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Death by Sugar: The Impact of Sugar on Acutely Ill Patients

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ABSTRACT

In 2012, according to the United States Centers for Disease Control and Prevention, approximately 78 million Americans were considered obese, with a body mass index > 30. Americans, on average, consume 80 pounds of sugar annually. Sugar is converted through metabolism into advanced glycation end-products, which are linked to worse health outcomes in hospitalized patients. In this article we review the different types of sugar typically consumed by Americans and its deleterious effects on human health.

Keywords: advanced glycation end products, dextrose, fructose, glucose, hyperglycemia, obesity, sucrose

INTRODUCTION

In 2012, according to the United States Centers for Disease Control and Prevention (CDC), approximately 78 million Americans were considered obese, with a body mass index (BMI) > 30.[1] This translates into 35% of the US population, in which there has been about a 10% increase in obesity rates since the year 2000.[2] The epidemic is also affecting children, with approximately 16.9% of those < 18 years old now considered obese.[3] A recent meta-analysis indicated that body weight is correlated with sugar consumption.[4] Americans, on average, consume 80 pounds of sugar each year (compared with the global average of 55 pounds annually). Although reduced from 137 pounds per person, according to the US Department of Agriculture in October 2012, this remains an excessive amount of sugar consumption.[5] Current consumption levels represent an increase of 40 pounds per person since 1900.[6] Sugar consumption is making people fatter and subsequently sicker.

As nurse practitioners, obesity affects the amount of care and types of diseases prevalent in our clinics and our intensive and progressive care units. Persons with obesity have higher rates of heart disease, hypertension, chronic obstructive pulmonary disease, pulmonary embolism while hospitalized, mild cognitive impairment, cardiac death, and an overall increase in risk of death. Obesity is recognized as a state of chronic low-grade inflammation, characterized by elevations in systemic acute phase proteins such as C-reactive protein, interleukin-6, and interleukin-1. Speculation about the cause of obesity centers on caloric intake, lack of exercise, and the biologic aspect that humans have evolved to be efficient machines that can store calories in times of excess. Currently, there is a debate about whether or not humans have a "thrifty" gene, which promotes storage of fat during times of excess, although this discussion is outside the scope of this study. What is known, however, is that, throughout most of human existence, calories have never been as bountiful as they are in today's American society. One of the main forms of available calories that are plentiful like never before is added sugar. Added sugars are those that are inserted into foods during preparation and processing, or simply the sugar that is added by us at the time of consumption, such as sugar added to coffee or cereal. Added sugars have been directly linked to a range of illnesses, such as fatty liver disease, type 2 diabetes, insulin resistance, and metabolic syndrome.[7]

In this review, we specifically examine the link between sugar, obesity, and illness. We also investigate advanced glycation end-products (AGEs) and their impact upon the acutely ill. AGEs are modifications of proteins or lipids that become nonenzymatically glycated and oxidized after contact with monosaccharide sugars. Evidence suggests that these metabolic byproducts are associated with negative patient outcomes.[8] Furthermore, we review the difference between glucose and fructose. In short, human health is significantly linked to sugar consumption. We postulate that there may be a direct correlation between sugar consumption and the delivery of sugar to critically ill patients and undesirable clinical outcomes.

PHYSIOLOGY

The most common form of sugar that humans consume is sucrose, which is a disaccharide molecule that is broken down by sucrose enzymes into glucose and fructose. Glucose and fructose are considered monosaccharides, or simple sugars. Dextrose, the form of sugar often found in intravenous solutions, is a form of glucose and, within the bloodstream, acts the same way as glucose. It is the components of glucose and fructose that have been significantly associated with human health. American's derive most of their consumed sugar calories in the form of sucrose that is processed from sugar cane and sugar beets. In general, fructose, the sugar that occurs naturally in fruits and vegetables, comprises about 10% of the total sugar consumed by Americans daily, which includes the fructose broken down from sucrose as well as fructose directly consumed from fruit and vegetable consumption. Most of the extra calories that Americans consume are from added sugar and sugar-sweetened beverages, which are sweetened from sugar cane and sugar beets.

Upon consumption, the body breaks carbohydrates into their respective monosaccharaides. The only form of sugar that the brain utilizes is glucose and therefore the body is programmed to maintain a relatively constant level of glucose to maintain cerebral functioning. Dietary glucose is stored in the liver and muscle cells in polysaccharides known as glycogen. For the body to harness energy from glucose, it must go through glycolysis, which is where it is broken down into pyruvate for the production of adenosine triphosphate. Fructose does not need to go through many of the steps of glycolysis for availability for energy that glucose does, making it a quicker producer of energy after consumption.[7] However, although both fructose and glucose converge at a single point for energy production, there are significant differences in the way the body responds to each of these sugar molecules. A recent study indicated glucose causes a reduction in cerebral blood flow to the hypothalamus compared with fructose.[9] High levels of fructose cause an increase in circulating triglycerides compared with glucose.[7] However, fructose consumption decreases 2-hour postprandial glucose levels,[10] whereas glucose causes an increase in circulating insulin and does not affect circulating cholesterol.[11]

THE DOWNSIDE OF GLUCOSE

Long-term chronic elevation of glucose has been associated with diseases of the endothelium such as diabetes, coronary artery disease, and stroke. In addition, the same vascular changes appear to affect brain function and overall cognition. There has been a significant push within the critical care arena to keep glucose levels tightly controlled to promote healing and decrease negative outcomes, especially in acutely ill postsurgical patients. High blood glucose levels have been associated with morbidity, multiorgan dysfunction, and poor outcomes in acutely ill patients, regardless of the associated disease physiology.[12] However, the debate between hyperglycemia and outcomes is still debated by practitioners. Nonetheless, it is presumed that keeping glucose levels under control protects the cellular mitochondria and prevents cytopathic hypoxia. The term cytopathic hypoxia refers to the cellular energy and metabolic disturbances that are a hallmark of critical illness in hyperglycemic conditions. Through tight glycemic control, inflammatory biomarkers are reduced and patient outcomes are improved.[13] The discussion regarding tight glycemic control and negative outcomes associated with hypoglycemia is beyond the scope of this review.

OBESITY

In addition to elevated inflammatory markers accompanying elevated glucose levels, patients who are obese generally have higher levels of circulating pro-inflammatory biomarkers. Loss of adipose tissue is directly correlated with a reduction in circulating