Plasma Apelin Unchanged With Acute Exercise Insulin Sensitization

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Abstract

Blood glucose and insulin responses to aerobic exercise are well defined yet the mechanisms effecting post-exercise insulin sensitization remain incomplete. Apelin has been reported to enhance glucose uptake and insulin sensitivity in vivo, but its role as a regulator of insulin sensitivity following acute aerobic exercise has not been investigated. Therefore, the purpose of this study was to investigate apelin’s response to acute bouts of maximal and sub-maximal exercise and to elucidate apelin’s influence on insulin sensitivity. Twelve (22.8 ± 2.9 yrs) healthy male (n = 7) and female (n = 5) subjects completed a graded to maximal (VO₂max) and sub-maximal (70-75% VO₂max) treadmill running bouts, as well as a 50g glucose challenge (GC). Blood was obtained at four time points (pre, post, 1hr post and 24hrs post) and assessed for glucose, insulin and apelin. Hepatic insulin sensitiv-ity was assessed at rest and at 1hr and 24hrs via HOMA-IR and QUICKI indices. Results demonstrated that plasma apelin did not significantly change by condition (p = 0.324) or time (p = 0.633). Blood glucose and plasma insulin were significantly elevated immediately after VO₂max and GC, but remained stable after sub-maximal exercise. Insulin sensitivity was significantly improved 1hr post-submaximal exercise, per HOMA-IR and QUICKI indices. Results demonstrated that plasma apelin did not significantly change by condition (p = 0.034) or time (p = 0.018). Plasma apelin was significantly correlated with plasma insulin (r = 0.699, p = 0.011), HOMA-IR (r = 0.626, p = 0.029) and QUICKI (r = 0.660, p = 0.019) at rest. We conclude that, although hepatic insulin sensitivity was improved 1hr post-submaximal exercise, this exercise-induced insulin sensitization occurred independent of plasma apelin changes.

Key words: Aerobic exercise, QUICKI, HOMA-IR, glucose challenge.

Introduction

Apelin is a peptide secreted from various tissues and has been classically characterized as an adipokine, though it has recently been described as a myokine (Besse-Patin et al., 2014; Kleinz and Davenport, 2005). Apelin is purported to assist in regulating glucose homeostasis (Dray et al., 2010; Dray et al., 2008) and plays an integral role in insulin sensitivity in mice (Yue et al., 2010). Additionally, apelin was noted to improve peripheral glucose uptake in normal and insulin-resistant mice (Dray et al., 2008) and apelin injections during a hyperinsulinemic-euglycemic clamp enhanced glucose disposal (Dray et al., 2010). Apelin-stimulated glucose uptake was confirmed in isolated adipocytes from healthy (Attane et al., 2011; Than et al., 2014) and type 2 diabetic (T2D) subjects (Dray et al., 2010). These results support apelin’s role as an exogenous insulin sensitizer under hyperinsulinemic conditions. However, apelin’s effect under normal insulinemia and its role as an endogenous glucose regulator in healthy individuals during and following exercise has not been determined and requires further clarity (Alexiadou et al., 2012).

Apelin is chiefly influenced by insulin, hypoxia and adiposity, among other factors (Kleinz and Davenport, 2005). Insulin is considered the prime regulator of apelin stimulating its synthesis and release (Boucher et al., 2005). Conversely, apelin exerts a depressive effect on insulin, acting via G proteins in islet cells (Ringström et al., 2010). Acute aerobic exercise, as a result of repeated muscular contractions, stimulates insulin-independent glucose uptake via GLUT translocation in skeletal muscle, and subsequently confers both peripheral and hepatic sensitization to insulin that typically lasts hours to days (Henriksen, 2002; Holloszy, 2005). This improved sensitization to insulin after acute aerobic exercise may be partially attributable to apelin. Previous studies concerning exercise and apelin have focused on apelin’s response to training in obese and T2D (Besse-Patin et al., 2014; Krist et al., 2013), populations known to exhibit considerable variability in basal apelin concentration due to a variety of contributing factors (Alexiadou et al., 2012, Castan-Laurell et al., 2012, Castan-Laurell et al., 2008, Cavallo et al., 2012, Dray et al., 2010, Krist et al., 2013).

