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Review

Association of fructose consumption and components of metabolic syndrome in human studies: A systematic review and meta-analysis

Roya Kelishadi M.D. [a], Marjan Mansourian Ph.D. [a,b], Motahar Heidari-Beni Ph.D. Cand. [c,*]

[a] Child Growth and Development Research Center, Isfahan University of Medical Sciences, Isfahan, Iran
[b] School of Health, Isfahan University of Medical Sciences, Isfahan, Iran
[c] Food Security Research Center and Department of Community Nutrition, School of Nutrition & Food Science, Isfahan University of Medical Sciences, Isfahan, Iran

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ABSTRACT

Objective: The aim of this study was to review the current corpus of human studies to determine the association of various doses and durations of fructose consumption on metabolic syndrome.
Methods: We searched human studies in PubMed, Scopus, Ovid, ISI Web of Science, Cochrane library, and Google Scholar databases. We searched for the following keywords in each paper: *metabolic syndrome x, insulin resistance, blood glucose, blood sugar, fasting blood sugar, triglycerides, lipoproteins, HDL, cholesterol, LDL, blood pressure, mean arterial pressure, systolic blood pressure, diastolic blood pressure, hypertens*, waist circumference, and fructose, sucrose, high-fructose corn syrup, or sugar.*
Results: Overall, 3102 articles were gathered. We excluded studies on natural fructose content of foods, non-clinical trials, and trials in which fructose was recommended exclusively as sucrose or high-fructose corn syrup. Overall, 3069 articles were excluded. After review by independent reviewers, 15 studies were included in the meta-analysis. Fructose consumption was positively associated with increased fasting blood sugar (FBS; summary mean difference, 0.307; 95% confidence interval [CI], 0.149–0.465; $P = 0.002$), elevated triglycerides (TG; 0.275; 95% CI, 0.014–0.408; $P = 0.002$); and elevated systolic blood pressure (SBP; 0.297; 95% CI, 0.144–0.451; $P = 0.002$). The corresponding figure was inverse for high-density lipoprotein (HDL) cholesterol (−0.267; 95% CI, −0.406 to −0.128; $P = 0.001$). Significant heterogeneity existed between studies, except for FBS. After excluding studies that led to the highest effect on the heterogeneity test, the association between fructose consumption and TG, SBP, and HDL became non-significant. The results did not show any evidence of publication bias. No missing studies were identified with the trim-and-fill method.
Conclusion: Fructose consumption from industrialized foods has significant effects on most components of metabolic syndrome.

Introduction

Fructose is a monosaccharide that naturally exists in fruits, honey, and some vegetables. These days, however, fructose is mostly ingested from industrial and commercial products such as soft drinks, sweetened beverages, and high-fructose corn syrup (HFCS) [1,2]. Natural foods that contain fructose, such as fruits and vegetables, only contain small amounts; moreover their fructose is absorbed slowly. Thus, after consuming these foods, the rise in serum fructose concentration is negligible [3]. The use of HFCS in beverages and soft drinks is increasing by food manufacturers because of its low cost compared with sucrose, and the ease with which it can be added to food products [4]. Actually, the consumption of fructose through manufactured products is concerning. The average daily intake of total fructose in the United Stated increased 12 g/d between 1978 (37 g/d) and 2004 (49 g/d) [5,6].

Fructose is more lipogenic than other carbohydrates, and unlike glucose it can be converted to glycerol-3-phosphate (required for tri-acylglycerol synthesis) without passing from the phosphofructokinase pathway, which is an important rate-limiting pathway of glycolysis. Therefore, high-fructose consumption can be the cause of triglyceride (TG) synthesis from

* Corresponding author: Tel.: +98 311 7923060; fax: +98 311 6682509.
E-mail address: heidari.motahar@gmail.com (M. Heidari-Beni).

