

Influence of exercise and body composition on fasting ghrelin, glucose, insulin and HOMA-IR in men

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Ghrelin, glucose and insulin can be influenced by exercise and body composition, and have a role in appetite control. Although, it is well established that a single exercise bout improves glucose metabolism acutely, the effects appear to diminish within 48 to 72 h⁽¹⁾. The aim of this investigation was to assess the effects of (1) habitual exercise, and (2) a short-term exercise intervention on fasting ghrelin, glucose, insulin and HOMA-IR in men, and to explore associations with body fat and energy expenditure.

In the first cross-sectional study, forty-four men (Active: $n = 22$, Inactive: $n = 22$; range BMI 21–36 kg/m²; range percent fat mass 9–42 %) were studied. For the second study, a subset of fifteen inactive overweight men subsequently completed a 4-wk supervised exercise intervention, consisting of 5 exercise sessions per week on a cycle ergometer. Body composition was assessed by air displacement plethysmography, activity energy expenditure (AEE) by accelerometry and fasting levels of glucose, insulin and ghrelin were taken. The measurements were repeated (≥ 48 h) after the final exercise session in the second study.

Active and inactive men differed significantly for a number of characteristics including ghrelin, insulin and HOMA-IR (Table 1a). When the data were pooled ($n = 44$) the strongest correlates of fasting insulin and HOMA-IR were percent fat mass ($r = 0.57$ and $r = 0.56$, $p < 0.01$) and resting heart rate ($r = 0.56$ and $r = 0.54$, $p < 0.01$) and the strongest correlate of ghrelin was AEE ($r = 0.33$, $p < 0.05$).

Following the 4-wk exercise intervention in inactive males, despite a modest reduction in body weight and fat mass and 13 % increase in VO₂max, fasting glucose, insulin and ghrelin were unchanged (Table 1b). Individual variability in response was explained mostly by changes in body fat, with changes in insulin and HOMA-IR from pre-to post-intervention being associated with changes in body fat (insulin: $r = 0.69$, $p < 0.01$; HOMA-IR: $r = 0.70$, $p < 0.01$). Change in glucose was negatively correlated with baseline glucose levels ($r = -0.68$, $p < 0.01$). Ghrelin did not correlate with changes in other variables or with baseline values.

Table 1a. $n = 22$ per group

Table 1b. $n = 15$ Pre/Post Intervention

	Inactive		Active		P-value	Pre		Post		P-value
	Mean	SE	Mean	SE		Mean	SE	Mean	SE	
Weight (kg)	87.1	15.8	79.2	11.7	0.07	95.6	13.0	94.7	13.0	<0.01
BMI (kg/m ²)	27.4	4.2	24.5	2.6	0.02	29.7	3.3	29.3	3.2	<0.01
Body Fat (%)	26.2	8.7	14.3	5.8	<0.01	30.0	6.8	29.0	6.7	0.01
VO ₂ max (ml/kg/min)	35.3	6.5 ¹	46.4	8.0 ²	<0.01	34.3	5.9	38.7	5.9	<0.01
Ghrelin (ng/L)	797	341	1067	308	0.01	805	338	761	331	0.12
Glucose (mmol/L)	5.4	0.3	5.5	0.5	0.35	5.5	0.3	5.4	0.2	0.39
Insulin (mU/L)	8.9	4.3	4.7	2.7	<0.01	9.4	4.7	8.7	4.2	0.19
HOMA-IR	2.1	1.1	1.2	0.8	<0.01	2.3	1.2	2.1	1.1	0.20

P-value: Independent t-test (Table 1a), Paired t-test (Table 1b). ¹ $n = 19$, ² $n = 13$.

In conclusion, fasting ghrelin is higher and insulin and HOMA-IR lower in active compared to inactive men. Despite significant improvements in VO₂max, four weeks of exercise training did not alter these measures in inactive overweight men. In the absence of acute exercise effects, these markers may only adapt to a greater volume of exercise or changes in other characteristics associated with regular exercise including reduced fat mass. Findings support the view that exercise should be performed on a regular basis or to induce a greater reduction in fat mass to improve fasting insulin and HOMA-IR. Further work is required to examine implications of these findings for food intake regulation with exercise.

1. Ross R (2003) *Diab Care* 26, 944–945.