

Adiponectin: A defense protein in catabolism

To the Editor:

In a recent very interesting review, Dr Fantuzzi discussed the functions of adiponectin and put forward several important questions about the role of the protein.¹ In this correspondence I will propose an explanation of why high adiponectin levels can be seen in conjunction with insulin resistance and inflammation.

Traditionally, adiponectin is considered to be a hormone with anti-inflammatory properties that in turn are protective against insulin resistance, type 2 diabetes, and atherosclerotic diseases. Proinflammatory proteins, especially TNF- α , are thought to be important inhibitors of adiponectin synthesis.² Thus it is a paradox when reports show that high adiponectin levels are seen in inflammatory diseases, such as rheumatoid arthritis and SLE. In fact, patients with SLE are more insulin resistant than control subjects, in spite of their increased adiponectin levels.¹ However, this apparent inconsistency can be approached from different points of view. The role of adiponectin, insulin, and inflammatory cytokines in an evolutionary context is not necessarily the same as their properties in conditions to which there has been no natural selection.

Insulin increases lipogenesis and synthesis of glycogen and proteins. These anabolic effects can only be exerted when substrates (ie, food) are available. Adiponectin, on the other hand, as postulated by me and lately also by others, is a protective protein in catabolic states, such as starvation.^{3,4}

Adiponectin increases fatty acid oxidation and insulin sensitivity but decreases energy expenditure and gluconeogenesis.⁵ Adiponectin is also believed to have antiproliferative properties because it impedes different growth factors.⁶ However, I do not think that adiponectin's effects on insulin sensitivity should be considered to be a response from the organism to counter type 2 diabetes. The role of adiponectin is more likely to minimize the effect of the anabolic effects of insulin when nutrients are scarce. The same argument could be put forward when the antiproliferative properties of adiponectin are discussed. These effects should rather be considered as antianabolic when energy supplies are low than being a defense mechanism against atherosclerosis. If not, what would the evolutionary mechanisms be?

Interestingly, both adiponectin and insulin decrease gluconeogenesis. This could, at first, appear slightly contradictory. However, a high insulin level normally indicates that energy is plentiful and that blood glucose levels are increased. Thus there is no need to produce glucose from nonsugar substrates. Adiponectin has an important role in starvation. During starvation, gluconeogenesis is of less importance because free fatty acids and ketone bodies replace glucose as the major source of energy.

The insulin resistance seen in inflammatory diseases in conjunction with high adiponectin levels could be explained by the opposite roles of insulin and adiponectin. An organism with inflammatory diseases is catabolic. In these states insulin resistance is a response to minimize the anabolic effects of insulin. Adiponectin, on the other hand, is a fuel-providing hormone in catabolic states, and its levels are consequently increased.

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Allergic rhinitis practice parameter update: Correction to information regarding OMNARIS (ciclesonide) nasal spray

To the Editor:

The recently published updated practice parameters for allergic rhinitis titled "The diagnosis and management of rhinitis: an updated practice parameter" contained incorrect information within Table VIII.¹ Specifically, the table inaccurately states that the age limit for OMNARIS (ciclesonide) is 12 years. Per the US Food and Drug Administration–approved package insert for OMNARIS, the product is indicated for the treatment of nasal symptoms associated with seasonal allergic rhinitis in adults and children 6 years and older and 12 years and older for perennial allergic rhinitis. The approved dose is 2 sprays per nostril once daily (200 μ g daily dose).²

We respectfully request that your readership be made aware that OMNARIS is approved for use in children 6 years and older with seasonal allergic rhinitis and 12 years and older for perennial allergic rhinitis. In addition, we are hoping that Table VIII will be updated (specifically, "2 sprays/nostril" for the "Usual child dose" column and "6 and older-SAR" and 12 and older-PAR" for the "Age limit" column) to accurately reflect the approved indication and use for OMNARIS.

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