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Table 1
Search strategy for PubMed, Scopus, Ovid, ISI Web of Sciences, Cochrane library and Google Scholar databases

No.	Search terms
1	"Metabolic syndrome x" [Mesh] OR "insulin resistance" [Mesh] OR "blood glucose" [Mesh] OR "blood sugar"[tiab] OR "FBS" [tiab] OR "Triglycerides" [Mesh] OR "Lipoproteins, HDL" [Mesh] OR "Cholesterol, HDL" [Mesh] OR "Lipoproteins, LDL" [Mesh] OR "Cholesterol, LDL" [Mesh] OR "Blood Pressure" [Mesh] OR "mean arterial pressure" [tiab] OR "BP" [tiab] OR "SBP" [tiab] OR "DBP" [tiab] OR "hypertens*"[tiab] OR "Waist Circumference"[Mesh]
2	"Fructose" [Mesh] OR "Sucrose" [Mesh] OR "high fructose corn syrup" [tiab] OR "sugar"[tiab]
3	1 AND 2

unchecked pathways [7]. According to animal studies, consumption of a 60% total energy diet with fructose can induce obesity and some components of metabolic syndrome (MetS) such as insulin resistance, dyslipidemia, and hypertension [8–10].

The escalating trend in the prevalence of chronic diseases in industrialized and developing countries [11] and the increase in fructose consumption in the population's diet is of concern. Researchers have proposed an association between fructose consumption and chronic diseases [12]. Therefore, many experimental and human studies are being conducted to assess the relationship of fructose consumption with the development of chronic diseases such as diabetes and MetS [4].

Studies of young healthy individuals have shown that fructose consumption (250 g/d) compared with the same amount of glucose significantly decreased insulin sensitivity [7]. There are fewer studies on the long-term effects of fructose consumption on human health than on the effects of glucose [3]. The adverse short-term effects of fructose seem to be dose-dependent. Short-term consumption of fructose in humans did not have adverse effects on health status, unless consumed in excessive amounts. Long-term consumption of fructose is associated with an increase in adiposity, dyslipidemia, and insulin resistance [3,7].

Findings about fructose and health status are controversial. Some studies supported the hypotheses that fructose consumption leads to an increase in chronic disorders such as MetS [13–16], whereas others did not confirm the positive association of fructose with the components of the disease [17–20]. Some reasons may be suggested for the differences between the findings of various studies. Low fructose dose, for instance < 20 g/d, mostly showed improved or no effect on MetS parameters; however, high doses of fructose mostly increased the features of MetS [16,20]. Different forms of fructose such as natural fructose, fructose alone, or fructose bonded with glucose in the form of sucrose, showed various findings [3]. Race, study design, animal or human studies, and different characteristics of participants such as sex, age, and body weight are other factors that influence differences in the findings obtained in various studies.

