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Review

Dietary glycemia as a determinant of health and longevity

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ABSTRACT

The role of diet in extending lifespan and healthspan has been the subject of much research and debate. Our recent epidemiological and *in vivo* data suggest that carbohydrate quality can be a major determinant in prolonging eye health. Additionally, excessive carbohydrate intake can contribute to the exacerbation of many different diseases. The metabolic diversity of the tissues that are affected by excessive carbohydrate intake suggests that dietary carbohydrate quality may affect cellular homeostasis.

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1. Introduction and definition of glycemic index

1.1. Introduction

Extending healthful life is a millennia-old dream and objective. During the intervening centuries a multitude of concoctions and remedies have been offered, usually with few substantiated results. During the last century it was demonstrated that limiting caloric intake is associated with extended life in many mammals, albeit results remain to be clarified in humans (Colman et al., 2009; Mattison et al., 2012; McCay et al., 1935; McCay and Crowell, 1934; Walford and Crew, 1989; Walford, 1986; Weindruch, 1984, 1991, 1996). A myriad of modeling studies have revealed signaling pathways that are associated with life extension and the last two decades have seen an interest in the types of dietary carbohydrates that might confer health advantage, and possibly longevity. Loss of vision due to age-related cataracts or age-related macular degeneration is widely prevalent, affecting about 85% and 15% of the elderly respectively. With

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centenarians among the fastest growing segments of societies, and with loss of vision a very costly personal and societal burden, there is keen interest in extending vision – that is, delaying age-related macular degeneration and cataract – or diminishing risk for these debilities. Using extensive epidemiologic and nutritional information from the Nurses' Health Study and Age-Related Eye Disease Study (AREDS) we determined that measures of total carbohydrate, and even more so, glycemic index (GI), are associated with visual health (Chiu et al., 2011; Chiu et al., 2006b, 2007a, 2007b, 2007c; Weikel and Taylor, 2011; Weikel et al., 2012a). We also modeled this relationship in mice in order to elucidate etiologic relationships between dietary glycemia, visual health, and genetics (Rowan et al., 2014; Uchiki et al., 2012).

1.2. Definition of glycemic index

The glycemic index (GI) of a food describes how quickly carbohydrates in our food are converted into blood glucose. GI is defined as the rise in blood glucose within a 2 hour period elicited by a portion of food containing 50 g of available carbohydrate, relative to the rise elicited by 50 g of glucose (see Fig. 1 for example). Dietary glucose or high GI foods increase blood glucose rapidly and lead to a compensatory postprandial hypoglycemia (green and red lines in Fig. 1) whereas a low GI food increases blood glucose to a lower extent (blue line in Fig. 1). Two foods can have identical total carbohydrate content and be isocaloric, but have different GI, based on how quickly the carbohydrates are converted to glucose. Recent studies indicate that the types or quality of carbohydrate foods consumed by an individual have a major impact on health. It has been shown that relative to consuming higher GI diets, consuming lower GI diets is associated with attenuated blood glucose increases, lower postprandial hypoglycemia, lower hyperlipidemia, lower insulinemia, less elevation of inflammatory markers, and less counter regulatory endocrine responses (Jenkins et al., 2002; Ludwig, 2002; Scribner et al., 2008). The typical diet in the United States is high glycemic, suggesting that many chronic diseases in the US may be ameliorated by dietary changes (Chiu et al., 2011).

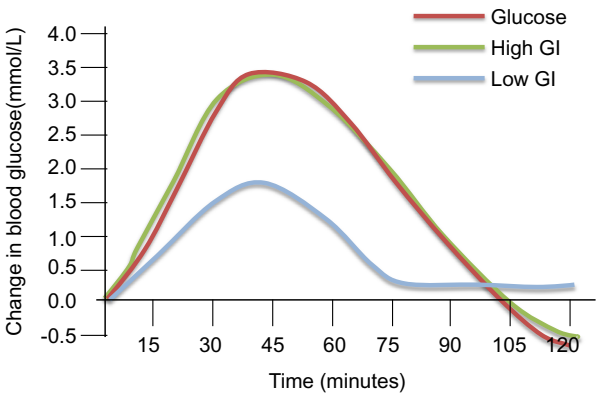

Fig. 1. Changes in blood glucose after consumption of high and low glycemic foods. Change in blood glucose in mmol/L is plotted versus time in minutes.

2. Glycemic index, disease and proteopoise

Throughout this work proteostasis is referred to as proteopoise to indicate that there is a dynamic rather than static relationship between proteins, insults and the cellular machinery that must recognize and selectively remove the damaged proteins.

2.1. Risk of age related macular degeneration (AMD)

Work in our lab has focused on relationships between the risk for age-related eye diseases, primarily age-related macular degeneration (AMD) or cataract and consuming high GI diets. AMD is the leading cause of irreversible blindness in the elderly. There are two types of AMD: "wet AMD" results from neovascularization in the choroid of the retina, while "dry AMD" is correlated with the appearance of drusen, which are white or yellow deposits of extracellular material that accumulate between Bruch's membrane and the retinal pigment epithelium (RPE). Dry AMD is the most prevalent type representing nearly 90% of all AMD cases. While there are several treatments which target the neovascularization in wet AMD, currently, there are no treatments to delay or reverse dry AMD. Both types of AMD result in death of the photoreceptor cells and vision loss. Late stage dry AMD is correlated with the loss of large areas of RPE and photoreceptors known as geographic atrophy.

Our epidemiological data indicate that consumption of high GI diets is associated with increased prevalence and increased progression of AMD (Fig. 2) (Chiu et al., 2006a, 2007a, 2007b); even when diets contain the same amount of total carbohydrate. Persons in the highest quintile of GI intake had increased risks of large drusen, neovascularization and geographic atrophy as compared to the lowest quintile (Chiu et al., 2007a). Work from other groups confirms that high GI diets increase risk of AMD including soft drusen (Kaushik et al., 2008). These studies suggest that changes in diet could diminish the risk of developing AMD and progression of the disease. The risks of AMD associated with smoking (Smith et al., 2001) and genetic variations (Fritsche et al., 2013) are within similar ranges to the risks associated with the consumption of high GI diets (Fig. 2).

Many other age-related diseases are also exacerbated by high GI diets: individuals consuming high GI diets are at increased risk for type II diabetes, cardiovascular disease as well as cataracts (Chiu et al., 2011). Since cataract results from post-synthetic modifications of lens proteins, called crystallins, it would appear that elevated levels of sugars might be involved in cataractogenesis as well. These relationships are intriguing because the lens is an avascular, anoxic environment with what is probably the slowest metabolism in the body. Right next to it is the retina, the most oxygenated and highly vascularized tissue in the body. The metabolic diversity of the tissues that are adversely affected by high GI carbohydrate intake suggests that the relationship between dietary carbohydrate quality and cellular homeostasis is of fundamental physiological importance and, perhaps, that there is a common etiologic link between dietary glycemia and the various diseases.

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