

# THE RELATIONSHIP BETWEEN ACTIVITY ENERGY EXPENDITURE, CARDIORESPIRATORY FITNESS, BODY COMPOSITION AND RISK FACTORS IN YOUNG, LEAN MEN AND WOMEN

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## Abstract

**Introduction:** Until now it has not been fully elucidated whether metabolic risk is most effectively reduced by increases in self-reported activity energy expenditure (AEE), total body movement, or objectively measured cardiorespiratory fitness ( $\dot{V}O_2\text{max}$ ). On the other hand, data concerning the association between AEE,  $\dot{V}O_2\text{max}$ , body composition and risk factors in young, lean individuals are incomplete.

**Aim of the study:** Our study aimed at the examination of the associations between AEE,  $\dot{V}O_2\text{max}$ , body composition and risk factors in college-aged lean men and women.

**Methods:** In 76 males with body fat  $\leq 20\%$  and 71 females with body fat  $\leq 30\%$  AEE was assessed using Seven-Day-Activity Recall (SDPAR),  $\dot{V}O_2\text{max}$  was determined during bicycle exercise until exhaustion. Subjects daily energy intake was briefly assessed from 24 h dietary recalls collected over 4 days preceding blood sampling. Blood was withdrawn from the antecubital vein under fasting conditions. Plasma glucose and lipoproteins levels were assayed colorimetrically. Circulating homocysteine was determined using FPIA method. Plasma insulin concentrations were determined using a standard radioimmunoassay.

**Results:** Exclusively in men AEE was significantly and inversely correlated with percent of body fat ( $r=-0.270$ ,  $P<0.001$ ) and markedly and positively correlated with  $\dot{V}O_2\text{max}$ . In women there was a tendency of higher LBM and lower circulating glucose, in men a tendency of lower plasma homocysteine levels with increasing AEE. In men  $\dot{V}O_2\text{max}$  was significantly and inversely correlated with body fat ( $r=-0.340$ ,  $P<0.003$ ), circulating total cholesterol ( $r=-.286$ ,  $P<0.02$ ) and LDL-cholesterol ( $r=-0.292$ ,  $P<0.02$ ). In women significant correlations was noted between  $\dot{V}O_2\text{max}$  and surrogate index of insulin resistance – HOMA-IR ( $r=-0.234$ ,  $P<0.05$ ). Multiple regression analysis has revealed that neither in men nor in women beneficial metabolic effects of  $\dot{V}O_2\text{max}$  were mediated by decreases in body fat stores with increasing cardiorespiratory fitness.

**Conclusions:** In young, lean men, but especially in women AEE effects on metabolic risk factors are minor. On the contrary,  $\dot{V}O_2\text{max}$  effects on metabolic risk factors seems to be sex-related, in men positively affecting circulating lipoproteins, in women decreasing index of insulin resistance-HOMA-IR. However, in both sexes  $\dot{V}O_2\text{max}$  exerts positive action irrespectively of the changes in body fat stores.

**Key words:** males, females, risk factors, physical activity

## Introduction

It is well documented that physical activity, irrespectively of age and sex, is associated with lower risk of metabolic disturbances and their adverse health consequences (1-3).

However, physical activity can be defined by several terms including time spent in activities, total body movement, activity energy expenditure (AEE) per kg of body mass, and physical activity level (PAL) defined as daily energy expenditure as a multiple of resting energy expenditure (4). It is worth noting that until now it is not fully elucidated whether metabolic risk is most effectively reduced by increases in self-reported activity energy expenditure (AEE), total body movement, or objectively measured cardiorespiratory fitness ( $\dot{V}O_2\text{max}$ ).

In children and adolescents  $\dot{V}O_2\text{max}$  was more strongly related to cardiovascular risk factors than physical activity expressed as total body movement, however, in adolescents aged 15-16 years the contribution of physical activity was greater than in children aged 9-10 years (5). In addition, in men younger than 65 years  $\dot{V}O_2\text{max}$  but not self-reported physical activity was a predictor of coronary events (6). However, in men over 65 years old both physical activity and  $\dot{V}O_2\text{max}$  were of similar importance. On the contrary, in middle-aged men and women the association of metabolic syndrome risk factors with physical activity measured using heart rate monitoring was three times greater than with  $\dot{V}O_2\text{max}$ , but  $\dot{V}O_2\text{max}$  modified the relationship between physical activity and metabolic syndrome (7). Furthermore, it was shown

that over a period of 5.6 years self-reported physical activity predicted the progression toward metabolic syndrome irrespectively of  $\dot{V}O_2\text{max}$  (8). The difference between metabolic effects of physical activity and  $\dot{V}O_2\text{max}$  was further supported by study which indicated that in men and women aged 26-36 years the associations between cardiometabolic risk, physical activity (self-reported and measured using pedometer) and  $\dot{V}O_2\text{max}$  were relatively independent, suggesting that a range of physical activity and fitness measures are needed to most accurately quantify associations between health and physical activity (9).