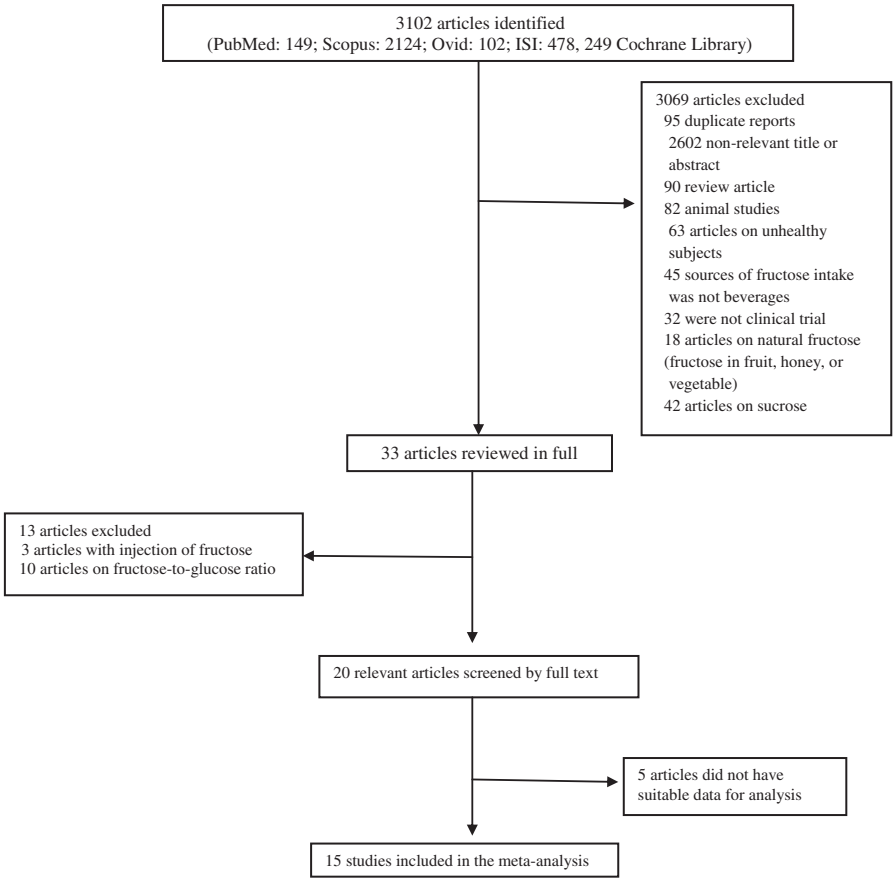

Fig. 1. Flowchart of the literature search.

Table 2
Summary of clinical trials on the association of fructose and components of metabolic syndrome in human studies

