NUTRITION RESEARCH 34 (2014) 391–400

Available online at www.sciencedirect.com

ScienceDirect

www.nrjournal.com

Postprandial oxidative stress is increased after a phytonutrient-poor food but not after a kilojoule-matched phytonutrient-rich food

Amanda Khor[a,b], *Ross Grant*[a,b,d,*], *Chin Tung*[b], *Jade Guest*[a,b], *Belinda Pope*[a,b], *Margaret Morris*[a], *Ayse Bilgin*[c]

[a] *School of Medical Sciences, Faculty of Medicine, University of New South Wales, NSW, Australia*
[b] *Australasian Research Institute, Sydney Adventist Hospital, NSW, Australia*
[c] *Department of Statistics, Macquarie University, NSW, Australia*
[d] *Sydney Medical School, University of Sydney, NSW, Australia*

ARTICLE INFO

Article history:
Received 18 September 2013
Revised 6 April 2014
Accepted 9 April 2014

Keywords:
Kilojoule
Fat
Sugar
Postprandial
Oxidative stress
Inflammation
Human

ABSTRACT

Research indicates that energy-dense foods increase inflammation and oxidative activity, thereby contributing to the development of vascular disease. However, it is not clear whether the high kilojoule load alone, irrespective of the nutritional content of the ingested food, produces the postprandial oxidative and inflammatory activity. This study investigated the hypothesis that ingestion of a high-fat, high-sugar, phytonutrient-reduced food (ice cream) would increase oxidative and inflammatory activity greater than a kilojoule-equivalent meal of a phytonutrient-rich whole food (avocado). The individual contributions of the fat/protein and sugar components of the ice cream meal to postprandial inflammation and oxidative stress were also quantified. Using a randomized, crossover design, 11 healthy participants ingested 4 test meals: ice cream, avocado, the fat/protein component in ice cream, and the sugar equivalent component in ice cream. Plasma glucose, cholesterol, triglycerides, and inflammatory and oxidative stress markers were measured at baseline and 1, 2, and 4 hours (t1, t2, t4) after ingestion. Lipid peroxidation was increased at 2 hours after eating fat/protein (t0-t2, $P < .05$) and sugar (t1-t2, $P < .05$; t1-t4, $P < .05$). Antioxidant capacity was decreased at 4 hours after eating ice cream (t0-t4, $P < .01$) and sugar (t0-t4, $P < .01$). Ingestion of a kilojoule-equivalent avocado meal did not produce any changes in either inflammatory or oxidative stress markers. These data indicate that the ingestion of a phytonutrient-poor food and its individual fat/protein or sugar components increase plasma oxidative activity. This is not observed after ingestion of a kilojoule-equivalent phytonutrient-rich food.

Abbreviations: ABTS, 2,2'-azino-di-[3-ethylbenzthiazoline sulfonate]; AUC, area under curve; ELISA, enzyme-linked immunosorbent assay; IL-6, interleukin 6; MDA, malondialdehyde; MFI, mean fluorescence intensity; NADPH, nicotinamide adenine dinucleotide phosphate; ROS, reactive oxygen species; TBA, thiobarbituric acid.

* *Corresponding author*. School of Medical Sciences, Wallace Wurth building, University of New South Wales, Sydney, NSW, 2052, Australia. Tel.: +61 2 9487 9602; fax: +61 2 9487 9626.
E-mail address: r.grant@unsw.edu.au (R. Grant).

http://dx.doi.org/10.1016/j.nutres.2014.04.005
0271-5317/

1. Introduction

Subclinical inflammation and oxidative stress are key players in the pathogenesis of non-communicable metabolic diseases such as coronary heart disease [1–3], cancer [4,5], and dementia [6,7]. Elevated plasma concentrations of inflammatory markers such as C-reactive protein [8] and interleukin 6 (IL-6) [9], oxidative stress markers including malondialdehyde (MDA) [10] and oxidized low-density lipoprotein to low-density lipoprotein cholesterol ratio, and reduced antioxidant capacity [11] have been correlated to coronary heart disease.

Although epidemiological evidence has shown that a diet high in saturated fats and low in fruits and vegetables is a risk factor for chronic metabolic disease [12], it is only in recent times that research has provided evidence of a direct pathophysiological link between foods high in saturated fats and/or sugars and the inflammatory and oxidative activity accompanying chronic metabolic diseases [13–16]. More specifically, postprandial lipemia and subsequent inflammatory activity are being increasingly reported as independent risk factors for atherosclerosis [17]. Hypertriglyceridemia, in particular, is thought to lead to acute inflammatory and oxidative stress cascades [18].

