meet the physical activity requirements and measurement of waist circumference [18]. To ensure that groups differed in central adiposity, only individuals who had a waist circumference of less than 84 cm [19] or greater than 94 cm (the cut-off point for increased risk of metabolic disease as defined by the WHO [20]) were recruited for the lean and overweight groups, respectively.

Central adiposity was used as an inclusion criterion as it has been shown to be a better predictor of obesity-related health risks than other measures such as BMI [12]. In subjects that were not excluded at this stage, physical activity energy expenditure was then estimated using synchronised accelerometry and heart rate (Actiheart, Cambridge Neurotechnology Ltd., Cambridge, UK). Subjects wore the monitor for seven whole consecutive days (day and night), recording data on a minute-by-minute basis [1, 21]. Subjects were instructed to remove the physical activity monitor only to change the electrodes. Subjects were not informed that the monitor recorded physical activity but that it recorded heart rate variability in order to avoid confounding from the Hawthorne effect (i.e., behavioural modification because of the act of being observed). Activity eligibility criteria were the same as those for active individuals in a previous study [17]. Briefly, this required participation in moderate/vigorous intensity activity for 30 minutes or more 5 times a week and vigorous intensity activity for a total of 90 minutes per week; with no reported change in physical activity over the previous 6 months.

***Preliminary measures***

We assessed cardio−respiratory fitness using an incremental treadmill-based test as previously described [17]. Briefly, this involved an incremental incline test on a treadmill (Woodway, ELG Weiss, Germany) comprised of 3-min exercise stages with the incline increased by 3% at the end of each stage until volitional fatigue. We also took several anthropometric measurements (i.e., height, body mass, blood pressure), a fasted preliminary blood sample and performed a dual energy X-ray absorptiometry (DEXA; Discovery, Hologic, Bedford, UK) scan in order to estimate body composition. Whole body and central abdominal adipose masses (combined central abdominal subcutaneous and visceral adipose tissue; were estimated from the DEXA scan [22]). The preliminary blood sample was taken to exclude any individuals who had elevated markers of inflammation during the intervention period due to external factors (e.g., acute infection or injury). Values for all inflammatory markers were excluded for one subject in the

lean group based on his CRP concentration at baseline being 10 times that measured in the preliminary blood sample.

***Reduced physical activity week***

Subjects were asked to try and reduce their step count to less than 4000 steps/day for the one-week intervention in order to replicate a sedentary lifestyle [23]. They were given a pedometer (Yamax Corp, Tokyo, Japan) to wear all day and asked to monitor their step count on a regular basis. Each subject was given a list of practical tips on how to reduce their step count. They were also instructed not to take part in any structured physical activity (i.e., playing sport, going to the gym). Subjects wore an Actiheart monitor during this period, so that physical activity level (PAL) and total energy expenditure (TEE) could be quantified [1, 21]. They were asked to maintain their normal diet during the intervention.

***Measurements***

On the morning of day 0 (baseline) and day 7, a venous blood sample was taken following an overnight fast (~12 h). An oral glucose tolerance test (OGTT) was then performed. Subjects consumed 75g of anhydrous glucose (113 ml of Polycal; Nutrica Clinical Care, Wiltshire, UK) and blood samples were taken, using finger prick blood sampling, every 15 minutes for the first hour and again after 2 hours. Two days before each trial day, subjects recorded their food and fluid intake. On the day before each trial day subjects were asked to refrain from the consumption of alcohol. Subjects were asked to perform a final typical structured moderate to vigorous intensity bout of exercise 36-48h before the start of the intervention and then to abstain from vigorous intensity exercise (this interval was introduced to reduce the likelihood that an acute increase in markers of inflammation would affect pre-intervention values [24].