Enhanced contraction-induced glucose uptake lasts up to two hours post-exercise, however, improved insulin sensitivity may last longer (Borghouts and Keizer, 2000; Holloszy, 2005). The benefits of insulin sensitivity improvements are transient and return to normal 12-48 hours post-exercise (Borghouts and Keizer, 2000; Christiansen et al., 2010; Fontana et al., 2010; Holloszy, 2005; Magkos et al., 2010). Endurance training studies have reported insulin sensitivity reversal after cessation of acute exercise (Heath et al., 1983; LeBlanc et al., 1981). Thus, insulin sensitivity improvements appear to be facilitated primarily by single exercise bouts (Goodyear and Kahn, 1998). Given this, acute aerobic exercise represents an excellent model by which the effect of apelin upon insulin sensitivity can be assessed. High-intensity aerobic exercise (e.g. VO₂max) demonstrably elevates blood glucose and insulin, while moderate-intensity aerobic exercise maintains blood glucose and insulin (Marlliss and Vranić, 2002); thus, these represent two exercise conditions with readily reproducible, disparate outcomes. Therefore, the primary aim of this study was to determine if plasma apelin concentration is altered by either bout of acute aerobic exercise or whether this is related to insulin-mediated sensitization in a healthy adult population.

Methods

Subjects

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The IRB of UNC Greensboro approved this study and all participants signed informed consent on their first visit. Twelve (n = 7 male; n = 5 female) apparently healthy subjects (22.8 ± 2.9 years), non-tobacco users and not taking medications or supplements for at least 6 months that could alter metabolism, oxidation status, blood glucose and/or insulin, were recruited. Subjects were instructed to maintain their normal diet throughout the study and dietary logs were reviewed to ensure compliance.

Pre-screening procedures
Volunteers (18-35 yrs) were screened for medical, metabolic, cardiovascular and activity factors, per American Heart Association (AHA) and American College of Sports Medicine (ACSM) guidelines (Pescatello, 2014). Obese, hypertensive or pregnant participants were excluded. Resting heart rate (HR), blood pressures (BP), % body fat (%BF) (7-site determination via Siri equation, Jackson and Pollock, 1978), weight and height were determined. Once all criteria were satisfied participants became enrolled subjects. Subjects were provided food logs to record diet intake three days prior to each visit, and for the 24-hour period post-treatment prior to the fourth blood draw time point.

Maximal and submaximal exercise and negative control condition procedures
Subjects reported to the laboratory (6-9 A.M.) on three different days to complete two different exercise conditions (graded to maximal [VO2max] and steady state [70-75% VO2max] aerobic exercise on treadmill) and a negative control glucose challenge (GC) of 50g, in a post-absorptive state (10-12 hrs) and after having not exercised ≥24 hrs. Each condition was separated by at least three days, but not more than 14 days and subjects returned 24 hrs after each condition. The first visit was randomized, either VO2max or GC, with the 30-minute bout (70-75% VO2max) necessarily performed after VO2max (2nd or 3rd visit).

Maximal treadmill graded exercise
Subjects completed a warm-up (5 min) on the treadmill until HR rose to 130 bpm. The test starting speed ranged between 5.5 and 8 mph amongst all subjects. Thereafter, both grade (by 2.5%) and speed (by 0.5 mph) increased each minute until reaching VO2max or volitional fatigue. Expired gases were collected and analyzed by a Parvo Medics True One 2400 analyzer system (Parvo Medics, Sandy, UT, USA) calibrated to known gases. Ratings of perceived exertion (RPE) were recorded each minute (Noble et al., 1983). All subjects satisfied at least four of five required criteria for a true VO2max (Midgley et al., 2007; Pescatello, 2014). Subjects were given water before and after the graded to maximal exercise trial and encouraged to drink water ad libitum (up to 1 L) in the 1-hr recovery period.

Submaximal treadmill exercise
Subjects completed a warm-up (3-5 min) on the treadmill. HR was continually monitored during the 70-75% VO2max exercise (Polar Electro Inc., Bethpage, NY, USA) and recorded every 5 minutes. VO2 was confirmed in the initial 5 minutes and for 1 minute every 5 minutes during the test to ensure proper intensity. Subject RPE was obtained every 5 minutes to further ensure appropriate subjective intensity was maintained. Subjects were given water every 5 minutes during the exercise and encouraged to drink water ad libitum (up to 1 L) in the 1 hour recovery period.

