

## Clinical Study

# Aged Garlic Extract Improves Adiponectin Levels in Subjects with Metabolic Syndrome: A Double-Blind, Placebo-Controlled, Randomized, Crossover Study

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Received 10 January 2013; Accepted 31 January 2013

Academic Editor: Giuseppe Valacchi

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**Background.** Garlic (*Allium sativum*) has been shown to have important benefits in individuals at high cardiovascular risk. The aim of the present study was to evaluate the effects of the administration of aged garlic extract (AGE) on the risk factors that constitute the cluster of metabolic syndrome (MS). **Methods and Design.** Double-blind, crossover, randomized, placebo-controlled clinical trial to assess the effect of 1.2 g/day of AGE (Kyolic), for 24 weeks of treatment (12 weeks of AGE and 12 weeks of placebo), on subjects with MS. **Results.** The administration of AGE increased the plasma levels of adiponectin ( $P = 0.027$ ). No serious side effects associated with the intervention were reported. **Conclusion.** The present results have shown for the first time that the administration of AGE for 12 weeks increased plasma adiponectin levels in patients with MS. This suggests that AGE might be a useful, novel, nonpharmacological therapeutic intervention to increase adiponectin and to prevent cardiovascular (CV) complications in individuals with MS.

## 1. Introduction

Metabolic syndrome (MS) is characterized by the presence of insulin resistance, low-degree inflammation, dysglycemia, low plasma high-density lipoprotein cholesterol (HDL-C), increased triglycerides (TG), elevated blood pressure, and abdominal obesity [1]. MS has been associated with an increased risk of type 2 diabetes mellitus (DM2) and cardiovascular diseases (CVDs) [1, 2]. The prevalence of MS varies between 15% and 40%, being greater in the population of Hispanic origin [3].

Abdominal obesity is considered a key characteristic of MS, which is related to decreased insulin-mediated glucose uptake [4]. Adipose tissue is known to express and secrete a variety of adipokines, including leptin, adiponectin, resistin,

and visfatin, as well as cytokines and chemokines such as tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin-6 (IL-6), and monocyte chemoattractant protein-1 (MCP-1) [5–8]. The release of adipokines by either adipocytes or adipose tissue-infiltrated macrophages plays a key role in the development of insulin resistance and DM2, as well as the increased risk of cardiovascular disease associated with obesity. Renin-angiotensin system components are also activated in adipose tissue, leading to hypertension and insulin resistance [4]. Adiponectin is considered to be a protective protein with antidiabetic, anti-inflammatory, and antiatherogenic effects [9]. Reduced plasma adiponectin levels have been reported in obese individuals, particularly in those with visceral obesity, and have been negatively correlated with insulin resistance. Furthermore, decreased adiponectin levels were found to