Abstract

Adipose tissue functions as an endocrine organ which releases biologically active adipokines. Adiponectin, an adipocyte-derived protein structurally similar to complement 1q, plays a significant role in metabolic disorders, due to its insulin sensitizing, anti-inflammatory and anti-atherogenic properties. AdipoR1 and AdipoR2, mediate the metabolic actions of adiponectin by activating adenosine monophosphate-activated protein kinase (AMPK) and peroxisome proliferator-activated receptors-alpha (PPAR-α) which leads to an increase in fatty acid combustion and energy consumption, fatty acid oxidation and glucose uptake in myocytes and reduces gluconeogenesis and thus leads to increased insulin sensitivity. Plasma adiponectin level is affected by multiple factors: gender (females have higher plasma adiponectin levels), obesity-linked diseases (metabolic syndrom, diabetes mellitus type 2 and atherosclerosis are associated with lower adiponectin levels), lifestyle - including exercise. Yet, to date, little is known about the response of adiponectin concentrations to exercise and, in particular, the response of this hormone to training in population of athletes. The aim of this review is to overview the published evidence for the effects of exercise on adiponectin levels in athletes.

Adiponectin concentration presents a delayed increase (30 min) after short-term intense performance, by athletes, both male and female. It seems that adiponectin concentrations do not change in response to long-term exercise. No significant difference was found in total adiponectin and/or high-molecular weight (HMW) oligomers in long-term effects of high physical training in athletes. Adiponectin can serve to monitor training loads and the establishment of individual limit values of training loads. Further studies are needed to clarify possible mechanisms by which adiponectin might influence energy homeostasis during heavy training in elite athletes.

Key words: adiponectin, adipose tissue, athletes, exercise.

Efekat treninga na vrednosti adiponektina u plazmi vrhunskih sportista

Sažetak

Masno tkivo je endokrini organ, koji oslobađa biološki aktivne adipokine. Jedan od adipokina je adiponektin, protein strukturno sličan komplementu 1q, koji ima značajnu ulogu u metabolitskim poremećajima zbog antiinflamatornog dejstva, anti-aterogenih svojstava i dejstva na insulin. Receptori AdipoR1 i AdipoR2 posreduju metaboličko dejstvo adiponektina aktiviranjem adenosin-monofosfat aktivirane protein-kinaze (AMPK) i perokszizom proliferator aktiviranog receptora alfa (PPAR-α), što dovodi do povećanog katabolizma i oksidacije masnih kiselina, preuzimanja glukoze u miocitima, smanjenja gluconeogeneze i povećanja osetljivosti na insulin. Na nivo adiponektina utiče više činilaca: pol (žene imaju više vrednosti adiponektina u plazmi), bolesti povezane sa gojaznošću (metabolički sindrom, tip 2 dijabetesa melitusa i aterosklerozu su povezane sa nižim vrednostima adiponektina) i način života (uključujući vežbanje). Do danas se ipak malo zna o uticaju rekretivnog vežbanja na nivo adiponektina, a posebno o uticaju treninga na nivo adiponektina u populaciji sportista. U ovom radu smo prikazali pregled literature o efekta treninga na nivo adiponektina u plazmi vrhunskih sportista.

Koncentracija adiponektina pokazuje odloženo povećanje (30 min) nakon kratkotrajnog intenzivnog treninga u sportista oba pola. Prema podacima iz literature, koncentracije adiponektina se ne menjaju kao odgovor na dugotrajno vežbanje. Kod sportista posle treninga nema značajne razlike u nivou ukupnog adiponektina vs. oligomeru visoke molekularne težine (HMW). Adiponektin može da služi za praćenje opterećenja tokom treninga i postavljanje individualnih normi za intenzitet treninga. Potrebna su dalja istraživanja radi pojašnjenja mogućih mehanizama kojima adiponektin utiče na energetsku homeostazu kod vrhunskih sportista tokom treninga.

Ključne reči: adiponektin, masno tkivo, sportisti, trening.
adipocyte-derived protein (3), plays a significant role in metabolic disorders, such as obesity, type 2 diabetes, coronary heart disease and metabolic syndrome (4, 5) due to its insulin sensitizing, anti-inflammatory and anti-atherogenic properties (6). Structurally, adiponectin contains three distinguishable domains: an N-terminal sequence, a collagenous region and a globular domain structurally similar to complement 1q (7). It belongs to a family of proteins that form characteristic multimers. Adiponectin exists in a wide range of multimer complexes in plasma and combines via its collagen domain to create 3 major oligomeric forms: a low-molecular weight (LMW) trimer, a middle-molecular weight (MMW) hexamer and high-molecular weight (HMW) 12- to 18-mer adiponectin (8, 9). Several investigations support the hypothesis that HMW adiponectin is a more active form of the protein and has a more relevant role in insulin sensitivity and in protecting against diabetes (10). AdipoR1 and AdipoR2, mediate the metabolic actions of adiponectin through phosphorylation and activation of adenosine monophosphate-activated protein kinase (AMPK) (11). The AMPK activation stimulates phosphorylation of acetyl-CoA carboxylase, fatty acid oxidation and glucose uptake in myocytes and reduces enzymes involved in gluconeogenesis in liver, leading to reduction of glucose levels. Adiponectin also increased the expression levels of peroxisome proliferator-activated receptors-alpha (PPAR-α) in vivo, increasing fatty acid combustion and energy consumption, which led to decreased triglyceride content in the liver and skeletal muscle and thus increased insulin sensitivity (12).