Blood collection and handling procedures
Subjects rested >15 minutes before blood was obtained from an antecubital vein prior to any intervention. Blood was drawn into vacutainer EDTA tubes and a small sample of whole blood (<10µl) was used to assess blood glucose immediately. Hematocrit (Hct) was determined by taking <25µl blood drawn into micro-hematocrit capillary tubes, sealed and centrifuged (micro centrifuge at 3000 rpm 5 min) and volume ratio determined (in duplicate). Blood was immediately centrifuged (Allegro, Beckman Coulter, Indianapolis, IN, USA) for 10 minutes at 3000 rpm (4°C). Plasma was aliquoted into tubes and placed in an -80°C freezer until analyzed. Assays for plasma apelin and plasma insulin were completed with batched samples.

Assays and insulin sensitivity measures
Blood glucose was determined immediately by a glucose meter (Bayer Contour, Bayer HealthCare, LLC, Whippany, NJ, USA), which was calibrated prior to use (within 0.2 mM). Plasma insulin was assayed using an ELISA immunoassay (#10-1113-01 Mercodia, Winston-Salem, NC, USA). Plasma apelin was assayed using an ELISA immunoassay (EK-057-15 Phoenix Pharmaceuticals, Inc., Burlingame, CA, USA), which was noted to have 100% cross-reactivity with human apelin-36, -13 and -12. Absorption for both assays was read at 450 nm via KC Junior microplate reader (BioTek Instruments, Winooski, VT, USA). Intra-assay variance was <10% for insulin and <8% for apelin. Using batched samples from the same subject on common plates eliminated inter-assay variance. Plasma lactate was determined monitoring absorption change at 340 nm using LDH enzyme (Sigma-Aldrich, Inc., St. Louis, MO, USA) on a Shimadzu (Shimadzu Scientific Instruments, Columbia, MD, USA) 1800 dual beam spectrophotometer and compared to lactate standards. HOMA-IR and QUICKI indices were calculated using the formulas: HOMA-IR = [FPG][FIPI] / 405 and QUICKI = 1 / (log (FPI) + log (FPG)), where FPG is fasting plasma glucose, in mg/dl, and FPI is fasting plasma insulin, in µU/mL. Both HOMA-IR and QUICKI indices have been reported to correlate well with glucose clamp methodologies representing hepatic insulin sensitivity measures (Marliiss and Vranic, 2002). All assays were conducted using a minimum of duplicates for each sample.

Statistical analysis
A three (3) condition (two exercise conditions [VO2max and 70-75% VO2max] and one GC) x four (4) time points (pre-, immediately post-, 1 hr post- and 24hrs post-condition) repeated measures analysis of variance (RMANOVA) design was employed. Bonferroni adjustments were utilized to evaluate main time effects if needed. Pearson product moment correlation analysis between apelin and insulin, as well as HOMA-IR and QUICKI, were
Table 1. Descriptive characteristics of participants. Data are presented as mean ± SD.

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<th>Variable</th>
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<tr>
<td>Age (yr)</td>
<td>22.75 ± 2.96</td>
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<td>Weight (kg)</td>
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<td>Height (m)</td>
<td>1.71 ± 0.08</td>
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<td>BMI (kg·m⁻²)</td>
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<td>23.66 ± 3.96</td>
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<td>BF%</td>
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<td>11.02 ± 6.11*</td>
<td>21.33 ± 6.84*</td>
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<td>HR (bpm)</td>
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<td>SBP (mmHg)</td>
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<td>DBP (mmHg)</td>
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<td>76.57 ± 8.04</td>
<td>79.6 ± 6.54</td>
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<td>MAP (mmHg)</td>
<td>91.78 ± 7.29</td>
<td>91.14 ± 8.51</td>
<td>92.67 ± 6.02</td>
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<td>VO2max (L·min⁻¹)</td>
<td>51.14 ± 11.07</td>
<td>56.17 ± 10.32</td>
<td>44.09 ± 8.42</td>
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<tr>
<td>VO2max (ml·kg⁻¹·min⁻¹)</td>
<td>51.14 ± 11.07</td>
<td>56.17 ± 10.32</td>
<td>44.09 ± 8.42</td>
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</tbody>
</table>

* = Significant difference between sex (p < 0.05).

