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Article Title: The post-exercise anti-inflammatory and glucose regulatory response is different between a sedentary Indigenous Australian and Caucasian cohort completing a single bout of cycle ergometry.

Running Title: Acute inflammatory and glucose response

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ABSTRACT

Objectives: This study compared the acute inflammatory and glucose responses following aerobic exercise in sedentary Indigenous Australian and Caucasian men, matched for fitness and body composition.

Methods: Sedentary Indigenous (n=10) and Caucasian (n=9) Australian men who were free from chronic disease volunteered to participate. Following baseline testing participants completed a 40min cycle ergometry bout at ~80% maximal heart rate. Fasting venous blood was collected pre, 0, 30, 60 min and 240 min post-exercise for analysis of glucose, insulin, cortisol, tumor necrosis factor (TNF)- α , interleukin (IL)-1 β , IL-6, IL-1 receptor agonist (ra) and C-reactive protein (CRP).

Results: Resting TNF- α and glucose concentrations were significantly higher in the Indigenous group ($P<0.05$). IL-6 and IL-1ra were elevated for longer in Caucasian ($P<0.05$), compared to the Indigenous group ($P>0.05$).

The post-exercise (0min) increase in cortisol and glucose for the Caucasians was higher ($P<0.05$) than the attenuated responses within the Indigenous group ($P>0.05$).

Conclusions: Despite being matched for fitness and body composition the Indigenous men had elevated resting TNF- α and glucose compared to the Caucasian men, which may have contributed to the suppressed post-exercise anti-inflammatory response of the Indigenous men; however, glucose normalized between groups post-exercise. As such, it is recommended for acute moderate-intensity exercise to be completed daily for long-term improvements in glucose regulation, irrespective of ancestry. Of note, results suggest it to be even more pertinent for exercise to be encouraged within the Indigenous Australian population due to their elevated resting glucose levels at a younger aged, when compared to the respective Caucasian group.

Key Words: Aboriginal, Ethnicity, Inflammation, Glucose, Cycling, Cytokine

Introduction

A comparison of physiological characteristics among different ancestral populations can provide insight into the disparity in health status between population groups. (Miller and Cappuccio, 2007; Thorburn and others, 1987). The worldwide incident rates of type 2 diabetes mellitus (T2DM) has been rising for the past 30 years with differences evident between population groups that are specific to ethnicity, geographical location and socioeconomic status (Albert, 2007; Cleland and Sattar, 2005; Naqshbandi and others, 2008). Many Indigenous groups experience a lower life expectancy consequent to increased prevalence of non-communicable chronic diseases (Albert, 2007; Cleland and Sattar, 2005; Welfare, Cat no: IHW 42, 2011). A prominent example of population-based differences is observed between Indigenous Australians and their Caucasian counterparts (Thorburn and others, 1987; Welfare, Cat no: IHW 42, 2011). As an illustration of this disparity, the age-standardised rate for Indigenous Australians with diabetes is estimated at 12 %, compared with 4 % for non-Indigenous Australians (Thomson and others, 2011; Trewin and Madden, 2005; Welfare, Cat no: IHW 42, 2011). Despite the higher prevalence of diabetes amongst Indigenous Australians, there is little published research reporting the acute physiological benefits of physical activity, which is known to be an essential component for reducing the potential disease risk in the long-term.

Physical inactivity is well known to influence inflammatory and glucose regulatory mechanisms that contribute to the development of T2DM and cardiovascular disease (CVD) (Kahn and others, 2006; Pradhan and others, 2001). An inverse relationship has been shown between physical activity, excess fat mass and systemic inflammatory biomarkers, which are predictors of all-cause mortality (Koenig and others, 2008). Therefore, regular physical activity may be an important factor in eliciting favourable acute inflammatory and glucose regulatory responses that positively impact on these parameters (Colberg, 2007; Rowley and others, 2000).