Reference	Population	Sex	Age (y)	Duration of study	Fructose form and dose	Results
Stanhope et al., 2009 [35]	32 overweight and obese participants	Male and female	52.5 ± 8.8	10 wk	Fructose-sweetened beverages provided 25% of energy requirements	Fructose promoted dyslipidemia, increased FBS and insulin levels, and decreased insulin sensitivity
Swarbrick et al., 2008 [40]	7 overweight or obese postmenopausal participants	Female	64 ± 7.9	10 wk	Fructose-sweetened beverages provided 25% of energy requirements	Fructose increased postprandial TG and fasting apoB concentrations; had no significant effects on fasting total cholesterol, LDL, HDL, TG, or IR
Perez-Pozo et al., 2010 [16]	74 healthy adults	Male	51 ± 7.8	2 wk	Fructose-sweetened beverages, 200 g/d	Fructose led to increased SBP, DBP, fasting TG, fasting insulin, and HOMA indices and decreased HDL cholesterol significantly
Aeberli et al., 2011 [31]	29 healthy, normal-weight (I)	Male	26.3 ± 6.6	3 wk	Fructose-sweetened beverages, 600 mL beverage containing 40 g fructose	FBS increased significantly; SBP, DBP, and postprandial glucose did not change significantly
	29 healthy, normal-weight (II)	Male	26.3 ± 6.6	3 wk	Fructose-sweetened beverages, 600 mL beverage containing 80 g fructose	FBS increased significantly; LDL decreased; SBP, DBP, and postprandial glucose did not change significantly
Hallfrisch et al., 1983 [45]	12 healthy, normal-weight	Male	39.8 ± 2.4	5 wk	15% of calories as fructose	Fructose significantly increased total cholesterol and LDL; did not affect TG level, SBP, or DBP
Silbernagel et al., 2011 [46]	20 healthy, normal-weight	Male and female	32.9 ± 10.5	4 wk	150 g of fructose dissolved in 250 cc water	Plasma NEFA, total cholesterol, LDL cholesterol; HDL cholesterol did not change; insulin sensitivity decreased; TG significantly increased
Le et al., 2006 [32]	7 healthy, normal weight	Male	24.7 ± 1.3	4 weeks	1.5 g fructose/kg BW daily (20% solution)	Fructose let to significant increase in fasting plasma levels of TG, VLDL, lactate, and glucose without causing IR
Teff et al., 2009 [15]	17 obese participants	Male and female	27 ± 2	2 d	Fructose-sweetened beverages (15% solution) provided 30% of energy requirements	Fructose led to less insulin secretion and increased postprandial TG level
Couchepin et al., 2008 [33]	16 healthy, normal weight	Male and female	22.5 ± 0.93	6 d	3.5 g fructose/kg FFM daily (25% of energy requirements)	Fructose significantly increased FBS, insulin, and TG levels.
Sock et al., 2010 [47]	11 healthy, normal weight	Male	24.6 ± 0.6	7 d	3.5 g fructose/kg FFM daily (35% of energy requirements)	Fructose increased VLDL, fasting hepatic glucose output; did not change fasting glycemia, insulin
Crapo et al., 1984 [34]	11 healthy, normal weight	Male and female	40.4 ± 12.4	2 wk	Fructose provided 24% of energy requirements (63–99 g/d)	Postprandial glucose and insulin levels increased slightly; TG level did not change
Bantle et al., 2000 [48]	24 healthy, normal weight	Male and female	41.2 ± 14.6	6 wk	Crystalline fructose (14% of energy came from added fructose)	Fructose increased TG in men but had no significant effect on women; did not change fasting plasma cholesterol, HDL, or LDL in men or women
Bossetti et al., 1984 [49]	8 healthy, normal weight	Male and female	27.7 ± 3	14 d	Crystalline fructose (range 50–107 g/d)	Fasting glucose, insulin levels, TG, total cholesterol, LDL, and HDL did not change
Stanhope et al., 2011 [50]	48 healthy participants	Male and female	28.0 ± 6.8	2 wk	Fructose-sweetened beverages provided 25% of energy requirements	24-h TG area under the curve increased; fasting LDL and apoB concentrations increased; HDL level did not change
Beck-Nielsen et al., 1980 [51]	17 healthy, normal weight	Male and female	28 ± 7	7 d	250 g fructose dissolved in water	Fructose reduced insulin sensitivity; FBS did not change
Stanhope et al., 2008 [26]	34 healthy participants	Male and female	34.7 ± 1.7	2 d	Fructose-sweetened beverages provided 25% of energy requirements	There were no significant changes in body weight and plasma concentrations of glucose, insulin, or TG

(*Continued on next page*)

Table 2 (*Continued*)

Reference	Population	Sex	Age (y)	Duration of study	Fructose form and dose	Results
Cox et al., 2012 [27]	31 healthy participants	Male and female	53.7 ± 3	10 wk	Fructose-sweetened beverages provided 25% of energy requirements	Fructose consumption developed risk factors of metabolic syndrome, such as accumulation of intraabdominal fat, dyslipidemia, and IR
Teff et al., 2004 [28]	12 normal-weight participants	Female	25 ± 2	2 d	Fructose-sweetened beverages provided 30% of energy requirements	Dietary fructose reduced circulating insulin and increased TG
Abdel-Sayed et al., 2008 [29]	6 healthy participants	Male	24.7 ± 3.1	7 d	Fructose-sweetened beverages (3 g fructose/kg BW daily)	Fructose increased fasting plasma TG concentrations significantly; inhibited lipolysis and lipid oxidation
Faeh et al., 2005 [30]	7 healthy, normal participants	Male	26.5 ± 4	6 d	20% fructose solution (3 g/kg daily, 25% of total calories)	Fructose significantly increased plasma TG concentration, fasting glycemia, *de novo* lipogenesis, and IR

BW, body weight; DBP, diastolic blood pressure; FBS, fasting blood sugar; FFM, fat-free mass; HDL, high-density lipoprotein; HOMA, homeostatic model assessment; IR, insulin resistance; LDL, low-density lipoprotein; SBP, systolic blood pressure; NEFA, non-esterified fatty acids; TG, triglyceride; VLDL, very low-density lipoprotein

To help clinicians and researchers make decisions when treating their patients, it is necessary to summarize the controversial findings of various studies concerning the health consequences of fructose consumption.