***Analytical methods***

Subjects remained in a seated position 15 minutes prior to and during all blood sampling. Venepuncture blood samples were distributed into 5 ml plain and EDTA-treated tubes (Sarstedt Ltd., Leicester, UK). Whole blood glucose and lactate were measured using an automated analyzer (YSI 2300 STAT plus, Yellow Springs, OH). Blood cell counts were measured using an automatic hematology system (SF-3000, Sysmex, Milton Keynes, UK). Commercially available enzyme-linked immunosorbent assays (ELISA) were used to measure CRP (Diagnostic Systems Laboratories, Webster, TX), IL-6 (Quantikine, R & D Systems, Abingdon, UK), and soluble intercellular adhesion molecule-1 (sICAM-1; R & D Systems). Serum TNF- α and adiponectin were measured by ELISA (R & D systems Inc., Abingdon, UK). Immunoassays were used to measure plasma triglyceride (TAG), free fatty acids (FFA), alanine transaminase (ALT), total and high density lipoprotein (HDL) cholesterol (Cobas, Roche Diagnostics Limited, Burgess Hill, UK) and serum insulin (AutoDELFIA, Perkin Elmer, Waltham, Massachusetts, USA). Low density lipoprotein (LDL) cholesterol was calculated using the Friedewald equation [25]. During the OGTT, finger prick samples were obtained using a lancet (Accu-Check Softclix Pro, Roche, Lewes, UK). The first drop of blood was removed using a tissue and approximately 200 µl of whole blood was collected using a microvette tube coated with EDTA (CB300, Sarstedt Ltd) followed by another 200 µl of whole blood using a microvette tube coated with clot activator (CB300, Sarstedt Ltd). From these capillary samples, whole blood glucose, plasma glucose and serum insulin were determined as described above.

***Statistical analysis***

Statistical analysis was performed using SPSS 14.0 for Windows (SPSS INC, Chicago, Illinois). All values are expressed as means ± SEM. Statistical significance was set at a value of $P \leq 0.05$. Single baseline comparisons between groups were compared using *t*-tests. Two-way repeated measures ANOVA (repeated measures on time) were used to compare results between groups

over time. The area under the curve was calculated for glucose and insulin OGTT using the trapezium rule. All values were checked for normality using Kolmogorov-Smirnov and Shapiro-Wilk tests. Any values that were not normally distributed were subsequently transformed. On one occasion, values could not be normally transformed (white blood cells; WBC). In this case, original values were used based on the assumption that ANOVA is robust to violations of the normality assumption, in that even when data are non-normal, the error is usually close to the desired value [26].

## Results

### *Descriptive measures*

Anthropometric and physiological descriptive data for the lean and overweight subjects are summarised in Table 1. Lean and overweight subjects were a similar age and height, and had a similar absolute maximal oxygen uptake and maximum oxygen uptake per kg fat free mass; but differed for all characteristics affected by adiposity (Table 1).

### *Physical activity at baseline and during the intervention*

Pre-intervention and intervention physical activity energy expenditure (i.e., PAL) and step counts are shown in Figure 2. Importantly, the groups were similar at baseline with no significant difference in PAL, TEE or step counts ($P$ = 0.422, 0.147 and 0.477 respectively). TEE was 12953 ± 821 KJ/day for the lean group and 14485 ± 531 KJ/day for the overweight group pre-intervention and 11242 ± 696 KJ/day for the lean group and 11173 ± 1573 KJ/day for the overweight group post-intervention. During the intervention, there was a significant decrease in PAL ($P$ < 0.001), TEE ($P$ = 0.001) and step counts ($P$ < 0.001). Participants in both groups reduced their physical activity by similar amounts and there was no difference in any of these parameters between the two groups during the intervention week.

### *Markers of inflammation*

There was no change in IL-6, CRP, TNF-α, sICAM-1, adiponectin, ALT or WBC count following reduced physical activity for 7 days (ANOVA time effect, $P$ = 0.785, $P$ = 0.230, $P$ = 0.340, $P$ = 0.662, $P$ = 0.076, $P$ = 0.658, $P$ = 0.569, respectively; Table 2). However, CRP and ALT were significantly higher in the overweight group compared to the lean group throughout the intervention despite the two groups having similar high physical activity energy expenditure (ANOVA group effect, $P$ = 0.021 and $P$ = 0.017, respectively). There was no significant