The clarification of differences in metabolic and inflammatory parameters enhanced by physical activity would be important in establishing whether the disparity in resting metabolic health between Indigenous and Caucasian Australians could be positively altered by an acute exercise bout. Therefore, the aim of this study was to compare the resting and exercise-induced inflammatory and glucose responses of an acute standardized bout of aerobic exercise in middle-aged, sedentary Indigenous Australian and Caucasian men, matched for body composition and aerobic fitness.

Materials and Methods

Participant Recruitment

Participants were recruited from a regional New South Wales community and were men whom self-identified to have Australian Indigenous (n=10) or Caucasian (n=9) ancestry. Participants were additionally matched for fitness and body composition (Table 1). Prior to data collection, verbal and written consent was provided followed by the completion of a pre-exercise health questionnaire and the Medical Outcomes Study Short-Form Health Survey (*MOS SF-36*). The life-expectancy of Indigenous and non-Indigenous Australian men is 67.2 y and 78.7 y, respectively (Welfare, Cat no: IHW 42, 2011). Thus, recruitment ensured participants whom represented their middle-aged cohort allowing for a ~11.5 y gap between the respective groups (Welfare, Cat no: IHW 42, 2011). Participants were sedentary (<1 exercise session per week for <60 min) but not clinically diagnosed with any pre-existing CVD or metabolic disorders. Exclusion criteria included: Smokers (< 1 y cessation); suffering from recurrent or recent influenza illness (including flu shot recipients); recent surgical patients; those on cholesterol lowering, anti-inflammatory, or any other medication reported to affect the inflammatory response; rheumatoid arthritis; known or recent periodontal disease. Any participant with a resting concentration $>10.0 \text{ mg}\cdot\text{L}^{-1}$ for CRP was excluded (Pearson and others, 2003). The study conformed to the Declaration of Helsinki and was approved by the Institutional Research in Human Ethics Committee.

Baseline Testing

Participants completed two sessions separated by a 7 d recovery period. The first session comprised of baseline testing, which also acted as an information and familiarisation session to explain all details of the study and testing procedures. Anthropometric data were obtained at baseline testing, including stature, body mass, and waist and hip girths using standard techniques (Norton and Olds, 1996). Manual blood pressure was obtained by aneroid sphygmomanometer and cuff (Welch-Alyn, Arden, North Carolina, USA) expressed as the mean of three measurements after the participant had been seated for 5 min. A supine whole body dual-energy x-ray absorptiometry (DXA) scan (XR800, Norland, Cooper Surgical Company, USA) was conducted with scanning resolution set at 6.5 x 13.0 mm, and scanning speed was set at 260 mm s⁻¹. Whole body scans were analysed (Illuminatus DXA, ver. 4.2.0, USA) for total body fat mass (TB-FM) and intra-abdominal fat mass (IA-FM) (Kim and others, 2002). Analysis of IA-FM was performed with the creation of a region of interest standardized across all participants according to previously outlined procedures (Hill and others, 2007).

Aerobic capacity measures were obtained via a graded exercise test (GXT) to determine sub-maximal oxygen consumption (VO_2) and power output (Watts). Prior to each test the metabolic gas analysis system (Parvo Medics, True2400, East Sandy, UT, USA) was calibrated and the GXT was performed on an electronically braked cycle ergometer (LODE Excalibur Sport, LODE BV, Groningen, The Netherlands) (Mendham et al. 2011). The test commenced at 25 W and increased by 25 W every min. Participants exercised until attainment of 80 % age-predicted ($220 - \text{age}$) maximum heart rate (HR_{max}) to minimise the risks associated with maximal physical capacity in potentially higher-risk participants. The last stage completed during the GXT test was converted from watts (W) into kilopond (kp) (Colton and Larsen, 2002) and used as the exercise intensity for a cadence of 60-65 rpm for the following cycle ergometry session.