However, it should be stressed that the relationships between metabolic effects of physical activity and/or  $\dot{V}O_2\text{max}$  are even more complicated since there are data indicating no relationship or close associations between both variables (10,11).

Additionally, controversy exists concerning the relationship between beneficial effects of physical activity and/or  $\dot{V}O_2\text{max}$  and body fat stores. Li et al. (12) have demonstrated that self-reported physical activity and excessive fat stores independently contribute to the development of coronary heart disease in women aged 34-59 years. Moreover, Nassis et al. (13) have noted that in overweight and obese girls aged 13 years improved  $\dot{V}O_2\text{max}$  in response to 12 weeks aerobic training decreases metabolic risk without any changes in body fat. Similarly, Eklund et al. (14) have found that increased level of physical activity registered using a heart rate monitor reduces metabolic risk in the absence of improved body fatness in middle-aged men and women. On the contrary, Arsenault et al. (15) have found that in healthy men lower body fat and metabolic risk is observed in subjects with higher than with lower  $\dot{V}O_2\text{max}$ .

The reasons for above discrepancies were partially highlighted by Eklund et al (16) who have demonstrated that physical activity measured using accelerometer and  $\dot{V}O_2\text{max}$  are separately and independently associated with individual and clustered metabolic risk factors in children.  $\dot{V}O_2\text{max}$  effects on risk factors are mediated or confounded by adiposity, which does not mediate physical activity action. Additionally, Holt et al. (17) have found that in middle-aged men  $\dot{V}O_2\text{max}$  affects the whole body, liver and adipose tissue insulin sensitivity. On the contrary, physical activity action was limited to liver insulin sensitivity. Thus, it seems that AEE and  $\dot{V}O_2\text{max}$  actions are targeted on different metabolic processes.

Due to worldwide escalation of obesity most of above cited studies were performed in overweight and obese subjects or populations characterized by wide range of adiposity (from lean to obese) (18). Data concerning the association between AEE,  $\dot{V}O_2\text{max}$ , body composition and risk factors in young, lean individuals are scarce and incomplete. White et al. (19) have indicated a significant relationship between metabolic risk factors (lipoprotein profile, HOMA-IR, homocysteine,

CRP and IL-6) and intramuscular lipids in physically active men and women aged 21-36 years. However, in the cited study the effects of physical activity and cardiorespiratory fitness on tested variables was not evaluated. Sadhan et al. (20) have shown that in lean men and women aged 19-26 years the effects of cardiorespiratory fitness on body fat and blood pressure differ with respect to sex affecting body fat and systolic blood pressure in male and exclusively systolic blood pressure in female subjects. Unfortunately, in this study metabolic risk factors were not measured.

Taking all the above together, the purpose of our study was to examine the associations between self-reported physical activity,  $\dot{V}O_2\text{max}$ , body composition and risk factors (circulating plasma glucose, insulin, total homocysteine and lipoproteins as well as calculated index of insulin resistance -HOMA-IR) in college-aged lean men and women.

## Materials and methods

### Subjects

The participants were recruited on the basis of advertisements in student dormitories and by word of mouth. A total of 83 college-aged men and 80 women volunteered to participate in the study. All the subjects were healthy non-smokers not taking any medication on a regular basis and they gave their written consent prior to the participation in the study. Any subject was engaged in high-performance activity. In all volunteers (83 men and 80 women) body mass and height were measured with calibrated physicians' scale and BMI was calculated ( $\text{kg/m}^2$ ). Five men and five women were excluded because of  $\text{BMI} > 30$  (21). In 78 men and 75 women body fat was determined using the bioelectrical impedance method and BC 418 MA equipment (Tanita Co., Japan). Inter- and intra-assay coefficients of variation for body fat measurements did not exceed 5 %. For further procedure only lean subjects (76 men with body fat  $\leq 20\%$  and 71 women with body fat  $\leq 30\%$ ) were accepted (22). The study protocol was approved by the Ethics Commission in the University of Physical Education in Warsaw.

### Activity energy expenditure and cardiorespiratory fitness

In all subjects accepted for the study activity energy expenditure (AEE) was evaluated by an experienced interviewer using Seven-Day Physical Activity Recall (SDPAR) (23). This is a detailed quantitative questionnaire assessing the duration, frequency and intensity of the most common life style and structured leisure-time activity. Light, moderate, hard and very hard physical activity was define as MET 1.5, 4, 6, and 10 respectively and finally expressed in kcal/day and in kcal/day/ kg of body mass.