252 difference in IL-6, TNF-α, sICAM-1, adiponectin or WBC count between the two groups
253 (ANOVA group effect, $P$ = 0.338, $P$ = 0.292, $P$ = 0.160, $P$ = 0.123, $P$ = 0.527, respectively;
254 Table 2). There was no difference in the manner in which the two groups responded to the
255 intervention for any of the inflammatory parameters (interaction effect; $P$ = 0.560, 0.147, 0.618,
256 0.727, 0.333 and 0.336 for IL-6, CRP, TNF-α, sICAM-1, adiponectin, ALT and WBC,
257 respectively).
258
259 ***Metabolic parameters***
260 Reduced physical activity increased the glucose and insulin area under the curve (AUC) and
261 fasted TAG concentrations following the reduced physical activity week in both groups
262 (ANOVA time effect $P$ = 0.018, $P$ = 0.001 and 0.021, respectively; Figure 3; Table 2, ANOVA
263 interaction effect; $P$ = 0.943, 0.252 and 0.372, respectively). There was no change in total, LDL
264 or HDL cholesterol over the intervention period ($P$ = 0.324, 0.323 and 0.075). The overweight
265 group had a significantly higher glucose and insulin AUC and TAG concentrations (ANOVA
266 group effect; $P$ = 0.046, 0.021 and 0.035, respectively) throughout the intervention compared to
267 the lean group and there was a trend for HDL cholesterol to be higher in the lean group ($P$ =
268 0.062). There was no significant difference in total and LDL cholesterol ($P$ = 0.461 and 0.442
269 respectively) between the two groups.
270
271

## Discussion

We hypothesized that overweight active middle-aged men would show a greater increase in markers of inflammation and other cardiovascular disease risk factors in response to one week of reduced activity than similarly-active lean middle-aged men. Whilst various measures of glucose control decreased and TAG concentrations increased over the week of reduced activity, this occurred to the same magnitude in both lean and overweight groups and there was no significant change in markers of inflammation. Thus, in the short-term, central overweight and lean middle-aged men respond similarly to a reduction in physical activity. Interestingly, in spite of similar (high) levels of physical activity, the overweight group had higher fasting markers of inflammation, TAG concentrations and a greater response to the OGTT (throughout).

Due to our rigorous inclusion criteria, we successfully recruited two groups with different body composition but similar physical activity. Free-living physical activity energy expenditure was assessed using one of the best techniques currently available [1, 21]. Free-living habitual physical activity level (PAL) for both groups was classified as ‘active’ according to physical activity guidelines from the Institute of Medicine [27]. This is equivalent to greater than 1000 kcal per day being expended through physical activity. Moreover, all subjects exceeded established physical activity guidelines of 30 minutes of activity at an intensity greater than 3 METs (moderate intensity) five times per week or more [28]; and, all subjects performed structured vigorous intensity exercise lasting at least 30 minutes three times a week. There was no difference in physical activity between the two groups in terms of physical activity energy expenditure (PAL) and step count. Importantly, the intervention was successful in moving PAL well below the ‘active’ category [27] and step count was reduced to a level considered sedentary [23].

As expected and in agreement with previous studies, we found a significant increase in insulin and glucose responses to an OGTT and fasting TAG concentrations over the week of reduced physical activity [8, 9]. In spite of these changes in metabolic markers, the one-week reduction in physical activity did not lead to a change in any marker of inflammation, in either group. In agreement with the current findings, previous research has found no change in several inflammatory markers (IL-6, CRP, TNF- α) in active, middle-aged men [5], and for IL-6 in active young men [4] following the removal of structured exercise for one week. In addition, Krogh-Madsen *et al* [9] found no increase in IL-6 or TNF-α after two weeks of reduced physical activity in young men using a similar model (i.e., limiting step counts). These prior findings and those from the present study indicate that these markers are relatively stable in the face of short-term changes in physical activity.

A key finding from the present study was that overweight middle-aged men had a more atherogenic profile than their lean counterparts for most of the risk factors we measured (CRP, ALT, TAG, glucose and insulin response to an OGTT) even though they expended considerable energy through participation in physical activity. Whilst epidemiological studies have previously suggested an association between body weight and markers of inflammation as well as other risk factors for cardiovascular disease (that is, independent of physical activity levels [11, 15]), these studies use less sensitive measures of physical activity (e.g. self-report questionnaires) and body fat (e.g. BMI) than those employed in the current study. The current findings extend these observations and improve the confidence with which we can conclude that adiposity (central and/or total) has a profound impact on health independent of precisely-measured physical activity. The effect of increased body fat on inflammation is likely to be attributed to the release of cytokines from adipose tissue [29]. Of course, from the present study design, we cannot determine whether high levels of physical activity ‘protected’ our overweight participants in comparison to a similarly overweight but sedentary population. Whilst we assume that this is the

case, the present results nevertheless indicate that high overall physical activity energy expenditure is not a direct substitute for leanness. With this in mind, a number of studies show that physical activity interventions only tend to reduce markers of inflammation when there is corresponding weight loss [30, 31]; although it is noteworthy that the reduction in fat mass by liposuction (i.e., without an energy deficit) does not lead to changes in markers of inflammation [32]. Clearly, whilst fat mass is independently important for various parameters, the effect may be secondary to adipose dysfunction (and function) rather than fat mass *per se* [33].