This study aims to review the findings of human studies on the association of fructose consumption with various doses and different duration with MetS.

Methods

Literature search

The search was conducted using PubMed, Scopus, Ovid, ISI Web of Science, Cochrane library, and Google Scholar databases from January to May 2013. Keywords such as *metabolic syndrome x, insulin resistance, blood glucose, blood sugar, fasting blood sugar, triglycerides, lipoproteins, HDL, cholesterol, LDL, blood pressure, mean arterial pressure, systolic blood pressure, diastolic blood pressure, hypertens*, waist circumference, and fructose, sucrose, high-fructose corn syrup, or sugar* were used. Keywords and Medical subject heading (MeSH) terms are presented in Table 1. The search was refined to the English language. We included human studies and clinical intervention trials that investigated the association of oral fructose on the components of MetS in a healthy population. We did not include animal studies, non-clinical trials, or trials in which fructose was recommended exclusively as sucrose or HFCS because these did not permit us to isolate the effect of fructose. Furthermore, studies that investigated the effect of fructose that is naturally present in fruits, honey, and some vegetables were excluded. The process of selecting studies was showed in Figure 1.

Titles and abstracts of papers were screened and relevant papers were selected. Then, full texts of relevant papers were read and findings were rescreened. Two independent reviewers (MM and MHB) screened titles and abstracts of papers identified by the literature searches for their potential relevance or assessed the full text for inclusion in the review. Two reviewers abstracted the data independently, including data on first author's last name, year of publication and country of the study population, the study name, study design characteristics, study population, the intervention, outcome measures used, and appropriate statistics, and in the case of disagreement, the discrepancy was resolved in consultation with a third arbitrating investigator (RK). Summary of clinical trials on the association of fructose and components of metabolic syndrome in human studies were shown in Table 2.

Statistical analysis

The groups before and after consuming fructose based on MetS components were compared. Measures of association were used for the meta-analysis: means and SD before and after consuming fructose in all clinical trial studies. We produced forest plots to assess the multivariate adjusted mean differences and corresponding 95% CIs visually across studies. We used the fixed-effects model for analyses. When heterogeneity existed ($I^2 > 50\%$), we used and compared both random-effect and fixed-effects models. The summary mean difference estimates from random-effect models were used to consider between-study variability, because the tests for heterogeneity were statistically significant in all analyses. Statistical heterogeneity of the statistics between studies was evaluated with Cochran's Q test and quantified with the I^2 statistic [21]. According to the texts, $I^2 > 50\%$ indicated substantial heterogeneity [22]. To identify sources of heterogeneity, sensitivity analysis was done by successively removing a particular study or group of studies (if any) that had the highest impact on the heterogeneity test. Publication bias was assessed by visual inspection of the funnel plot [23]. In these funnel plots; the mean differences were plotted against the inverse of the square of the standard error (a measure of precision). Asymmetry of the funnel plots was assessed formally with Egger's regression asymmetry tests and adjusted rank-correlation tests [24]. Additionally, Begg's adjusted rank-correlation test and the trim-and-fill method were used [24,25]. All statistical analyses were done with Comprehensive meta-analysis version 2 software. All *P*-values were two-sided with a significance level < 0.05.

Data extraction

Data of articles that investigated the effect of fructose beverage consumption on components of MetS among a healthy adult population were interred to meta-analysis. Mean ± SD of systolic blood pressure (SBP), fasting blood sugar (FBS), high-density lipoprotein cholesterol (HDL-C), and TG before and after fructose consumption were extracted. Some studies expressed changes of MetS components after fructose consumption as changes in area under curves (AUC), another did not reported amount of baseline of these components and some data were unable to be extracted [26–30]. We excluded these studies for meta-analysis. One study [31] used different concentrations of fructose as moderate (40 g/d) and

high (80 g/d) and compared the effect of these concentrations on MetS components. Results of each concentration were interred to meta-analysis separately.