Cardiorespiratory fitness ( $\dot{V}O_2\text{max}$ ) was determined on an mechanically braked ergometer Ergomedic 874

E (Monark, Sweden). Oxygen uptake was measured with breath-by-breath using respiratory gas exchange analyzer (Sensor Medics 2900/2900c, USA). The gas analyzer was calibrated before each test with gases of known concentration. All subjects cycled for 4 min at 0 W. Thereafter the work rate increased by 15 W/min until volitional exhaustion and highest individual oxygen uptake was recognized as  $\dot{V}O_{2\max}$ . Participants were verbally encouraged to provide a maximal effort. The pedal rate was 60 rpm throughout the test (24).

### Dietary records

Daily energy intake was briefly assessed from 24 h food records collected over 4 days preceding blood collection and analyzed using computer program. A set of meals and different foods were shown to the participants by an experienced interviewer. The household measures of food intake were converted into gram weights. An interviewer assigned codes to the food reported by the subjects and performed computer analysis using the Food 2 program purchased from the Institute of Food and Nutrition in Warsaw.

### Calculation of basal metabolic rate (BMR)

BMR was calculated from lean and fat mass according to the formula:

$$[116,76 \times \text{LBM (kg)}] + [26,88 \times \text{fat mass (kg)}] \quad (25).$$

Daily energy intake to BMR ratio (EI/BMR) was calculated to validate the dietary records. EI/BMR values lower than 1.35 indicated underreporting of daily energy intakes.

### Blood sample collection and biochemical analyses

Blood was drawn between 8:00–8:30 a.m. after overnight fasting from the antecubital vein under aseptic conditions using disposable needles and syringes into plastic tubes containing lithium heparin. To separate plasma, blood samples were immediately centrifuged (15 min, 4000 rpm, 4° C) and stored at –70° C until analysis. Plasma glucose levels were determined using the oxidase method. Plasma triacylglycerols (TG), total cholesterol (TC), and HDL-cholesterol (HDL-C) concentrations were assayed colorimetrically. All analyses were performed using Randox commercial kits (Randox Laboratories, United Kingdom). Inter and intra-assay coefficients of variation for glucose, TG, TC and HDL-C determination did not exceed 5%. Plasma LDL-cholesterol levels were calculated according to Friedewald formula (26). Plasma total homocysteine (Hcy) concentrations were determined by fluorescence polarization immunoassay (FPIA) and Abbot commercial kits (USA). Inter- and intra-assay coefficients of variation for the Hcy assay did not exceed 5.2%. Insulin concentrations were measured by a standard radioimmunoassay method with mono-

clonal antibodies against insulin, using BioSource commercial kits (Belgium). Inter- and intra-assay coefficients of variation for insulin were 2.2% and 6.5%, respectively. All assays were run in duplicate. Insulin resistance index (HOMA-IR) was evaluated by homeostasis assessment model and calculated from fasting insulin and glucose concentrations [ $\text{insulin } (\mu\text{IU}\cdot\text{ml}^{-1}) \times \text{glucose (mmol}\cdot\text{L}^{-1})/22.5$ ] (27).

### Statistical analyses

Data distribution was evaluated using the Shapiro-Wilk test. The comparisons of variables in male and female subjects were performed using either unpaired Student t-test (body mass, body height, body fat, lean body mass,  $\dot{V}O_{2\max}$ , TC, HDL-C) or the Mann-Whitney test (age, physical activity, glucose, insulin, HOMA-IR, triacylglycerols, LDL-C, Hcy). Spearman rank correlations were calculated between variables which were not normally distributed. For normally distributed data correlations were calculated according to Pearson. To evaluate the contribution of body composition and cardiorespiratory fitness or activity energy expenditure to the variation in metabolic variables multiple regression analyses were performed. Data are presented as means  $\pm$  SD. Significance was set at  $P < 0.05$ . All calculations were performed using Statistica v.7.1 (Statsoft, USA).

### Results

Daily physical activity of female participants expressed in kcal/day or in kcal/day/kg was significantly lower than in their male counterparts (by 35.5% and by 8.1%, respectively,  $P < 0.001$ ) (Table 1). Daily energy intake in females was markedly lower than in males (by 37.3% and 19.3% for kcal/day and kcal/day/kg,