In addition to baseline differences in glucose control and TAG between groups (discussed above) we found that both glucose and insulin responses to an OGTT and fasting TAG increased following the one-week reduced physical activity intervention in both groups. Importantly, the overweight group had a similar response (change) in comparison to their lean counterparts for these parameters. This suggests an independent (of fat mass) benefit from recent physical activity. Even one day of increased sitting has a profound effect on insulin action [34]. Thus, whilst a lean phenotype is clearly beneficial for a number of outcomes, it does not prevent the negative metabolic changes that occur in response to reduced physical activity.

The current study design does not allow us to separate the effects of changes in energy balance during the intervention week from reduced physical activity *per se*. Assuming energy intake remained constant (participants were asked to maintain their normal diet), the reduced energy expenditure (due to reduced physical activity) would mean that subjects were in positive energy balance. Some studies have found the beneficial effects of exercise on metabolic factors have been attenuated when energy is replaced [35-37] and the short-term impact of increased sitting is also attenuated when energy intake is reduced [34]. Thus, energy balance may mediate some of the changes in TAG, glucose and insulin that occur with decreased physical activity. Whilst some studies indicate that energy restriction does not reduce postprandial TAG to the same magnitude

as energy-matched exercise the day before [38], other studies have shown that one day of energy restriction affects postprandial responses [39]. Thus, it is possible that in the current study that a more positive energy balance, rather than reduced physical activity *per se*, contributed to the poorer glucose control and increased TAG concentrations.

We asked subjects to perform a typical bout of exercise 32 to 48h before the pre-intervention blood sample before abstaining from structured exercise until the baseline trial day. This was to allow markers of inflammation to return to baseline following any increase caused by acute exercise [24]. It is very difficult to determine the optimal time to abstain from exercise in these kinds of experiments. Since many of the parameters that changed in the current study (TAG, glucose and insulin concentrations) have been shown to be affected by acute exercise [40, 41], we cannot rule out that the lower values at baseline were the effect of the last exercise bout that are manifested as changes in response to the reduced physical activity intervention. This would not undermine the relative importance (and independence) of physical activity; but it would perhaps shift the emphasis onto acute behaviour in terms of very recent exercise or physical activity. Furthermore, we should highlight that we recruited overweight men based on a central adiposity phenotype and we cannot therefore determine whether differences due to adiposity are specific to this population.

As described above, we recruited a slightly lower number of subjects than originally planned (9 in each group rather than 11). This was due to difficulty in recruiting individuals who were met rigid pre-defined criteria (i.e, being very active and waist circumference < 84 cm or > 94 cm). Most subjects were not eligible for the study even after advertising for these specific groups. Importantly, post-hoc power calculations suggest that if we had recruited 11 per group this would not have made a difference to any of the findings.

In summary, in contrast to our hypothesis, we found that one week of reduced physical activity induced similar changes in glucose control (response to OGTT) and TAG in active lean and active overweight middle-aged men, with no changes in various markers of inflammation. Importantly, overweight middle-aged men had higher values for many parameters when compared to similarly-active lean middle-aged men (i.e., CRP, ALT, TAG, glucose and insulin response to an OGTT). Thus, an experimental reduction in physical activity leads to important and similar changes in both lean and overweight middle-aged men; but, these results also suggest that similar levels of habitual physical activity cannot completely override the negative impact of central adiposity.

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## Figure Legends

**Figure 1**; Flow chart summarising subject recruitment and eligibility screening.

**Figure 2;** Physical activity level (PAL; a) and daily step count (b; mean ± SE) over the lifestyle monitoring week (normal) and the reduced activity week (intervention) * significant change over time ($p \leq 0.05$).

