Short Communication

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ARCHIVES OF MEDICINE
ISSN 1989-5216

2017
Vol.9 No.6:4

DOI: 10.21767/1989-5216.1000246

The Secret Linkage Amid Sugar and Heart Disease: Is Sugar Truly Foe Rather than Just Friend?

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Received date: November 17, 2017; **Accepted date:** November 27, 2017; **Published date:** December 02, 2017

Citation: Uddin MS, Ahmed M (2017). The Secret Linkage Amid Sugar and Heart Disease: Is Sugar Truly Foe Rather than Just Friend? Arch Med Vol No:9 Iss No:6:4

Keywords: Sugar; Heart disease; Coronary heart disease; Cardiovascular disease

Short Communication

Sugar is the crucial cradle of sweetness that embodies a prompt energy boost. It is something that we intake relentlessly and toils to precisely estimate sugar content in diet and food stuff because of its rife distribution [1]. Numerous studies advocated the occurrence of high amounts of added sugars up to 75% of all foods and beverages [2,3]. Owing to the arrival of modern food-processing methods, nowadays added sugar is a significant factor of the human diet. The endorsement for the intake of added sugar is not fixed yet it differs substantively. The World Health Organization praises that added sugar is accountable for less than 10% of total calories and as per Institute of Medicine it is less than 25% [4,5].

The American Heart Association indorses to limit the intake for added sugars to less than 150 and 100 calories for men and women per day respectively [6]. Abundant studies recommended that, high sugar intake is a universal delinquent and associated with excess weight [7], increase the risk of obesity [8], Type 2 diabetes mellitus [9], dyslipidemias [10], hypertension [11] and cardiovascular disease (CVD) [12], etc. But, numerous dominant appraisals and studies suggested sugar as an innocent. In 1967, McGandy et al. published 2 parts review article entitled "Dietary Fats, Carbohydrates and Atherosclerotic Disease," in the New England Journal of Medicine (NEJM) based on synopses of epidemiologic, experimental and mechanistic studies [13,14]. In the epidemiologic studies, researchers affirmed that high sucrose intake is allied with coronary heart disease (CHD) [13,14]. The experimental studies stated sucrose as causative factor for the rise in serum cholesterol and serum triglyceride levels amid typical people [13]. Furthermore, a surge in triglyceride levels was also reported amid hypertriglyceridemia people [14]. The mechanistic studies conveyed that sucrose prompting the level of serum cholesterol by vicissitudes of the intestinal microbiome and fructose swaying the level of serum cholesterol by lipogenesis in organs especially liver and adipose tissues [14]. Kearns et al. as per the analysis of internal documents of Sugar Research Foundation (SRF) published an article in JAMA Internal Medicine in 2016 [15]. This study suggests that the sugar industry funded its first coronary heart disease inquiry project in 1965 to abate primary warning signals that sucrose intake was a jeopardy aspect in coronary heart disease [15]. In this study, researchers affirmed that 1967 reviews were probably skewed **(Figure 1)** [15]. This review of McGandy et al. engrossed on promising prejudice in the epidemiologic, experimental and mechanistic studies. It abated epidemiologic proof owing to multifactorial puzzling in case of recognizing dietary reasons of coronary heart disease [13]. For experimental studies, only interim researches with gigantic doses of sucrose were reflected, because they were not analogous with quantities usually intake in the American diet [13]. In case of mechanistic studies, they abated researches escorted with only fructose or glucose, not for sucrose (i.e. glucose + fructose) [14].

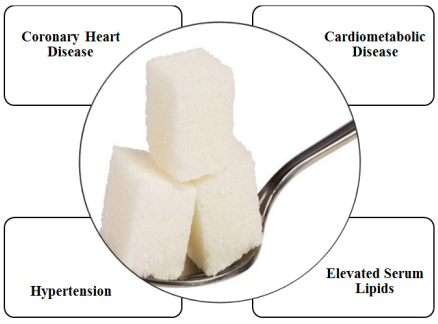

Figure 1: Sugar is the crucial culprit for numerous heart diseases, not fat. Eating too much added sugar surges the peril of defunct with heart disease.