Exercise Protocol

Following an overnight fast (10-12 h), participants arrived at the laboratory (between 0600 and 0800 h) and completed an acute bout of cycle ergometry. During all testing procedures the laboratory was at a controlled temperature of 18-20 °C. Participants refrained from any physical activity 48 h prior and alcohol and/or caffeine consumption 24 h prior to testing. During exercise and for 240 min after all testing sessions, participants remained fasted and consumed water *ab libitum* (~500 mL). The cycling session was conducted on a stationary cycle ergometer (Monark 828E, Varburg, Sweden) and comprised of 4 x 10 min efforts, at an intensity of 80 % HR_{max} , interspersed by 2 min passive recovery. Participants maintained a cadence of 60-65 rpm with baseline resistance set at the last completed workload of the GXT and (if required) manipulated to maintain the target intensity. Heart rate was monitored throughout (Vantage NV, Polar, Finland) and ratings of perceived exertion (RPE; Borg's 6-20 scale) were recorded as a session-RPE 30 min post-exercise (Herman et al. 2006). Of note, whilst the current study only involved the participants completing one exercise session, the methodology was designed to match an aerobic exercise prescription approach for a sedentary population (Garber and others, 2011).

Venous Blood Collection

Prior to the commencement of the protocol participants were cannulated for the collection of venous blood samples before and immediately (0 min), 30, 60 and 240 min after exercise (Ostrowski and others, 1999). The medial antecubital vein was cannulated and flushed with saline to ensure a clear line. Prior to collecting blood for analysis, saline was drawn from the line in another syringe and discarded. During cannulation and all blood

draws, participant postural position was standardised in the up-right position. Serum was collected in an SST tube for analysis of lipid profile, C-reactive protein (CRP), insulin and cortisol. Plasma was collected in an EDTA tube for the analysis of glycosylated haemoglobin (HbA1c), total leukocyte count, interleukin (IL)-6, IL-1 receptor agonist (ra), IL-1 β and tumor necrosis factor (TNF)- α ; as well as a fluoride oxalate (FO) tube for analysis of glucose. Following the clotting of the sample (SST) or immediately following collection (EDTA, FO) samples were centrifuged at 3500 rpm for 15 min at 4°C. Aliquots were frozen immediately at -80°C and -20°C for EDTA and SST, respectively. Whole blood was refrigerated (4°C) for a maximum of 6 h until analysis of total leukocyte count and HbA1c.

Venous Blood Analysis

Venous blood was also collected for descriptive purposes of the respective populations the following parameters were analysed (Maple-Brown and others, 2012). Fasting total cholesterol (Enzymatic method and polychromatic endpoint technique), high density lipoprotein (Accelerator selective detergent methodology), low density lipoprotein (Friedwald Equation), triglycerides (Enzymatic method and biochromatic endpoint technique) (Dimension Xpand Plus, Siemens Healthcare Diagnostics, Sydney, Australia), total leukocyte count (Cell counter: Cell-Dyn 3200, Abbott Laboratories, Abbott Park, IL, USA) and HbA1c (High-performance liquid chromatography: Bio-Rad Variant, Bio-Rad Laboratories, Sydney, Australia). For the analysis of glucose regulatory parameters (Pedersen and Febbraio, 2008; Steensberg and others, 2002), 20 mL was collected at each time point for analysis of glucose (ABL825 Flex Analyzer, Radiometer Medical ApS, Bronshoj, Denmark), insulin, cortisol (Solid-phase chemiluminescent enzyme immunometric assay: Immulite 2000, Siemens Healthcare Diagnostics, Los Angeles, CA, USA). Analysis of biochemistry variables glucose, insulin, cortisol and CRP showed intra and inter-assay coefficients of variation between 4.0-7.4 %. For the analysis of inflammatory parameters (Ostrowski and others, 1999; Pedersen and Febbraio, 2008), CRP (Particle enhanced turbidimetric immunoassay: Dimension Xpand Plus, Siemens Healthcare Diagnostics, Sydney, Australia), IL-6, IL-1 β , IL-1ra and TNF- α were measured at all-time points (Sandwich enzyme immunoassay ELISA: Quantikine, R & D Systems, Minneapolis, MN, USA), with intra and inter-assay coefficients of variation between 4.3-5.6 %. As an indication of insulin resistance, Homeostasis Model Assessment (HOMA-IR) was calculated based on (fasting insulin x fasting glucose)/22.5 (Matthews and others, 1985). Of note, hematocrit was not measured and none of the respective variables have been corrected for plasma volume change. This is a potential limitation in the current study, however, procedures were enforced within and between all testing

procedures to minimise any potential plasma volume shifts ie. controlled environmental temperature, posture collection and standardised fluid consumption prior to, during and following exercise. (Kargotich and others, 1998).