There is a sexual dimorphism in the circulating levels of adiponectin. Females have higher plasma adiponectin levels (16.6 ± 5 mg/ml) than males (7.9 ± 0.5 mg/ml), suggesting that sexual hormones regulate the production of adiponectin (13). Plasma adiponectin level is affected by multiple factors: gender, aging, obesity-linked diseases (insulin resistance, diabetes mellitus type 2 and atherosclerosis), lifestyle, including exercise. It is well documented that exercise or regular physical activity has beneficial effects on metabolic and cardiovascular diseases (14). Considering previous literature, it is unclear whether exercise increases adiponectin in circulation and its receptors in insulin-sensitive tissues (15). Complicating interpretation of the existing data is dependent on multiple factors, including the pathological condition, types (endurance vs resistance exercise), intensity (low, moderate and intense) and duration of exercise (acute vs chronic, short-term vs long-term) and sex. For example, in healthy, young subjects, it seemed that both acute and chronic aerobic exercise did not alter plasma level of adiponectin (16). However, exercise in combination with diet-induced weight loss (17), significantly increases plasma adiponectin levels in both obese and insulin-resistant subjects. Furthermore, recent data suggest that exercise training might modulate the expression of AdipoR1 and AdipoR2, in PBMCs (peripheral blood mononuclear cells) in young men (18). On the other hand, there is a fact that a relationship between exercise and increased adiponectin levels was not observed in the majority of randomized controlled trials (16). Only moderate-high intensity resistance training, not low intensity, increased plasma adiponectin in inactive subjects, suggesting that the intensity of exercise may be an important factor in the expression of adiponectin.

From the point of view of the intensity of exercise, what are the possible mechanisms by which adiponectin might influence energy homeostasis during heavy training in elite athletes? Vigorous training program represents a physical stress condition in which heavy changes in energy expenditure might increase adiponectin concentration in athletes, but a number of different hormones are known to be immediately altered during intense exercise (19), such as insulin, catecholamines, and cortisol (20), and they may affect adiponectin levels via suppressed adiponectin gene expression (20).

Adiponectin influences metabolic adaptations that would prove beneficial to the endurance of athletes, but there aren’t many studies that have investigated the response of this hormone to training in a population of athletes and how adiponectin is regulated during cumulative exercise.

In professional cyclists, adiponectin significantly increased during the cycling race and recovery periods (21), when compared to the baseline. Lombardi G. et al (21) found adipokines trends were clearly related to the power output and the energy expenditures. In another study about adiponectin, significant increases were observed in cumulative exercise (22) within 5 days of commencing the race of cyclists, with these elevated values failing to return to baseline levels after 3 days of recovery, suggesting that adiponectin can serve to monitor training loads and the establishment of individual limit values.

In a study with graded treadmill walk/run protocol in trained runners (23), significant increases in post-exercise levels of adiponectin were elicited. On the other hand, no significant changes were noted in serum adiponectin levels during an ultra-marathon endurance race (24).

Jurimae et al. (25) found a significant reduction in adiponectin levels immediately after high-intensity rowing exercise and correction in plasma volume expansion, but adiponectin was significantly increased above the resting value after the first 30 min of recovery (uncorrected for plasma volume + 19.3%; corrected for plasma volume + 20.0%). Adiponectin does not change throughout the prolonged training period in elite male rowers (26). Jurimae J et al. observed differences in the study (27) of ad-
Adiponectin significantly increased, when compared to the baseline, in young female handball and basketball players (16.7 ± 7.8 vs 21.0 ± 9.8 μg/ml, p < 0.001) after intensive fitness and speed exercise (34). Plasma adiponectin remained unchanged during long-term moderate aerobic exercise in young female handball and basketball players (34) and in elite female water polo athletes throughout a season (35).

Conclusion

Adiponectin is secreted by adipocytes and has been implicated in the regulation of energy homeostasis. Vigorous training program represents a physical stress condition in which heavy changes in energy expenditure might increase adiponectin concentration in athletes. Adiponectin concentration presents a delayed increase (30 min) after short-term intense performance by trained athletes, male and female. It seems that adiponectin concentrations do not change in response to long-term exercise. Adiponectin can serve to monitor training loads and the establishment of individual limit values. Further studies are needed to clarify possible mechanisms by which adiponectin might influence energy homeostasis during heavy training in elite athletes.

Literature