Data were analyzed using SPSS 21 software (IBM, Armonk, NY, USA) with significance set at P ≤ 0.05.

**Results**

**Subject characteristics**

Each subject completed all treatment conditions, as well as achieved true VO2max (51.14 ± 11.07 ml·kg⁻¹·min⁻¹). VO2max (L·min⁻¹) and %BF were significantly different between genders (Table 1).

**VO2max and 70-75% VO2max exercise outcomes**

Total work completed for the submaximal exercise was not significantly different between genders (p = 0.118), though males (1064.5 ± 122.18 W) performed significantly more work during VO2max than females (612.97 ± 111.53 W). The mean duration of VO2max across all subjects was 7.21 ± 0.3 mins. Mean HRmax values (187.2 ± 1.8 bpm) were similar between genders. Exercise intensity was maintained (72.71 ± 1.01%) between 70.62-74.33% for the 70-75% VO2max, with mean HR (166.22 ± 3.36 bpm). Plasma lactate at rest was similar across exercise visits (Figure 1) with post-VO2max (11.79 ± 0.51 mM) lactate elevated greater than 70-75% VO2max (2.72 ± 0.45 mM) lactate (p < 0.01).

**Figure 1. Plasma Lactate Response to Exercise.** Plasma lactate (mean ± SEM) response to both VO2max (black) and 70-75% VO2max (grey). * Significantly greater than pre (p < 0.05). †Significantly greater than 70-75% VO2max post (p < 0.01).

**Figure 2. Blood glucose, plasma insulin and apelin.** Values are (mean ± SEM) response to VO2max (black), 70-75% VO2max (striped) and GC (gray) across each of four time points. Blood glucose (A), Insulin (B) and Apelin (C) * Significantly greater than all other time points (p < 0.05). § Significantly lower than pre (p < 0.05).

**Hematocrit, blood glucose, plasma insulin and plasma apelin**

No differences in Hct were observed across time in any condition. Blood glucose analysis revealed a significant time (p < 0.001), condition (p = 0.016) and condition by time interaction (p = 0.014). Plasma insulin had a significant time (p < 0.001), condition (p < 0.001) and condition by time (P=0.001) effect. Post-hoc analysis
revealed that blood glucose was significantly elevated immediately post-VO2max and post-GC and returned to normal 1hr post-treatment (Figure 2). Insulin was similar at rest across visits and increased significantly immediately post-VO2max (21.49 mU/L, p < 0.001) and post-GC (30.51 mU/L, p < 0.001) and returned to normal 1hr post-treatment. The submaximal exercise resulted in a significant decrease in insulin 1hr post-exercise compared to pre-exercise. Plasma apelin remained unchanged for both time (p = 0.633) and condition (p = 0.324), though a near-significant interaction effect occurred (P=0.073) (Figure 2).

**Plasma apelin and insulin sensitivity**

The HOMA-IR and QUICKI index scores over time for each condition are displayed in Figure 3. HOMA-IR and QUICKI scores immediately post-treatment are not shown in Figure 3, as these measures do not reflect steady state, nor do they adequately approximate hepatic insulin sensitivity in the period immediately following exercise. No significant effects by gender were noted for HOMA-IR (p = 0.736) or QUICKI (p =0.350). Compared to resting, HOMA-IR significantly decreased (p = 0.034) and QUICKI significantly increased (p =0.018) at 1hr post-70-75% VO2max but was reversed at 24hr post-70-75% VO2max. Insulin sensitivity remained unchanged for both VO2max and GC conditions.

**Figure 3. HOMA-IR and QUICKI indices.** Values are (mean ± SEM), response to VO2max (black), 70-75% VO2max (striped) and GC (gray). HOMA-IR (A) and QUICKI (B) indices. Immediate post measures have been omitted, as these do not reflect the resting requirement of indirect insulin sensitivity measures. § Significantly lower than pre (p < 0.05). *Significantly greater than pre (p < 0.05).