Statistical Analysis

All data are reported as mean \pm SEM. Within group analysis for all time-points were assessed by a two-way repeated measures ANOVA (condition \times time). Differences between-groups for all time-points were assessed by one-way analysis of variance (ANOVA) with a Tukey's correction. Significance was accepted at $P < 0.05$. All data that were not normally distributed were log transformed prior to analysis. All statistical analyses were performed using PASW™ for MS-Windows version 17.0 (Statistical Package for the Social Sciences, Chicago, IL, USA).

Results

Resting Venous Blood Chemistry, Body composition and Fitness Variables

All descriptive measures of anthropometry, DXA (TB-FM %, IA-FM kg), and resting venous blood chemistry are presented in Table 1. Both groups were matched for TB-FM % and VO_2 ($P > 0.05$); however, the Indigenous group was significantly younger and showed higher concentrations of fasting glucose and TNF- α ($P < 0.05$) compared to the Caucasians. Aerobic power assessed during baseline testing was reported as the last completed stage (W), which did not differ ($P = 0.52$) between Indigenous (183 ± 11 W) and Caucasian groups (172 ± 14 W). MOS SF-36 questionnaire results showed no significant difference between groups ($P = 0.67$) with a total of 79.2 ± 5 % for the Indigenous group and 83.9 ± 4 % for the Caucasian group.

Cycle Ergometry Demands

Resistance for the cycling protocol was not significantly different between groups ($P = 0.80$) at 1.9 ± 0.1 kp for Caucasian and 2.0 ± 0.3 kp for the Indigenous group. The mean HR response to the cycling protocol was not significantly different ($P = 0.17$) between groups at 81 ± 2 % for Indigenous and 83 ± 1 % HR_{max} for the Caucasian group. Session-RPE was not significantly different between groups (13.9 ± 0.5 and 13.6 ± 0.4 for Indigenous and Caucasian groups, respectively; $P = 0.54$).

Inflammatory Response to Cycle Ergometry

The acute post-exercise responses to cycle ergometry for IL-6, IL-1ra, CRP, TNF- α and IL-1 β are shown in Figure 1. IL-6 exhibited a significant post-exercise increase ($P < 0.05$) for both groups and was not different between groups ($P = 0.63$). However, from 30 to 60 min post-exercise IL-6 significantly decreased within the Indigenous group ($P = 0.004$), whilst the Caucasian group, remained elevated ($P = 0.27$). IL-1ra was significantly elevated post-exercise within both groups ($P < 0.05$), though the Caucasian group showed a sustained elevation of IL-1ra above pre values to 240 min post-exercise ($P = 0.03$) that was not evident in the Indigenous group ($P = 0.12$). Post-exercise CRP and IL-1 β responses did not differ within or between groups ($P > 0.05$). No exercise-induced response for TNF- α was evident within either group ($P > 0.05$), whilst between group comparisons indicated the Indigenous group to have significantly higher TNF- α values at all time-points compared to the Caucasian group ($P < 0.04$).

Cortisol, Glucose and Insulin Response to Cycle Ergometry

The acute post-exercise response for cortisol, glucose and insulin are shown in Figure 2. Cortisol showed a significant increase immediately post-exercise (0 min) within the Caucasian group ($P = 0.003$), but not in the Indigenous group ($P = 0.15$). Between group comparisons showed the Caucasian group to have significantly elevated cortisol values compared to the Indigenous group at 0 ($P = 0.008$), 30 ($P = 0.014$) and 60 min ($P = 0.002$) post-exercise. Glucose responses increased significantly immediately post-exercise within the Caucasian group ($P = 0.01$), but not the Indigenous group ($P = 0.57$). The exercise-induced insulin response showed a pre to 240 min post-exercise decrease within both groups ($P < 0.05$), without significant differences between groups ($P = 0.39$). Furthermore, HOMA-IS values from pre to 240 min post-exercise showed a significant decline within both the Indigenous (53.3 ± 7.3 %; $P = 0.007$) and Caucasian (42.6 ± 5.2 %; $P = 0.03$) groups, with no significant differences between groups ($P = 0.11$).