**Figure 3;** Whole blood glucose concentrations (a), 2 h AUC (b), serum insulin concentrations (c), and 2 h AUC (d; mean ± SE) in response to the OGTT before and after the reduced physical activity week in lean (n=9) and overweight similarly-active middle-aged men (n=9). Samples were taken every 15 minutes for the first hour and then 2 h following the administration of glucose in lean and overweight subjects before (Day 0) and after a reduced physical activity intervention (Day 7). * Significant change in AUC over time, ‡ 2 h AUC significantly different between lean and overweight similarly-active middle-aged men. ($P$ <0.05)

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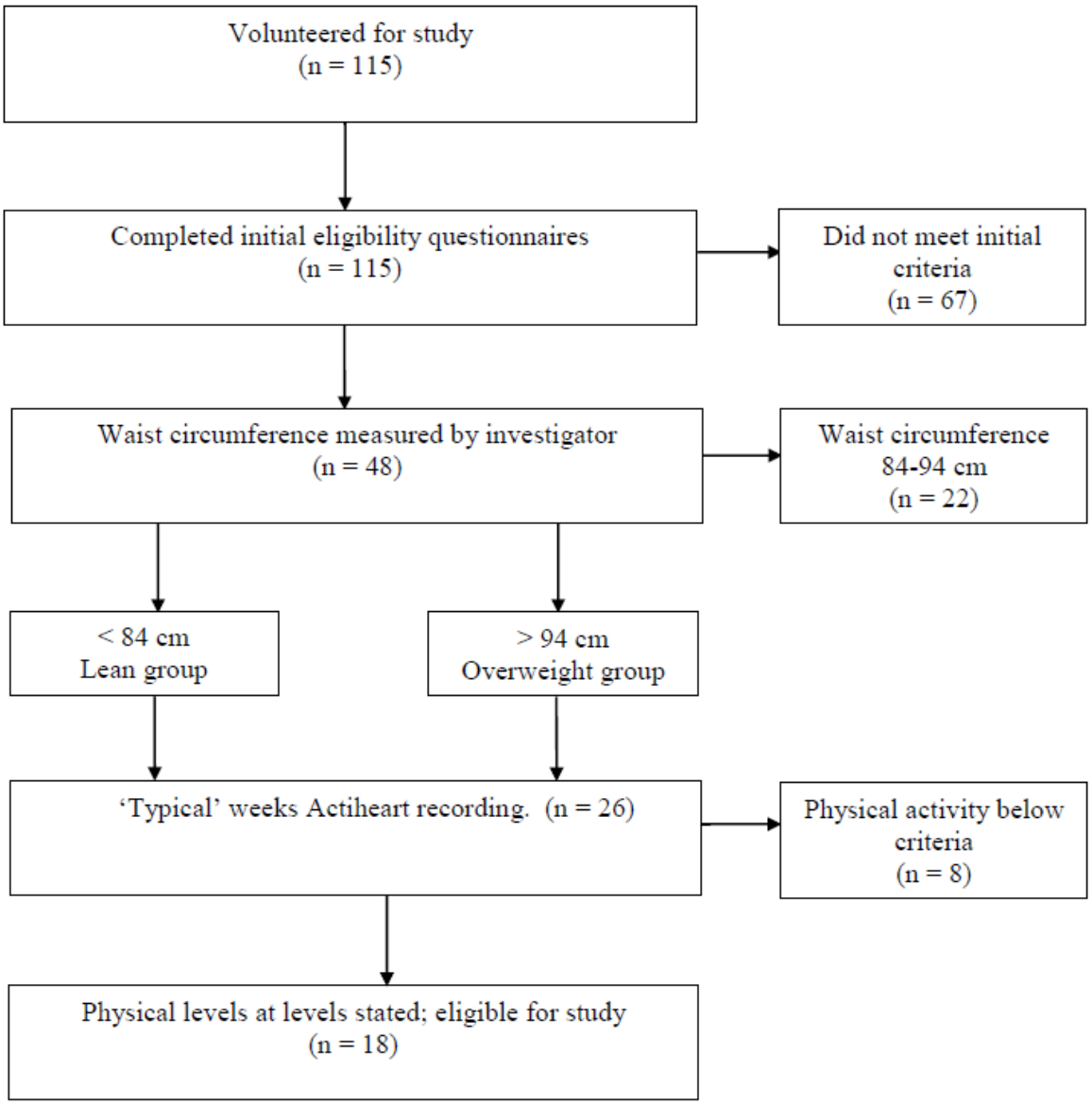