The relationship between plasma apelin and insulin, as well as with HOMA-IR and QUICKI are shown in Figure 4. Resting plasma apelin significantly correlated with plasma insulin (r = 0.699, p = 0.011), HOMA-IR (r = 0.626, p = 0.029) and QUICKI (r = 0.660, p = 0.019) scores.

**Discussion**

This study sought to establish resting plasma apelin concentration in a healthy adult cohort, as well as to assess the relationship of plasma apelin to insulin sensitivity, both at rest and following exercise. The current study noted no significant difference in resting plasma apelin concentration by gender (p = 0.141), which is supported by previous research (Krist et al., 2013). Additionally, resting plasma apelin concentration amongst all 12 subjects remained stable across visits (p = 0.990) at the times measured.

VO2max, 70-75% VO2max and GC elicited hypothesized blood glucose and plasma insulin responses; blood glucose and insulin were significantly elevated after VO2max and GC but not following submaximal exercise. Despite the anticipated elevation in glucose and insulin, plasma apelin was not significantly altered from baseline immediately post-, one hour post- or 24 hours post-treatment following VO2max and GC. The response of glucose and insulin to GC in our study mirrors the effects observed from a 75g glucose bolus in healthy subjects (Alexiadou et al., 2012). As such, our choice to use this modified glucose load in apparently healthy subjects was supported. Our results also support a previous finding that a glucose load of sufficient dose to increase insulin does not elevate apelin in apparently healthy subjects (Alexiadou et al., 2012). Laboratory and subject time constraints, as well as costs, precluded the performance of a 75g, two-hour OGTT, prompting utilization of the 50g glucose bolus. The observed insulin sensitivity responses following VO2max and 70-75% VO2max aerobic exercise conditions confirm the findings of Brestoff et al. (2009), that 45 minutes at 75% VO2max aerobic cycling on an ergometer, but not supramaximal cycling exercise, improved insulin sensitivity. However, in their study, insulin sensitivity was determined via OGTT 12 hours after exercise cessation, whereas the present study assessed insulin sensitivity at one hour following exercise and glucose bolus ingestion. Our study suggests this enhanced insulin sensitivity, as assessed via hepatic indices, was not related to a change in plasma apelin at the times measured. The present study did not determine insulin sensitivity in peripheral tissues (e.g. skeletal muscle) following exercise. Computation of the Matsuda index, an indirect composite measure of whole body insulin sensitivity that considers both hepatic and peripheral tissue sensitivity, following administration of a 2 hour OGTT is warranted for future study. We assessed insulin sensitivity at one hour and 24 hours post-exercise via HOMA-IR and QUICKI indices; nevertheless it is possible that apelin demonstrated endocrine and/or myokine actions at the skeletal muscle level (Besse-Patin et al., 2014). Future study in this area should adequately assess peripheral glucose disposal via administration of a 12- or 24-hour post-treatment OGTT or through application of the minimal model to stable label intravenous glucose tolerance test findings (Avogaro et al., 1989).

Apelin concentration remained within a narrow range across time after exercise (0.2-1.0 ng/ml) in the...
present study. No significant change in plasma apelin was observed, despite VO₂max eliciting blood glucose and plasma insulin elevations. Moreover, a significant correlation was revealed between plasma apelin and plasma insulin ($r = 0.699$, $p = 0.011$) at rest, suggesting that elevated insulin could lead to enhanced plasma apelin. It is possible that the transient insulin elevation might not have met the threshold necessary to significantly alter apelin or the length of time was insufficient. Our study suggests that apelin is unaffected by the transient hyperinsulinemia in the period immediately following an acute graded maximal aerobic exercise test. Additionally, apelin concentration may have peaked outside of our sampling window, as apelin half-life ($t_{1/2}$) was noted previously to be $< 5$ minutes (Japp et al., 2010). This potential limitation could be mitigated with increased sampling frequency over the two-hour window following exercise or GC treatment. Greater sampling frequency, in concert with a glucose tolerance test, as noted above, may shed further light on the potential role of apelin as an endogenous insulin sensitizer in vivo after exercise.