Results

Fructose consumption and FBS

Individual study results and the overall summary results for the 10 clinical trials investigating the effects of fructose consumption on FBS are shown in Figure 2. When all 10 studies were analyzed, consumption of fructose was positively associated with an increase in FBS (summary mean difference, 0.307; 95% CI, 0.149–0.465; $P = 0.002$). There was no statistically significant heterogeneity among studies ($I^2 = 43.49\%$; $P_{heterogeneity} = 0.075$). The results for random-effect model were 0.357 (95% CI, 0.079–0.63; $P = 0.012$). Two clinical trials [32,33] contributed the most to heterogeneity. In an analysis excluding these studies, the association between fructose consumption and FBS became slightly weaker (mean difference, 0.285; 95% CI, 0.126–0.444; $P = 0.001$), and the test for heterogeneity was not statistically significant ($I^2 = 11.7\%$; $P_{heterogeneity} = 0.335$).

Fructose consumption and HDL-C

Individual study results and the overall summary results for the nine clinical trials that investigated the effects of fructose consumption on HDL-C are shown in Figure 3. When all nine studies were analyzed, consumption of fructose was inversely associated with an increase in HDL (summary mean difference, −0.267; 95% CI, −0.406 to −0.128; $P = 0.001$). There was statistically significant heterogeneity among the studies ($I^2 = 71.34\%$; $P_{heterogeneity} = 0.002$). The results for random-effect model were −0.224 (95% CI, −0.514 to 0.065; $P = 0.128$). Two clinical trial studies [16,34] contributed the most to heterogeneity. In an analysis excluding these studies, the association between fructose consumption and HDL became non-significant (mean difference, −0.074; 95% CI, −0.244 to 0.097; $P = 0.399$), and the test for heterogeneity was not statistically significant ($I^2 = 20\%$; $P_{heterogeneity} = 0.455$).

Fructose consumption and SBP

Individual study results and the overall summary results for the seven clinical trials investigating the effects of fructose consumption on SBP are shown in Figure 4. When all seven studies were analyzed, fructose consumption was positively associated with increased SBP (summary mean difference, 0.297; 95% CI, 0.144–0.451; $P = 0.002$). There was statistically significant heterogeneity among studies ($I^2 = 83.36\%$; $P_{heterogeneity} = 0.001$). The results for the random-effect model were 0.170 (95% CI, −0.224 to 0.564; $P = 0.398$). The clinical trial [16] contributed the most to heterogeneity. In an analysis excluding this study, the association between fructose consumption and SBP became non-significant (mean difference, 0.008; 95% CI, −0.192 to 0.176; $P = 0.932$) and the test for heterogeneity was not statistically significant ($I^2 = 19\%$; $P_{heterogeneity} = 0.961$).

Fructose consumption and levels of TG

Individual study results and the overall summary results for the 13 clinical trials investigating the effects of the consumption of fructose on TG are shown in Figure 5. When all 13 studies were analyzed, fructose consumption was positively associated with increased TG (summary mean difference, 0.275; 95% CI, 0.014–0.408; $P = 0.002$). There was statistically significant heterogeneity among studies ($I^2 = 77.42\%$; $P_{heterogeneity} = 0.002$). The results for random-effect model were 0.182 (95% CI, −0.139 to 0.504; $P = 0.267$). The clinical trials [16,32–34] contributed the most to the heterogeneity. In an analysis excluding these studies, the association between consumption of fructose and TG became non-significant (mean difference, 0.182; 95% CI, −0.139 to 0.504; $P = 0.267$) and the test for heterogeneity was not statistically significant ($I^2 = 40\%$; $P_{heterogeneity} = 0.079$).