Table 1. *Subjects characteristics (means  $\pm$  SD)*

|                                | Men (n=76)                             | Women (n=71)                            |
|--------------------------------|----------------------------------------|-----------------------------------------|
| Age (years)                    | 19.8 $\pm$ 0.8                         | 20.3 $\pm$ 1.2                          |
| Body mass (kg)                 | 75.4 $\pm$ 8.9                         | 58.7 $\pm$ 5.7                          |
| Body height (cm)               | 180.6 $\pm$ 6.1                        | 167.2 $\pm$ 5.4                         |
| Fat (%)                        | 11.8 $\pm$ 4.5                         | 22.2 $\pm$ 4.2                          |
| Fat (kg)                       | 8.9 $\pm$ 3.2                          | 13.1 $\pm$ 3.2                          |
| LBM (kg)                       | 66.5 $\pm$ 6.5                         | 45.6 $\pm$ 4.0                          |
| AEE (kcal/day)                 | 792.7 $\pm$ 334.0                      | 511.9 $\pm$ 338.1*                      |
| AEE (kcal/day/kg)              | 10.5 $\pm$ 4.4                         | 8.6 $\pm$ 5.3*                          |
| $\dot{V}O_{2\max}$ (ml/kg/min) | 49.3 $\pm$ 6.1                         | 41.5 $\pm$ 4.4*                         |
| Energy intake (kcal/day)       | 2814 $\pm$ 695                         | 1767 $\pm$ 560*                         |
| Energy intake (kcal/day/kg)    | 37.3 $\pm$ 10.7                        | 30.1 $\pm$ 10.0*                        |
| BMR (kcal) <sup>a</sup>        | 1914 $\pm$ 188.8                       | 1723 $\pm$ 175.9*                       |
| Energy intake/BMR              | 1.53 $\pm$ 0.41<br>(18.4) <sup>b</sup> | 1.07 $\pm$ 0.34*<br>(47.8) <sup>b</sup> |

<sup>a</sup> calculated basal metabolic rate; <sup>b</sup> - percent of underreporters; \*  $P < 0.001$

respectively,  $P<0.001$ ). Similarly, calculated BMR value in women was markedly lower than in men (by 11%,  $P<0.001$ ). However, a much higher percent of females (47.8) than males (18.4) underreported their daily energy intake.

Plasma glucose concentrations in females were slightly (by 6.4%) but significantly ( $P<0.002$ ) lower than in males (Table 2). In contrast, plasma insulin levels in female subjects were markedly higher (by 23.5%,  $P<0.001$ ) than in their male counterparts. Surrogate index of insulin resistance (HOMA-IR) in female participants was significantly higher than in males (by 15.7%,  $P<0.005$ ). Plasma TG concentrations in women were significantly lower than in men (by 12.5 %,  $P<0.05$ ). No sex-related differences were found with respect to TC and LDL-C plasma levels. However, circulating HDL-C concentrations in women were slightly (by 7.1%,  $P<0.05$ ), but significantly higher than in men. Total plasma Hcy levels in men was markedly higher than in women (by 22.9%,  $P<0.001$ ).

Table 2. Biochemical variables in male and female participants (means  $\pm$  SD)

|                        | Men (n=76)        | Women (n=71)         |
|------------------------|-------------------|----------------------|
| Glucose (mmol/l)       | 4.7 $\pm$ 0.5     | 4.4 $\pm$ 0.4*       |
| Insulin ( $\mu$ IU/ml) | 6.8 $\pm$ 2.0     | 8.4 $\pm$ 2.9**      |
| HOMA-IR                | 1.420 $\pm$ 0.044 | 1.643 $\pm$ 0.051*** |
| TG (mmol/l)            | 0.8 $\pm$ 0.3     | 0.7 $\pm$ 0.3****    |
| TC (mmol/l)            | 4.5 $\pm$ 0.7     | 4.5 $\pm$ 0.7        |
| HDL-C (mmol/l)         | 1.4 $\pm$ 0.4     | 1.5 $\pm$ 0.3*****   |
| LDL-C (mmol/l)         | 2.6 $\pm$ 0.7     | 2.6 $\pm$ 0.4        |
| Hcy ( $\mu$ mol/l)     | 10.2 $\pm$ 3.0    | 8.3 $\pm$ 2.4**      |

\* $P<0.002$ ; \*\* $P<0.001$ ; \*\*\* $P<0.005$ ; \*\*\*\* $P<0.05$ ; \*\*\*\*\* $P<0.03$ —significantly different vs. men

Table 3. Correlation coefficients between AEE (kcal/day/kg), and  $\dot{V}O_2$ max, body composition and biochemical variables in male and female subjects

|                              | Men (n=76)       | Women (n=71)      |
|------------------------------|------------------|-------------------|
| $\dot{V}O_2$ max (ml/kg/min) | <b>0.385*</b>    | 0.168             |
| Body fat (%)                 | <b>-0.270**</b>  | 0.070             |
| LBM (kg)                     | 0.070            | <b>0.222***</b>   |
| Glucose (mmol/l)             | -0.048           | <b>-0.226****</b> |
| Insulin ( $\mu$ IU/ml)       | -0.070           | 0.070             |
| HOMA-IR                      | -0.105           | 0.018             |
| TG (mmol/l)                  | 0.040            | 0.017             |
| TC (mmol/l)                  | -0.024           | -0.137            |
| HDL-C (mmol/l)               | -0.127           | -0.020            |
| LDL-C (mmol/l)               | 0.135            | -0.138            |
| Hcy ( $\mu$ mol/l)           | <b>-0.212***</b> | -0.144            |