A number of studies scrutinized the linkage amid sugar intake and heart disease (HD). Jacobs et al. studied 34,492 postmenopausal women aged 55-69 years examined for 9 years [16]. They showed no kin concerning the intake of sweets or desserts and peril of ischemic heart disease. But the key inadequacy of this

study was foremost sources of sugar (i.e. soft drinks) were not considered [16]. In another study by Bolton-Smith et al. found that neither extrinsic (i.e. added) sugar, intrinsic (i.e. naturally occurring) sugar, nor the fat to sugar ratio are marked independent forecasters of prevalent of coronary heart disease [17]. This study was conducted among 10,359 men and women aged 40-59 years. The researchers clinched that total sugar intake is not an utmost causal marker of coronary heart disease [17]. But in this study data were not accustomed for other dietary parables [17].

Cholesterol is a natural metabolite performing a number of functions [18]. They carried in the blood attached to proteins by 2 main forms, LDL (i.e. low density lipoprotein) and HDL (i.e. high density lipoprotein) cholesterol [18]. LDL cholesterol is referred to as "bad cholesterol" owing to atherosclerosis and related complications as a result of low density. HDL is often referred to as “good cholesterol” owing to its compact denseness [18,19]. Numerous studies have revealed an opposite linkage amid dietary sucrose and HDL cholesterol. The study of the Coronary Artery Risk Development In young Adults (CARDIA) by Archer et al. reported reverse affairs amid augmented dietary sucrose intake and HDL cholesterol levels in both cross-sectional and longitudinal studies as well as in diverse race and gender sets [20]. The researchers recommended that dropping dietary sucrose intake may be favorable for people with low HDL-C [20]. Sucrose enrich diet (i.e. 20% of energy) is linked with a raise in the level of plasma triglyceride owing to augmented hepatic discharge and diminished in clearance of very LDL (VLDL) cholesterol [21,22]. Total cholesterol, truly gives us slight beneficial facts, because both good and bad factors are associated with it. One might (i.e. mistakenly) think that diminishing dietary cholesterol levels may efficient to moderate blood cholesterol levels. Though, 80% of the cholesterol in our blood is produced by the liver, therefore decreasing dietary cholesterol is fairly abortive [23].

Stanhope et al. investigated the effect of ingesting high-fructose corn syrup (HFCS) sweetened beverages on lipid/lipoprotein in risk factors for CVD amid young adults [24]. Consuming beverages containing 10%, 17.5%, or 25% energy requirements from HFCS for 2 weeks exerted dose-related upsurges in circulating lipid/lipoprotein and uric acid. The denouements suggested mechanistic buttress for the epidemiologic proof that the risk of CVD is absolutely allied with intake of high added sugars [24].

In the review of McGandy et al. suggested the consequence of sucrose and fat interferences on serum cholesterol and triglyceride consistent with the human randomized clinical trials (RCTs). In case of fat interferences, it indicted surrogating polyunsaturated fat for saturated fat instigated an enormous increase in the level of serum cholesterol amid typical people [13,15]. The same denouements also claimed for decreasing dietary cholesterol [13,15]. In case of sucrose interferences, it appealed that surrogating fat for sucrose; starch for sucrose and leguminous vegetables for sucrose triggered a great progress in the levels of serum triglyceride amid typical, hypertriglyceridemia and typical people respectively [13-15]. Furthermore, surrogating starch for sucrose instigated for minor upgrading in the levels of serum cholesterol amid typical people [13,15]. In this area, researchers also belied, they only reflected these studies that had used the level of serum cholesterol as a biomarker of coronary heart disease [15]. For sucrose interventions, they abated that surrogating starch for sucrose had a huge outcome for the elevation of levels of serum triglyceride [13-15]. They also abated RCTs had exposed that surrogating fat or vegetables for sucrose had a big impact on increasing the level of serum cholesterol [13,15]. Not only that, the effect of surrogating refined starches (i.e. sweetened with artificial sweeteners) for sucrose was sacked owing to insignificant greatness on the level of serum cholesterol with respect to the level of reducing dietary cholesterol and surrogating polyunsaturated fat for saturated fat [13,15].