Discussion

The findings demonstrate that despite being matched for VO_2 and TB-FM %, Indigenous Australian and Caucasian groups varied in both baseline and post-exercise levels of some plasma inflammatory markers and glucose concentrations. Specifically, the younger Indigenous group had significantly higher fasting concentrations of glucose and TNF- α compared with the Caucasian group. Furthermore, although both groups showed an immediate post-exercise increase in anti-inflammatory markers (IL-6 and IL-1ra), the Caucasian

group sustained this elevated anti-inflammatory response longer than the Indigenous group. However, neither group showed any exercise-induced pro-inflammatory response (CRP, IL-1 β and TNF- α). Collectively, these findings suggest an acute suppression of anti-inflammatory cytokines to standardised aerobic exercise in sedentary Indigenous men.

A possible limitation in the present study is the recognised difference in chronological age between groups. However, when comparing chronological age between Indigenous and Caucasian populations, life expectancy is significantly different relative to their health status (Thomson and others, 2011). Therefore, matching of age does not provide a fair physiological comparison and can conflate the interpretation of the findings. For this reason the groups in the current study were matched for VO₂ and TB-FM %, as these have a more salient influence on inflammatory markers and glucose regulation, rather than chronological age *per se* (Arsenault and others, 2009; Church and others, 2002). The present results show that younger, Indigenous men have high fasting glucose concentrations that are subsequently reflected in altered acute post-exercise responses compared to an older Caucasian group. Of concern, the Indigenous group were ~10.2 y younger than the Caucasians group, suggesting that the metabolic health of Indigenous Australians declines at an earlier age than Caucasian Australians. These higher fasting glucose concentrations, if evident over prolonged periods, can lead to the development of T2DM and associated co-morbidities (Thomson and others, 2011; Trewin and Madden, 2005). Interestingly, on a national scale the Indigenous Australian population have a known susceptibility to developing T2DM and CVD when compared to their Caucasian counterparts (Thomson and others, 2011; Trewin and Madden, 2005; Welfare, Cat no: IHW 42, 2011). While data from the current study is from a small sample size, it reiterates these differences in health status between Indigenous and Caucasian Australians. Furthermore, these data also highlight the need for targeted exercise interventions for young Indigenous Australians as a disease preventative approach and also to determine the causative mechanisms that underlie the poorer metabolic health of this group.

Systemic inflammatory biomarkers are highly correlated with increased risk for chronic disease development and all-cause mortality (Koenig and others, 2008; Pradhan and others, 2001). Chronic elevation of TNF- α , IL-6 and IL-1 from adipose tissue stimulates the nuclear factor-kappa beta pathway and regulates the hepatic release of CRP; in turn highlighting a causative role in the development of T2DM and CVD (Kahn and others, 2006). The present study showed no difference between groups in resting concentrations of IL-6, IL-1 β or CRP.

However, in comparison to the Caucasian group, the Indigenous group had a significantly higher concentration of TNF- α and fasting glucose. Given the strong association of elevated TNF- α with impaired glucose control; higher TNF- α concentrations may provide reason for the observed baseline insulin resistance (>4 HOMA-IR) in the Indigenous group (Pedersen, 2011). Previous longitudinal studies have compared the health characteristics and effects of chronic exercise training between populations of differing ancestry (Miller and Cappuccio, 2007; Skinner and others, 2001). However, to date no evidence is available involving the acute physiological response to exercise for inflammatory and glucose markers between groups of differing ancestry.