\* $P<0.001$ ; \*\* $P<0.02$ ; \*\*\* $P<0.07$ ; \*\*\*\* $P<0.06$

Table 4. Correlations coefficients between  $\dot{V}O_2$ max, (ml/kg/min), body composition and biochemical variables in male and female subjects

|                        | Men (n=76)      | Women (n=71)      |
|------------------------|-----------------|-------------------|
| Body fat (%)           | <b>-0.340*</b>  | <b>-0.216***</b>  |
| LBM (kg)               | -0.050          | 0.198             |
| Glucose (mmol/l)       | -0.159          | -0.059            |
| Insulin ( $\mu$ IU/ml) | -0.063          | <b>-0.216***</b>  |
| HOMA-IR                | -0.118          | <b>-0.234****</b> |
| TG (mmol/l)            | 0.094           | 0.044             |
| TC (mmol/l)            | <b>-0.286**</b> | -0.174            |
| HDL-C (mmol/l)         | 0.050           | -0.209*****       |
| LDL-C (mmol/l)         | <b>-0.292**</b> | -0.044            |
| Hcy ( $\mu$ mol/l)     | 0.040           | -0.090            |

\* $P<0.003$ ; \*\* $P<0.02$ ; \*\*\* $P<0.08$ ; \*\*\*\* $P<0.05$ ; \*\*\*\*\* $P<0.09$

Table 5. The best models predicting circulating TC and LDL-C in young, lean men (A) and HOMA-IR in young lean women (B)

|                     |                       |                                   |                                  |          |
|---------------------|-----------------------|-----------------------------------|----------------------------------|----------|
| A.                  |                       |                                   |                                  |          |
| *Dependent variable | Independent variables | Multiple correlation coefficients | Partial correlation coefficients | <i>P</i> |
| TC                  | Body fat (%)          | 0.331<br>( <i>P</i> <0.0096)      | -0.046                           | 0.0678   |
|                     | ṀO <sub>2</sub> max   |                                   | -0.325                           | 0.0028   |
| LDL-C               | Body fat (%)          | 0.344<br>( <i>P</i> <0.0071)      | 0.003                            | 0.015    |
|                     | ṀO <sub>2</sub> max   |                                   | -0.324                           | 0.0030   |
| B.                  |                       |                                   |                                  |          |
| *Dependent variable | Independent variables | Multiple correlation coefficients | Partial correlation coefficients | <i>P</i> |
| HOMA-IR             | Body fat (%)          | 0.300<br>( <i>P</i> <0.033)       | 0.109                            | 0.355    |
|                     | ṀO <sub>2</sub> max   |                                   | -0.249                           | 0.032    |



Exclusively in men AEE expressed in kcal/day/kg was significantly and positively correlated with  $\dot{V}O_{2\max}$  ( $P<0.001$ ) and significantly and inversely correlated with percent of body fat ( $P<0.02$ ) (Table 3). There was a tendency of lower total plasma Hcy levels with increasing AEE in male ( $P<0.07$ ) but not in female participants. On the contrary, in women there were no apparent effects of AEE on any tested variables, however, with increasing AEE there was a tendency to higher lean body mass ( $P<0.07$ ) and lower plasma glucose levels ( $P<0.06$ ). Neither in men nor in women was AEE correlated with circulating glucose, insulin, lipoproteins and HOMA-IR.

In men a significant and inverse correlation was noted between  $\dot{V}O_{2\max}$ , percentage of body fat ( $P<0.003$ ), TC ( $P<0.02$ ) and LDL-C ( $P<0.02$ ) (Table 4). In women the tendency of lower body fat and lower circulating insulin ( $P<0.08$ ) with increasing  $\dot{V}O_{2\max}$  was observed. Exclusively in women  $\dot{V}O_{2\max}$  was significantly and inversely correlated with HOMA-IR ( $P<0.05$ ). In female participants plasma lipoprotein levels were not affected by  $\dot{V}O_{2\max}$ , however the tendency ( $P<0.09$ ) of lower plasma HDL-C concentrations with increasing  $\dot{V}O_{2\max}$  was observed.

Multiple regression analyses revealed that both in men and women body fat did not contribute to the effects of  $\dot{V}O_{2\max}$  on metabolic variables. In men cardiorespiratory fitness had a beneficial effect on circulating TC and LDL-C with no effect of body fat stores (Table 5A). Similarly in women HOMA-IR was positively affected by cardiorespiratory fitness with non-significant contribution of the percent of body fat (Table 5B).

## Discussion

In participants of our study body fat stores was lower and lean body mass was higher than that reported by Pichard et al. (28) and Kyle et al. (29) in healthy young subjects of similar age. However, the mean AEE of our participants was higher than recommended for one's health and  $\dot{V}O_{2\max}$  were characteristic for active subjects, both contributing to decreased body fat and increased lean body mass in comparison with the general population of a similar age (30). The mean daily energy intake of both men and women was similar to that found in our previous study (31). Significantly higher plasma insulin and HDL-C levels as well as HOMA-IR, but lower circulating TG in females than in males were found by others in healthy subjects (32,33). In addition, higher plasma Hcy levels in men than in women has been noted in other studies (34,35).