Figure 1

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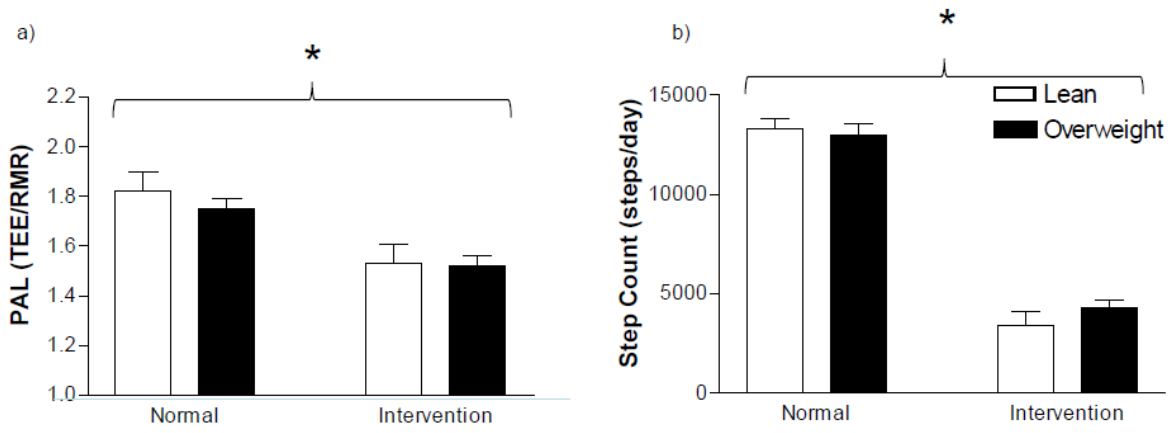
a)
*
PAL (TEE/RMR)
2.2
2.0
1.8
1.6
1.4
1.2
1.0
Normal
Intervention
b)
*
Step Count (steps/day)
15000
10000
5000
0
Lean
Overweight
Normal
Intervention

Figure 2

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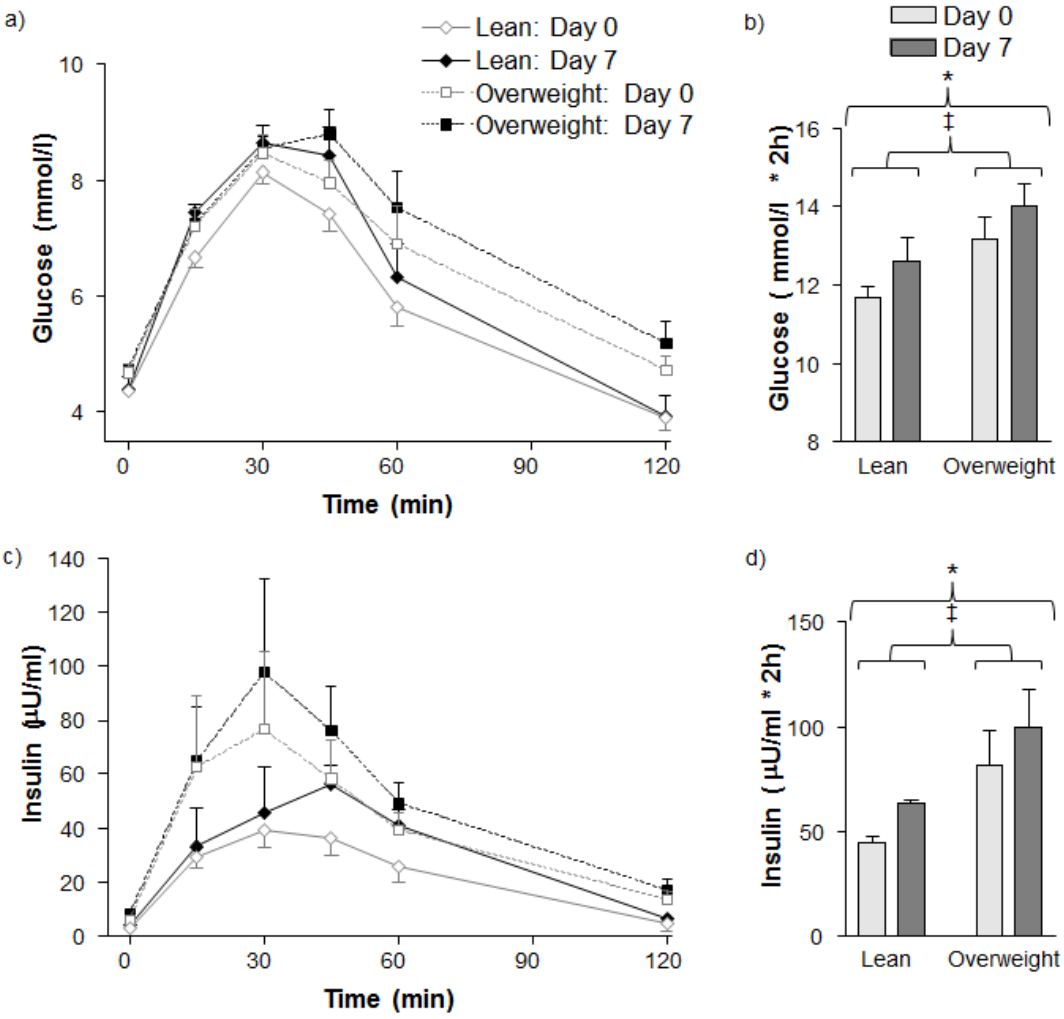


