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Low advanced glycation end product diet improves the lipid and inflammatory profiles of prediabetic subjects

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BACKGROUND: Prediabetes is associated with risk for cardiovascular disease, and the first step in its management emphasizes lifestyle and diet modifications; however, modern diets are high in advanced glycation end products (dAGEs), derived from processing methods that exert a pivotal role in promoting atherosclerotic risk.

OBJECTIVE: We studied the effect of low vs standard dAGE diets (L-dAGEs vs S-dAGEs) on lipid profile, inflammation, and cardiovascular risk in prediabetic subjects.

METHODS: A 24-week randomized dietary intervention was conducted on 62 prediabetic subjects. We evaluated lipid profile, endogenous secretory receptors for AGEs, high-sensitivity C-reactive protein, arterial stiffness, and intima-media thickness.

RESULTS: After 24 weeks, patients with L-dAGEs showed a significant reduction of total cholesterol, apolipoprotein B, and low-density lipoprotein compared with controls (5.26 ± 1.09 vs 5.53 ± 0.87 mmol/L, $P < .05$; 0.77 ± 0.25 vs 1.16 ± 0.13 mmol/L, $P < .05$; and 3.53 ± 0.93 vs 3.68 ± 0.7 mmol/L, $P < .05$); with respect to baseline, high-sensitivity C-reactive protein levels were significantly reduced in the L-dAGEs group (0.21 [0.11–0.69] vs 0.12 [0.08–0.48] mg/dL, $P < .05$) but not in the S-dAGEs group. Endogenous secretory receptor for AGEs was similar in both the groups at baseline and at the 24-week follow-up. With respect to baseline, L-dAGE patients showed a significative reduction of intima-media thickness (0.77 [0.73–0.81] vs 0.73 [0.70–0.75] mm, $P < .05$). We did not observe the same reduction in S-dAGEs. No difference in arterial stiffness was found from baseline to follow-up in both the groups.

CONCLUSIONS: L-dAGEs improved the lipid and inflammatory profiles of prediabetic subjects and seemed to reduce atherosclerotic burden compared with a standard diet. Further studies are needed to recommend this dietary regimen for prevention of cardiovascular risk in prediabetes.
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Prediabetes, a common disorder of glucose homeostasis, is considered a state associated with increased risk of diabetes and cardiovascular disease.[1,2] As prediabetes does not typically present with clinical symptoms, measurement

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of its prevalence has been largely underestimated, and according to current guidelines, the first step in the management of this condition should emphasize therapeutic lifestyle modifications; however, successful management of prediabetes requires an approach that involves screening and treatment of modifiable risk factors for cardiovascular disease such as dyslipidemia and hypertension.[3]

It is well known that lifestyle or pharmacologic interventions can prevent diabetes in high-risk subjects; however, the evidence regarding the prevention of cardiovascular disease is less clear.[4–6]

The role of nutrition in the prevention of type 2 diabetes and cardiovascular disease has been extensively investigated, and specifically designed regimens based on targeted food properties have attracted attention as potentially useful in reducing the risk of developing macrovascular disease and promoting lifelong health.[6,7] In clinical practice and in most clinical studies, dietary strategies have often been centered on nutrients or caloric restriction but not on risk-associated processing methods; however, a previous study has shown that processed foods and dietary fat are high in glycotoxins known as advanced glycation end products (AGEs).[8]

AGEs form in common foods during spontaneous reactions between reducing sugars and proteins or lipids, and their potential role in promoting inflammation and atherosclerotic risk has been explored in several studies.[9,10] AGEs are naturally present in uncooked animal-derived foods, and cooking results in the formation of new AGEs within these foods. The fact that the modern diet is a large source of AGEs is now well documented[11]: grilling, broiling, roasting, searing, and frying propagate and accelerate new AGE formation. In the past few years, the potential role of dietary AGEs in human health has largely been ignored; however, recent studies with the oral administration of a single AGE-rich meal to human beings as well as labeled single-protein AGEs or diets enriched with specific AGEs such as carboxymethyllysine and methylglyoxal to mice, have clearly shown that dietary AGEs are absorbed and contribute significantly to the body's AGE pool.[8] In line with these considerations, previous studies showed that restriction of dietary AGEs directly correlates with lower circulating AGEs, such as carboxymethyllysine and methyl-glyoxal, as well as with markers of inflammation and oxidative stress in patients with diabetes or kidney disease as well as in healthy subjects.[12] AGEs play a role in different diseases increasing inflammation through a specific receptor (receptor for AGEs, RAGE). RAGE is also found in the circulation in a spliced form called endogenous secretory RAGE (esRAGE) that may contribute to the removal/neutralization of circulating ligands, thus functioning as a decoy by competing with cell-surface RAGE for ligand binding.[13]

Clinical data on dietary strategies in subjects with prediabetes have often been centered on diet composition or caloric restriction, and data on risk-associated processing methods are lacking.

The aim of this work was to investigate the effect of a controlled dietary intervention that compared the chronic effects of a low dietary advanced glycation end product (L-dAGEs) with a standard dietary AGE (S-dAGEs) regimen on lipid profile, inflammatory markers, and plasma levels of esRAGE in individuals with prediabetes. Furthermore, we examined the effects of an L-dAGE regimen on early markers of cardiovascular disease.

Materials and methods

Study design and participants

The study was a randomized, controlled, 24-week dietary observational perspective trial involving 62 adults with prediabetes attending our University Hospital for diabetes and cardiovascular risk evaluation. The inclusion criteria were the following: age range between 35 and 65 years; body mass index (BMI) between 18.5 and 40 kg/m^2; prediabetes identified according to the American Diabetes Association (ADA) recommendation (impaired fasting glucose and/or impaired glucose tolerance, and/or glycated hemoglobin [HbA_{1c}] between 5.7% and 6.4%), and Caucasian race. The exclusion criteria were the following: low-density lipoprotein (LDL) cholesterol serum levels $\geq$190 mg/dL at baseline; previous history of diabetes; previous history of overt cardiovascular events (atrial fibrillation, stroke, ischemic heart disease, chronic obstructive peripheral arteriopathy, or heart failure); active smoking; clinical evidence of advanced liver or renal disease; anemia or hemoglobinopathies; use of medications known to affect glucose metabolism or lipid profile; use of vitamin supplements; major food allergies; history of eating disorders or dietary pattern different from the typical Mediterranean diet; significant weight loss or change in dietary habits in the previous 3 months; chronic gastrointestinal diseases associated with malabsorption or chronic pancreatitis; rheumatic diseases; and/or recent history of acute illness, malignant disease, and drug or alcohol abuse.

Participants who met the inclusion criteria and gave informed consent returned on a subsequent morning for screening tests including a physical examination and review of their clinical history, smoking status, and alcohol consumption.

Inclusion visit

Anthropometric characteristics were registered, and cardiovascular risk evaluation was performed (arterial stiffness and intima-media thickness [IMT]). Body weight and height were measured, and BMI was calculated as weight (kg)/height (m^2). Blood pressure (BP) was measured with a calibrated sphygmomanometer after the subject had rested in the supine position for 10 minutes. Venous blood samples were withdrawn from the antecubital vein on the morning after an overnight fast. Baseline venous blood

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