There are two important findings in the present study. First of all the effects of AEE or  $\dot{V}O_{2\max}$  on body composition and metabolic parameters in young, lean subjects are markedly different and this is in agreement with others data (16,17). Secondly, the response of

body composition and metabolic variables either to AEE or  $\dot{V}O_{2\max}$  seems to be sex-related.

Our data concerning significant correlations of AEE with body fat in men and no effect of AEE on body fat in women corroborate previous results of Westerterp et al. (36) and may be due to more efficient fat protection in female than in male subjects (37).

Interestingly, AEE effects on metabolic variables both in men and women was minor. This may be due to relatively small number of participants affecting accuracy and precision of AEE measurement, since several population-based studies have indicated beneficial effects of AEE on metabolic variables (38). However, it should be pointed out that a tendency of lower plasma Hcy levels in men and glucose concentrations in women with increasing AEE is beneficial for health (39,40). Moreover, slight but positive effect of AEE on LBM in women possibly has more positive effects than that tested in our study due to significant relationship between LBM, basal metabolic rate, insulin sensitivity and fat metabolism (41-43).

Additionally it is worth noting that exclusively in male subjects of our study AEE was significantly and positively correlated with  $\dot{V}O_{2\max}$ . The reason for discordant results in men and women is unclear, however data in the literature has indicated that females more frequently overreport their physical activity and underreport daily energy intake (44,45). Taking into account that almost 48% of our female subjects underreported their energy intake, it seems feasible that they overreported their daily physical activity and in consequence no correlations between objectively measured  $\dot{V}O_{2\max}$  and AEE has been found.

The effect of  $\dot{V}O_{2\max}$  on metabolic variables differs with respect to sex. Our data indicated that in men increasing  $\dot{V}O_{2\max}$  resulted in lower plasma TC and LDL-C concentrations possibly due to lower body fat stores significantly and inversely correlated with  $\dot{V}O_{2\max}$ . This assumption seems feasible since it is well documented that free fatty acids released from adipose tissue contribute to liver lipoprotein synthesis and prevalence of upper body fat in men make them more susceptible to disturbances in lipoprotein profile (46,47). However, multiple regression analyses have revealed that in lean subjects of the current study  $\dot{V}O_{2\max}$  effect on lipoprotein profile is not mediated by its effect on body fat stores.

In females participants metabolic response to  $\dot{V}O_{2\max}$  is more complicated. Significant decrease in HOMA-IR with increasing fitness is undoubtedly beneficial for health especially in females who are probably genetically predisposed to lower insulin sensitivity than males (48). However, the tendency of lower HDL-C level with increasing fitness indicates adverse possible health consequences of strenuous physical activity in women due to disturbed sex hor-

mone secretion (49). This assumption seems feasible since there were no associations between circulating HDL-C and dietary habits of our female participants (Lutosławska et al., unpublished). On the other hand, taking into account a close relationship between insulin sensitivity and lipoprotein metabolism (50) and marked improvement of HOMA-IR in response to increasing fitness it could not be excluded that slight adverse effect of fitness on HDL-C in lean females is of minor importance.

It is worth noting that in lean women of our study positive effect of cardiorespiratory fitness on HOMA-IR was not mediated by its effect on body fat stores.

The comparison of the effects of  $\dot{V}O_2\text{max}$  on metabolic risk factors in lean men and women probably indicates that they are differently targeted with respect to sex with more pronounced action on lipoprotein metabolism in men and markedly greater influence on insulin sensitivity in women.

Summing up, in young lean men, but especially in lean women activity energy expenditure (AEE) obtained from self-reported questionnaires is poorly correlated with metabolic variables and body composition. More information is provided by precisely measured  $\dot{V}O_2\text{max}$ , however its effects on metabolic variables and body composition at least in lean subjects, are sex-related and not mediated by its influence on body fat stores.