Markers indicative of a pro-inflammatory state (TNF- α , IL-1 β , CRP) showed no significant exercise-induced responses; though, TNF- α remained elevated at all time-points in Indigenous compared to the Caucasian group. Moreover, in a rested state, increased IL-6 in the presence of elevated TNF- α is indicative of low-grade chronic systemic inflammation, whilst an exercise-induced increase of IL-6 in the absence of an elevated TNF- α is indicative of increased energy demand (Walsh and others, 2011). Though absent in the present study, an increase in TNF- α has been shown in response to strenuous prolonged exercise, with the magnitude potentially related to exercise duration and associated immunological parameters (Walsh and others, 2011). Notably, CRP is reported to peak 24-48 h post-exercise, and represents one potential limitation of the current data that reports up to 240 min post-exercise (Pedersen and Febbraio, 2008). Despite this limitation, the present study shows that regardless of ancestry, neither group to have exercise-induced increases in pro-inflammatory markers, which may be attributable to the short duration, moderate intensity and/or the interval nature of the condition.

Pro-inflammatory cytokines are suggested to be mediated by anti-inflammatory cytokines and cytokine inhibitors (e.g. cortisol), which can increase markedly in the circulation following prolonged exercise (Fischer, 2006; Pedersen, 2011). Additionally, during strenuous exercise IL-6 has anti-inflammatory properties mediating skeletal muscle signalling processes via the activated protein kinase pathway (AMPK) and stimulating the expression of other anti-inflammatory cytokines, such as IL-1ra and IL-10 (Pedersen, 2011; Walsh and others, 2011). Such increases of anti-inflammatory markers within the circulation may provide positive metabolic changes through increased fat oxidation and glucose uptake (Pedersen, 2006; Pedersen and Febbraio, 2008; Walsh and others, 2011). We observed that both groups increased anti-inflammatory cytokines IL-6 and IL-1ra immediately post-exercise. However, IL-6 remained elevated up to 60 min post-exercise and IL-1ra remained elevated up to 240 min post-exercise in the Caucasian group, but not the Indigenous group. This sustained

elevation of IL-6 and IL-1ra following exercise in the Caucasian group may partially explain the differences observed between groups in the pre to post-exercise glucose response. Similarly, since cortisol is a stimulatory hormone that contributes to increased hepatic glucose production (Kindermann and others, 1982), and IL-6 stimulates peripheral glucose metabolism (muscle and adipose tissue) (Pedersen, 2006), the combined blunted cortisol response and increased IL-6 may explain the decreasing post-exercise plasma glucose levels in the Indigenous group. In contrast, the Caucasian group showed a pre to post increase in cortisol and glucose concentration, which may explain the sustained elevation of IL-6 following exercise (Steensberg and others, 2001). Moreover, the higher pre-exercise glucose concentration in the Indigenous group may have accounted for the blunted cortisol response to the exercise condition. While speculative, the known metabolic contribution of cortisol (stimulate hepatic glucose production) during exercise could infer that the metabolic requirements to the same relative intensity was different between groups and thus had a substantial impact on the post-exercise anti-inflammatory response. Given the small sample size and lack of previous literature, it is speculative to suggest there are ancestry dependent mechanisms responsible for these differences. However, it is suggested that the higher resting TNF- α and glucose concentrations within the Indigenous group may have contributed to the difference in post-exercise cortisol and anti-inflammatory responses between groups.

In conclusion, despite being matched for VO₂ and TB-FM %, there were differences in resting TNF- α and glucose concentrations between an Indigenous Australian and Caucasian group. This disparity at baseline may have contributed to different post-exercise anti-inflammatory responses (IL-6 and IL-1ra) between groups. The similar post-exercise (0-240 min) glucose response between groups highlights the value of acute moderate-intensity exercise to be completed daily to assist with long-term improvements in glucose, irrespective of ancestry. Of note, the results of the current study suggest it to be even more pertinent for exercise to be encouraged within the Indigenous Australian population due to their elevated resting glucose levels at a younger age, when compared to the respective Caucasian group. Furthermore, exercise prescription within high-risk Indigenous populations should be designed to specifically focus on risk stratification, cultural sensitivity and thus ensure appropriate adaptations to exercise for chronic disease prevention. Whilst the outcomes of the current study are specific to Indigenous Australians and Caucasians, the findings highlight the need for additional research on exercise prescription involving acute differences within and between other Indigenous populations at high-risk of metabolic abnormalities.