Figure 3

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**Table 1:** Baseline anthropometric and physiological measures for lean and overweight groups.

| | Lean (n = 9) | Overweight (n = 9) |
|---|---|---|
| **Age (yr)** | 51.5 ± 1.4 | 49.0 ± 1.0 |
| **Height (m)** | 1.80 ± 0.02 | 1.78 ± 0.02 |
| **Body Mass (kg) ‡** | 74.4 ± 2.4 | 93.0 ± 3.0 |
| **BMI ($kg/m^2$) ‡** | 23.8 ± 0.7 | 29.3 ± 1.2 |
| **Total Body Fat (kg) ‡** | 14.5 ± 1.0 | 23.1 ± 2.1 |
| **% Body Fat ‡** | 19.3 ± 1.7 | 24.9 ± 1.6 |
| **Abdominal Body Fat (kg) ‡** | 0.95 ± 0.13 | 2.10 ± 0.49 |
| **% Abdominal Fat ‡** | 22.5 ± 2.6 | 31.8 ± 2.3 |
| $\dot{V}O_2$ **max (ml/kg/min) ‡** | 50.5 ± 1.3 | 44.7 ± 2.5 |
| $\dot{V}O_2$ **max (l/min)** | 3.86 ± 0.18 | 4.15 ± 0.22 |
| **Systolic Blood Pressure (mmHg) ‡** | 122 ± 3 | 142 ± 8 |
| **Diastolic Blood Pressure (mmHg) ‡** | 82 ± 3 | 96 ± 4 |
| **Waist (cm) ‡** | 82.3 ± 0.5 | 99.2 ± 2.1 |

Data represent means ± SEM. ‡ significantly different between the two groups ($P \leq 0.05$).

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**Table 2**: Inflammatory and metabolic parameters at baseline and after 7 days reduced physical activity in lean and overweight groups.

| | Lean (n=9) | | Overweight (n=9) | |
|---|---|---|---|---|
| | **Day 0** | **Day 7** | **Day 0** | **Day 7** |
| **TNF-α (pg/ml)** | 3.19 ± 0.65 | 3.30 ± 0.42 | 2.46 ± 0.47 | 2.39 ± 0.46 |
| **sICAM (ng/ml)** | 168 ± 19 | 174 ± 15 | 196 ± 16 | 204 ± 20 |
| **Adiponectin (pg/ml)** | 11.0 ± 1.6 | 10.5 ± 1.5 | 8.4 ± 0.8 | 7.6 ± 0.7 |
| **White Blood Cells ($\times 10^6$/ml)** | 4.49 ± 0.32 | 5.20 ± 0.80 | 5.36 ± 0.63 | 5.30 ± 0.57 |
| **TAG (mmol/l)* ‡** | 0.95 ± 0.08 | 1.06 ± 0.09 | 1.33 ± 0.25 | 1.77 ± 0.24 |
| **FFA (mmol/l)‡** | 0.40 ± 0.05 | 0.21 ± 0.02 | 0.49 ± 0.10 | 0.36 ± 0.06 |
| **Cholesterol (mmol/l)** | 5.68 ± 0.26 | 5.77 ± 0.30 | 6.18 ± 0.39 | 6.05 ± 0.38 |
| **HDL Cholesterol (mmol/l)** | 1.58 ± 0.11 | 1.53 ± 0.09 | 1.41 ± 0.06 | 1.31 ± 0.06 |
| **LDL Cholesterol (mmol/l)** | 3.91 ± 0.20 | 4.03 ± 0.28 | 4.48 ± 0.39 | 4.39 ± 0.35 |

N.B., n=8 for inflammatory markers (TNF-α, sICAM-1, adiponectin, white blood cells) in the lean group. * significant change over time, ‡ significant different between groups ($P \leq 0.05$).

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