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## References

- Mora S, Cook N, Buring JE, et al. Physical activity and reduced risk of cardiovascular events. *Circulation* 2007; 116: 2110-8.
- Murphy MH, Nevill AM, Murtagh EM, et al. The effect of walking on fitness, fatness and resting blood pressure: A meta-analysis of randomized, controlled studies. *Prev Med* 2007; 44: 377-85. (doi: 10.1016/j.ypmed.2006.12.008)
- Krekoukia M, Nassi GP, Psarra G, et al. Elevated total and central obesity and low physical activity are associated with insulin resistance in children. *Metabolism* 2007; 56: 206-13. (doi: 10.1016/j.metabol.2006.09.014)
- Goris AHC, Westerterp KA. Physical activity, fat intake and body fat. *Physiol. Behavior* 2008; 94: 164-8. (doi: 10.1016/j.physbeh.2007.11009)
- Hurtig-Wennlöf A, Ruiz JR, Harro M, et al. Cardiorespiratory fitness relates more strongly than physical activity to cardiovascular disease risk factors in healthy children and adolescent: the European Youth Heart Study. *Eur J Cardiovasc Prev Rehab* 2007; 14: 575-81.
- Talbot LA, Morell CH, Metter EJ, et al. Comparison of cardiorespiratory fitness versus leisure time physical activity as predictors of coronary events in men aged < or + 65 years. *Am J Cardiol* 2002; 89: 1187-92.
- Franks PW, Eklund U, Brage S, et al. Does the association of habitual physical activity with the metabolic syndrome differ by the level of cardiorespiratory fitness? *Diabetes Care* 2004; 27: 1187-93.
- Eklund U, Brage S, Franks PW, et al. Physical activity energy expenditure predicts progression towards the metabolic syndrome independently of aerobic fitness in middle-aged healthy Caucasians. *Diabetes Care* 2005; 28: 1195-200.
- Schmidt MD, Cleland VJ, Thompson RJ, et al. A comparison of subjective and objective measures of physical activity and fitness in identifying associations with cardiometabolic risk factors. *Ann Epidemiol* 2008; 18: 378-86. (doi: 10.1016/j.annepidem.2008.01.005)
- Simmons RK, Griffin SJ, Steele R, et al. Increased overall physical activity and aerobic fitness is associated with improvement in metabolic risk: cohort analysis of the ProActive trial. *Diabetologia* 2008; 51: 787-94.
- Church TS, Earnets CP, Skinner JS, et al. Effects of different doses of physical activity on cardiorespiratory fitness among sedentary overweight or obese postmenopausal women with elevated blood pressure. *JAMA* 2007; 297: 2081-91.
- Li TY, Rana JS, Manson JE, et al. Obesity as compared with physical activity in predicting risk of coronary heart disease in women. *Circulation* 2006; 113: 499-506. (doi: 10.1161/CirculationAHA.105574087)
- Nassis GP, Papantakou K, Skenderi K, et al. Aerobic exercise training improves insulin sensitivity without changes in body weight, body fat, adiponectin, and inflammatory markers in overweight and obese girls. *Metabolism* 2005; 54: 1472-9. (doi:10.1016/j.metabol.2005.05.013)
- Eklund U, Franks PW, Sharp S, et al. Increase in physical activity energy expenditure is associated with reduced metabolic risk independent of change in fatness and fitness. *Diabetes Care* 2007; 30: 2101-6.
- Arsenault RJ, Lachance D, Lemieux I, et al. Visceral adipose tissue accumulation, cardiorespiratory fitness, and features of the metabolic syndrome. *Arch Intern Med* 2007; 167: 1518-25.
- Eklund U, Anderssen SA, Forberg K, et al. Independent associations of physical activity and cardiorespiratory fitness with metabolic risk factors in children: the European youth heart study. *Diabetologia* 2007; 50: 1832-40.
- Holt HB, Wild SH, Wareham N, et al. Differential effects of fatness, fitness and physical activity energy expenditure on whole-body, liver and fat insulin sensitivity. *Diabetologia* 2007; 50: 1698-1706.
- Wareham N. Physical activity and obesity prevention. *Obes Rev* 2007; 8: (Suppl 1) 109-14.
- White LJ, Ferguson MA, McCoy SC, et al. Cardiovascular/non-insulin-dependent diabetes mellitus risk factors and intramyocellular lipid in healthy subjects: a sex comparison. *Metabolism* 2006; 55: 128-34. (doi:10.1016/j.metabol.2005.08.026)
- Sadhan B, Koley S, Sandhu JS. Relationship between cardiorespiratory fitness, body composition and blood pressure in Punjabi collegiate populations. *J Human Ecol* 2007; 22: 215-9.
- Stolic M, Russel A, Hutley L, et al. Glucose uptake and insulin action in human adipose tissue-influence of BMI, anatomical depot and body fat distribution. *Int J Obes* 2002; 26: 17-23.
- Abernathy RP, Black DR. Healthy body weights: an alternative perspective. *Am J Clin Nutr* 1966; (3 Suppl): 448S-451S.
- Sallis JF. Seven-Days physical activity recall. *Med Sci Sports Exerc* 1997; 27 (Suppl): S89-S103.
- Storer TW, Davis JA, Caizzo VJ. Accurate prediction of  $\dot{V}O_2\text{max}$  in cycle ergometry. *Med Sci Sports Exerc* 1990; 22: 704-12.
- Graby L, Garrow JS, Jorgensen B, et al. Relation between energy expenditure and body composition in man: specific energy expenditure in vivo of fat and fat-free mass. *Eur J Clin Nutr* 1988; 42: 301-5.
- Friedewald WT, Levy R, Fredrikson D. Estimation of concentration of low density lipoprotein without use of the preparative ultracentrifugation. *Clin Chem* 1972; 18: 449-504.
- Matthews DR, Hosker JP, Rudenski AS, et al. Homeostasis model assessment: insulin resistance and  $\beta$ -cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985;28:412-9.