Te Morenga et al. inspected the possessions of the alteration of dietary free sugars on blood pressure as well as lipids based on systematic review and meta-analyses of RCTs [25]. They suggested that high intake of sugar is linked to hypertension and elevated serum lipids [25]. In a study DiNicolantonio et al. suggested that not salt but sugar as an aetiological factor in hypertension and cardiometabolic disease [26]. They advocated abating the intake of processed foods (i.e. prime source of added sugars, mostly fructose) so as to subside the incidence of hypertension [26].

For decades, physicians have suggested decreasing the ingestion of saturated fat to prevent CVD. In fact, still now numerous physicians still consider that a low-fat diet is the greatest food choice to reduce the chance of CVD [27]. But the real leitmotif is not like that. But some people believe that vegetarians appear to have the low risk of CVD. But its travail to prove that it’s owing to an intake of less fat and cholesterol, because vegetarians eat less sugar as well. Is not it? de Souza et al. reported that saturated fats intake are not connected with CVD, coronary heart disease, and ischemic stroke [28]. But a positive linkage was reported for trans fats intake and coronary heart disease and coronary heart disease mortality [28]. In the North American diet, saturated fats are accountable for about 10% of energy but for trans fats it’s only about 1-2% [29,30]. Animal products (i.e. butter, cows’ milk, meat, salmon and egg yolks) and some plant products (i.e., chocolate, cocoa butter, coconut and palm kernel oils) are the main sources of saturated fatty acids in the food stuff [28]. Conversely, trans fats are generated industrially [28]. In a study Bendsen et al. advocated that industrially produced trans fats might increase the risk of coronary heart disease [31]. Yang et al. consistent with the data of the National Health and Nutrition Examination Survey (NHANES) published an article in JAMA Intern Med in 2014 [32]. In this study, they observed a momentous link amid added sugar intake and augmented risk for CVD mortality [32].

The special communication of Kearns et al. suggested that the study of McGandy et al. was to blame fat, instead of sugar, for great rates of coronary heart disease [15]. Kearns et al., mentioned that the SRF, in the beginning offered $500 (i.e. $3800 in 2016 dollars) to Hegsted (i.e., second author) and $1000 (i.e. $7500 in 2016 dollars) to McGandy (i.e. first author), “half to be paid when they start work on the project and the remainder when they inform that the article (i.e. Dietary Fats,

Carbohydrates and Atherosclerotic Disease) has been accepted for publication [15].” Eventually, the SRF would pay them $6500 (i.e. $48 900 in 2016 dollars) for the 2 parts review article based on the numerous articles which find some special metabolic risk in sucrose and in particular, fructose [15]. Moreover, owing to disputes NEJM began requiring authors to disclose all conflicts of interest in 1984 [33].

Today numerous studies say sugar does exert a role in heart disease. Cutting the intake of saturated fat doesn't ineludibly lessen the risk of heart disease. The foremost drive of the appraisal of McGandy et al. was to introduce sucrose as a pal and fat as a nemesis for coronary heart disease. Yet coronary heart disease risk is paradoxically cited as a health consequence of added sugars intake, because the industry may have a long history of swaying central policy. Policymaking teams should cogitate giving less priority to food industry funded studies. The research and development is the life blood of a nation. Researcher must be vigilant and must conduct researches in the absenteeism of any profitable or monetary dealings that could be interpreted as a latent conflict of interest.

Acknowledgement

The authors wish to thank the anonymous reviewer(s)/ editor(s) of this article for their constructive reviews.

Conflict of Interests

The authors proclaim no conflict of interest.

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