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Conflict of Interest

None

Figure Legends

Fig. 1.

Mean \pm SEM response of IL-6, IL-1ra, CRP, TNF- α and IL-1 β within and between the respective groups.

^a Significant change within the Indigenous group (P<0.05); ^b Significant change within the Caucasian group (P<0.05); ^c Significant difference between groups (P<0.05); ^d Pre to 240 min change significantly different in both groups (P<0.05)

Fig. 2.

Mean \pm SEM response of cortisol, glucose and insulin within and between the respective groups.

^a Significant change within the Indigenous group (P<0.05); ^b Significant change within the Caucasian group (P<0.05); ^c Significant difference between groups (P<0.05); ^d Pre to 240 min change significantly different in both groups (P<0.05)

Table 1.

Mean \pm SEM Baseline characteristics, body composition and fasting blood chemistry of Indigenous and Caucasian participants.

Variable	Indigenous (n=10)	Caucasian (n=9)
Age (y) ^a	38.5 \pm 3.2	48.8 \pm 1.7
Height (cm)	177.7 \pm 3.1	177.4 \pm 1.8
Mass (kg)	101.5 \pm 7.9	101.9 \pm 3.3
BMI (kg·m ²)	31.9 \pm 2.0	32.3 \pm 0.9
Sub-maximal VO ₂ (mL·kg ⁻¹ ·min ⁻¹)	30.8 \pm 1.7	30.8 \pm 1.3
Systole blood pressure (mmHg)	131 \pm 3	131 \pm 4
Diastole blood pressure (mmHg)	84 \pm 2	82 \pm 1.9
Waist circumference (cm)	103.6 \pm 5.9	107.0 \pm 2.5
WHR	0.95 \pm 0.03	0.99 \pm 0.02
Total Body – Fat Mass (%)	27.8 \pm 3.6	31.4 \pm 1.6
Intra-Abdominal Fat Mass (kg)	3.56 \pm 0.7	3.57 \pm 0.29
Total cholesterol (mmol·L ⁻¹)	5.1 \pm 0.3	5.8 \pm 0.4
HDL (mmol·L ⁻¹)	1.1 \pm 0.1	1.3 \pm 0.1
Triglycerides (mmol·L ⁻¹)	1.6 \pm 0.2	2.2 \pm 0.4
Hazard ratio (Total : HDL)	4.8 \pm 1.5	4.6 \pm 0.4
HbA1c (%A1c)	5.7 \pm 0.2	5.7 \pm 0.1
Glucose (mmol·L ⁻¹) ^a	5.4 \pm 0.2	4.6 \pm 0.9
Insulin (μ U·mL ⁻¹)	17.7 \pm 4.6	11.5 \pm 2.9
Insulin resistance (HOMA-IS)	4.4 \pm 1.2	2.4 \pm 0.6
Total Leukocyte Count (10 ⁹ ·L ⁻¹)	6.9 \pm 0.6	6.1 \pm 0.4
Cortisol (nmol·L ⁻¹)	400.8 \pm 34.5	463.9 \pm 43.6
CRP (mg·L ⁻¹)	3.3 \pm 0.8	3.0 \pm 0.7
IL-6 (pg·mL ⁻¹)	1.0 \pm 0.2	1.4 \pm 0.3
TNF- α (pg·mL ⁻¹) ^a	2.6 \pm 0.4	1.2 \pm 0.2
IL-1ra (pg·mL ⁻¹)	252.6 \pm 27.9	235.2 \pm 24.3
IL-1 β (pg·mL ⁻¹)	1.42 \pm 0.11	1.6 \pm 0.1

BMI = Body mass index; VO₂ = Oxygen Consumption; WHR = Waist to hip ratio;

HDL = High density lipoprotein; CRP = C-reactive protein;

IL = interleukin; ra = receptor agonist; TNF = Tumor necrosis factor.

^a Significant difference between groups (P<0.05)

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