28. Pichard C, Kyle U, Bracco D, et al. Reference values of fat-free and fat masses by bioelectrical impedance analysis in 3393 healthy subjects. *Nutrition* 2000; 16: 245-54.
29. Kyle U, Genton L, Slosman DO, et al. Fat-free and fat mass percentiles in 5225 healthy subjects aged 15-98 years. *Nutrition* 2001; 17: 534-41.
30. Thompson JL. Exercise in improving health v. performance. *Proc Nutr Soc* 2009; 68: 29-33. (doi:10.1017/S0029665108008811)
31. Lutosławska G, Malara M, Mazurek K, et al. Daily intake of micronutrients and selected minerals in physically active female students in comparison with males of matched age and physical activity. *Med Sport* 2007; 11: 119-23. (doi: 10.2478/v10036-009-0006-5)
32. Rubin DA, McMurray RG, Harrell JS, et al. The association between insulin resistance and cytokines in adolescent: the role of weight status and exercise. *Metabolism* 2008; 57: 683-90. (doi:10.1016/j.metabol.2008.01.005)
33. Imperatore G, Cheng YJ, Williams DE, et al. Physical activity, cardiorespiratory fitness, and insulin sensitivity among U.S. adolescents. *Diabetes Care* 2006; 29: 1567-72.
34. Okura T, Rankinen T, Gagnon J, et al. Effect of regular exercise on homocysteine concentrations: the Heritage Family Study. *Eur J Appl Physiol* 2006; 98: 394-401. (doi:10.1007/s00421-006-0294-6)
35. Panagiotakos DB, Pitsavos Ch, Zeimbekis A, et al. The association between lifestyle-related factors and plasma homocysteine level in healthy individuals from the "AT-TICA" study. *Int J Cardiol* 2005; 98: 471-7. (doi: 10.1016/ij-card.2003.12.036)
36. Westerterp KR, Goran MI. Relationship between physical activity related energy expenditure and body composition: a gender difference. *Int J Obes* 1997; 21: 184-8.
37. Westertrep KR. Alterations in energy balance with exercise. *Am J Clin Nutr* 1998; 68(suppl): 970S-4S.
38. Shephard RJ, Balady GJ. Exercise as cardiovascular therapy. *Circulation* 1999; 99: 963-72.
39. Hayward R, Ruangthain R, Karnilaw P, et al. Attenuation of homocysteine-induced endothelial dysfunction by exercise training. *Pathophysiology* 2003; 9: 207-14.
40. Godsland IF, Jeffs JA, Johnston DG. Loss of beta cell function as fasting glucose increases in the non-diabetic range. *Diabetologia* 2004; 47: 1157-66. (doi: 10.1007/s00125-004-1454-z)
41. Alpert SS. The cross-sectional and longitudinal dependence of the resting metabolic rate on the fat-free mass. *Metabolism* 2007; 56: 363-72. (doi: 10.1016/j.metabol.2006.10.018)
42. Cortright RN, Sandhoff KM, Basilio JL, et al. Skeletal muscle fat oxidation is increased in African-American and white women after 10 days of endurance exercise training. *Obesity* 2006; 14: 1201-7.
43. McGee SL, Hargreaves M. Exercise and skeletal muscle glucose transporter 4 expression: molecular mechanism. *Clin Exp Pharmacol Physiol* 2006; 33: 395-9.
44. Lichtamn SW, Pisarska K, Berman ER, et al. Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. *N Eng J Med* 1992; 327: 1893-8.
45. Okubo H, Sasaki S. Underreporting of energy intake among Japanese women aged 18-20 years and its association with reported nutrient and food group intakes. *Public Health Nutr* 2004; 7: 911-7. (doi: 10.1079/PHN2004635)
46. Arner P. Human cell lipolysis: biochemistry, regulation and clinical role. *Best Pract Res Endocrinol Metab* 2005; 19: 471-82.
47. Walton Ch, Lees B, Crook D, et al. Body fat distribution, rather than overall adiposity, influences serum lipids and lipoproteins in healthy men independently of age. *Am J Med* 1995; 99:459-64.
48. Wilkin TJ, Murphy MJ. The gender insulin hypothesis: why girls are born lighter than boys, and the implications for insulin resistance. *Int J Obes* 2006;30: 1056-61.
49. Rickelund A, Eriksson MJ, Schenck-Gustafsson K, et al. Amenorrhea in female athletes is associated with endothelial dysfunction and unfavorable lipid profile. *J Clin Endocrinol Metab* 2005; 90: 1354-9.
50. Gill JMR, Malkova D. Physical activity, fitness and cardiovascular disease risk in adults: interactions with insulin resistance and obesity. *Clin Sci* 2006; 110: 409-25. (doi:10.042/CS20050207)

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