Researchers have begun to explore the mechanisms linking intake of specific foods and the acute inflammatory response seen in the postprandial period. Alipour et al [19] showed that oral consumption of fats resulted in leukocytes enriched with fatty acids resulting in their activation [19]. Erridge et al [20] reported that ingestion of a high-fat meal could also increase postprandial inflammation through an elevation in plasma lipopolysaccharide. The transient exposure of monocytes to lipopolysaccharide in this study resulted in the release of tumor necrosis factor-α, a key proinflammatory cytokine [20].

Hyperglycemic conditions and the presence of advanced glycation end products have also been shown to increase endothelial nicotinamide adenine dinucleotide phosphate (NADPH) oxidase activity and hence reactive oxygen species (ROS) production [21].

Consistent with the biochemical changes discussed above, reduced-kilojoule diets have been associated with lower levels of inflammation and oxidative stress markers. Mraz et al [22] showed that a 2-week consumption of a diet containing less than 2500 kJ/d decreased inflammatory markers and mRNA expression of chemokines and chemokine receptors in monocytes of obese, diabetic females. Skrha et al [23] also reported a decrease in oxidative markers in obese diabetic and nondiabetic participants when placed on a diet of 2500 kJ for 1 week, whereas inflammatory and oxidative stress markers dropped after an 8-week diet of alternate-day calorie restriction of 1339 to 1590 kJ in obese, asthmatic patients [24]. Although these studies showed that low-kilojoule diets could decrease inflammation and oxidative stress over the longer term, the immediate postprandial effects were not assessed.

Recent studies have shown that inflammatory and oxidative activity increases within hours of ingestion of a complex meal [16,25–27]. However, the use of complex meals means that the postprandial inflammatory and oxidative stress response cannot be attributed to a specific nutritional component, such as the fat or sugar in the meal. To address this knowledge gap, a small number of studies have investigated the effects of fat and sugar, separately, on inflammation and oxidative stress. Bloomer et al [28] demonstrated higher levels of oxidative stress after consumption of a pure-lipid meal compared to a kilojoule-equivalent pure-sugar meal. Others have reported that high sugar and high fat had an independent and additive effect [18] on the acute rise of plasma inflammatory and oxidative stress markers, although this has not always been observed [29]. Although the effect of postprandial hyperglycemia on endothelial function is beginning to be understood [30], it is not yet clear from these studies whether postprandial oxidative (and/or inflammatory) activity will always increase in the presence of an evocative kilojoule load, irrespective of the nutritional context of the ingested food.

Therefore, the current study was designed to test the hypothesis that ingestion of a high-fat/protein, high-sugar, phytonutrient-reduced food (ice cream) would increase oxidative and inflammatory activity greater than a kilojoule-equivalent meal of a phytonutrient-rich whole food (avocado). Specifically, this study investigated the level of postprandial oxidative stress and inflammation induced by (*a*) ice cream and its sugar (glucose) and fat/protein (dairy cream) components and (*b*) a meal of the whole-food avocado (kilojoule matched to ice cream).

2. Methods and materials

2.1. Participants

Participants were recruited through advertisements placed on the grounds of the Sydney Adventist Hospital, Sydney, Australia. A questionnaire was completed by all potential participants to assess their suitability. Participants were accepted into the study if they had no present or past history of inflammatory diseases, diabetes, cardiovascular disease, cancer, or acute upper respiratory tract infection; were not lactose intolerant; and had no known allergies to the foods tested in the study. Participants were also required to have not taken any anti-inflammatory medications, antioxidants, or vitamin supplements within 48 hours of commencing the study. Eleven healthy participants (5 males and 6 females) between 21 and 78 years of age were selected for inclusion into the study. The means ± SD ages for males and females were 62.4 ± 12.6 and 45.2 ± 14.2 years, respectively. Written informed consent was obtained from all participants. This study was conducted in accordance with the Helsinki Declaration on Ethical Principles in Research, Sixth Revision 2008, and approved by the Sydney Adventist Hospital Human Research Ethics Committee (project approval 2011-039).

2.2. Study design

This study used a randomized, crossover design. Participants were required to consume 4 test meals with a minimum 2-week washout period between each test meal. The 4 foods/food components given to each participant were as follows: (1) 500 g