A broad time course for insulin-induced apelin induction and secretion signaling has been previously proposed, with in vitro insulin administration upregulating apelin mRNA significantly between three and twelve hours post-treatment in isolated adipocytes (Borghouts and Keizer, 2000; Daviaud et al., 2006). These findings, however, do not necessarily reflect in vivo insulin-apelin signaling or local apelin action on peripheral insulin-sensitive tissues. Apelin’s short circulating $t_{1/2}$ could also have affected our findings. Despite the relatively short apelin $t_{1/2}$, substantive physiological effects may not be elicited during this time period, perhaps requiring longer duration to manifest changes in circulating apelin and influence insulin sensitivity. Apelin itself is also known to exert considerable influence on insulin secretion, inhibiting glucose-stimulated insulin release (Ringström et al., 2010; Sörhede-Winzell et al., 2005). Consequently, full consideration of this dynamic relationship should be further investigated, especially during the post- to one-hour post-treatment window and between the one to 24 hour post time.

Thirty minutes of treadmill running at 70-75% VO₂max in the present study elicited a significant improvement in insulin sensitivity at one hour post-exercise, per HOMA and QUICKI scores. However, VO₂max did not elicit insulin sensitivity improvements. Maximal aerobic exercise did not enhance insulin sensitivity likely due to its short exercise duration ($7.21 \pm 0.3$ mins); moreover, subjects spent only several minutes of this time performing aerobic exercise near or at their maximal aerobic capacities. Submaximal exercise likely elicited significant improvements to HOMA and QUICKI scores through sufficient duration or threshold energy expenditure that exercise of shorter duration, independent of intensity, does not elicit (Magkos et al., 2008). Future studies also should focus on exercise chronicity, as repeated acute bouts or aerobic or interval training may elicit interesting findings, such as those noted in a recent study (Izadi et al., 2018). Interestingly, an improvement in insulin sensitivity was observed in our cohort following the submaximal bout, despite our subjects entering the study as healthy adults with normal glucose tolerance. Furthermore, significant relationships were noted between resting plasma apelin and HOMA-IR ($r = 0.626$, $p = 0.029$) and QUICKI ($r = 0.660$, $p = 0.019$) scores, supporting claims of elevated plasma apelin with insulin resistance (Alexiadou et al., 2012; Cavallo et al., 2012; Krist et al., 2013). Further studies of a similar acute nature should be undertaken in obese and/or T2D populations, so as to observe whether any differences in apelin alteration may be noted compared to our normal, apparently healthy cohort.

**Conclusion**

The current study demonstrated that circulating apelin does not respond significantly to either acute graded maximal exercise or steady state moderate intensity exercise and did not seem to contribute to hepatic exercise-induced improvements in insulin sensitivity one hour following steady state moderate exercise. Additionally, the transient enhancement in insulin sensitivity one hour following submaximal exercise suggests that this response was independent of circulating apelin changes. Other factors may have contributed to improved insulin sensitivity following submaximal exercise such as: nitric oxide (Roy et al., 1998), calcium and calmodulin/Ca²⁺-dependent protein kinases (Stanford and Goodyear, 2014), AMPK or PI3K/Akt (O’Neill, 2013), hypoxia and ROS (Sandström et al., 2006) or GLUT4 upregulation. Research remains largely equivocal on the mechanism of exercise-mediated improvements in insulin-dependent insulin sensitivity. The improvement of insulin sensitivity following a single bout of exercise is undoubtedly a multifaceted phenomenon, involving a number of key proteins, blood factors and glucose receptor regulation in insulin-sensitive tissues.

**Acknowledgements**

The study comply with the current laws of the country in which were performed. The authors report no conflict of interest.

**References**


Plasma apelin unchanged to acute exercise

Key points

- Acute steady state aerobic exercise enhances insulin sensitization without an increase in plasma apelin in apparently healthy young subjects.
- Graded exercise to VO2max increases plasma glucose and insulin without a concomitant change in plasma apelin.
- An acute glucose load can increase plasma glucose and insulin but does not increase plasma apelin in apparently healthy young subjects.

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Research interests

The endocrine effects of physical activity and exercise, sport nutrition and the metabolic effects of acute and chronic dietary intervention.

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