Assessment of publication bias

Begg's funnel plot and Egger's test were conducted to assess the publication bias of the literature. The shape of funnel plots did not reveal any evidence of funnel plot asymmetry. Egger's test further provided statistical evidence of funnel plot symmetry (fructose consumption and SBP: $P = 0.38$; fructose consumption and HDL: $P = 0.80$; fructose consumption and TG: $P = 0.20$; fructose consumption and FBS: $P = 0.21$). The results did not show any evidence of publication bias. No missing studies were identified with the trim-and-fill method.

Discussion

This meta-analysis showed that fructose consumption has significant adverse effects on FBS. Concerning other components

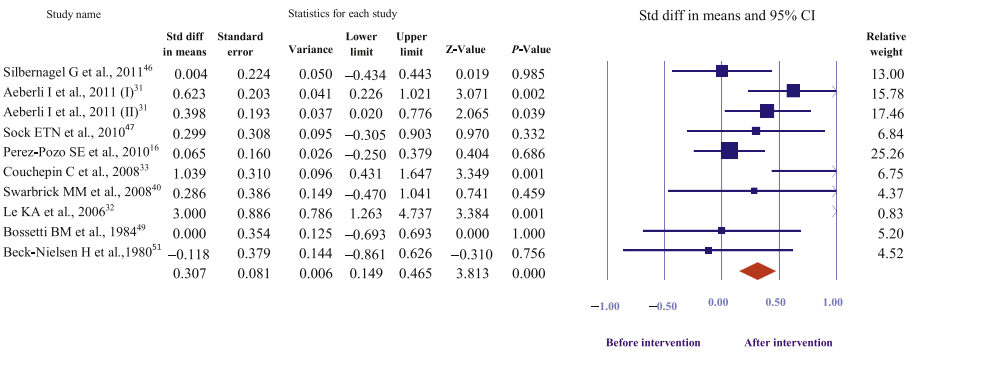

Fig. 2. Meta-analysis of the effect of fructose consumption on fasting blood sugar in published clinical trials. The sizes of the data set indicate the weight of each study in the analysis.

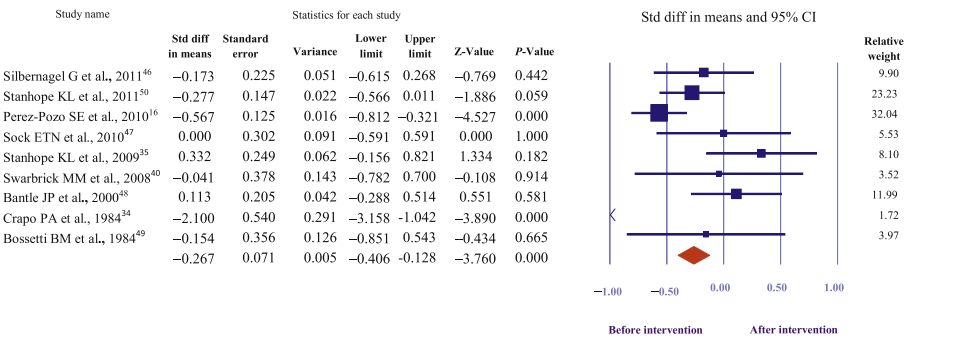

Fig. 3. Meta-analysis of the effect of fructose consumption on HDL-cholesterol in published clinical trials. The sizes of the data set indicate the weight of each study in the analysis.

of MetS, fructose consumption increased levels of TG and SBP and decreased levels of HDL-C. However, after excluding studies that led to heterogeneity, these adverse effects were not statistically significant. The present study is in agreement with some human and animal studies. Human studies indicated that fructose developed some of the features of MetS as hyper-triglyceridemia, insulin resistance, and abdominal obesity [16,27,30,32,35]. Some animal studies showed a high-fructose diet was associated with hyper-triglyceridemia, impaired glucose tolerance, hyperinsulinemia, insulin resistance, and an increase in blood pressure and body weight [36–39]. These adverse effects were not indicated with equivalent calories of glucose. One study [39] investigated the effects of acute and chronic intake of fructose, finding that both types of fructose intake increased some risks for MetS such as elevated SBP, FBS, and TG concentrations. Some studies reported that 25% or 30% of daily energy requirements that were provided by fructose-sweetened beverages increased TG concentrations [15,28,29,32], FBS, insulin resistance [30,35], apolipoprotein B [40], SBP, and diastolic blood pressure (DBP) [16], and inhibited lipolysis and lipid oxidation [29]. One study that was conducted on an Iranian population showed fructose intake of $>$ 72 g/d and 63 g/d was associated with 11% and 9% enhance risk for hypertension in men and women, respectively [14]. Another study [16] reported that daily intake of 200 g fructose increased SBP and DBP. It was previously reported [12] that fructose intake $>$ 50 g/d is associated with the etiologies of MetS.

Previous research found that fructose intake approximately $>$ 8% to 12% of energy intake ($>$ 50 g/d) is associated with MetS [14]. The researchers found that the amount of fructose intake through fruits and vegetables even in the highest quartile was only 5% of energy (30 g/d). Thus, fructose in industrialized foods is considered the main underlying cause for the increasing risk for MetS.

However, some studies did not find any association between fructose intake and serum cholesterol, uric acid, FBS levels [41], plasma insulin concentration, body weight [37,39], and BP [17].

Heterogeneity between studies was identified statistically. In each analysis, one study was excluded and finally homogeneous studies were found. Factors that led to the highest heterogeneity were differences in experimental design, type, and amount of fructose consumed, characteristics of participants (e.g., being overweight, obese, or normal weight), sex differences, and the study duration. Four studies [16,32–34] had the greatest differences according to these criteria of heterogeneity.

Studies showed free fructose presumably was more dangerous than sucrose on the components of MetS [27]. Fructose is phosphorylated in the liver and is converted to glycerol-3-phosphate. Fructose bypasses the phosphofructokinase pathway, which is the major rate-limiting step of glycolysis. Then it is used for glycerol synthesis or fatty acids through *de novo* lipogenesis. Conversion of fructose to fatty acid is more rapid than glucose [42]. Furthermore, fructose decreases TG clearance by lipoprotein lipase [43].

Sucrose is composed of one molecule of glucose and fructose. Investigators believe that absorption and metabolism of free fructose is different from fructose contained in sucrose. Serum fructose concentration after sucrose ingestion compared with

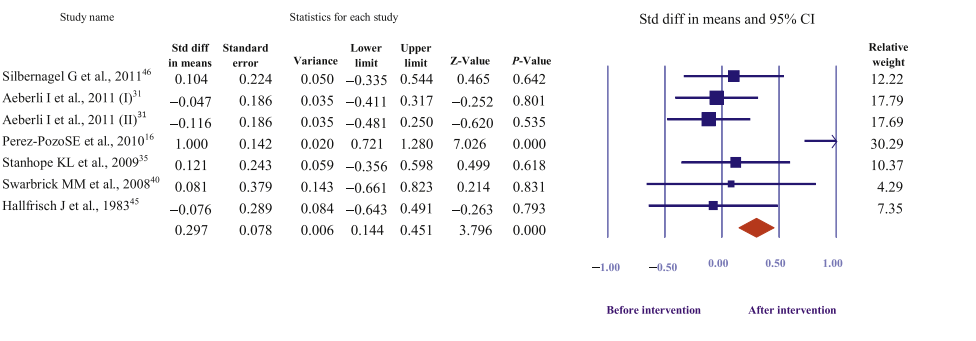

Fig. 4. Meta-analysis of the effect of fructose consumption on blood pressure in published clinical trials. The sizes of the data set indicate the weight of each study in the analysis.

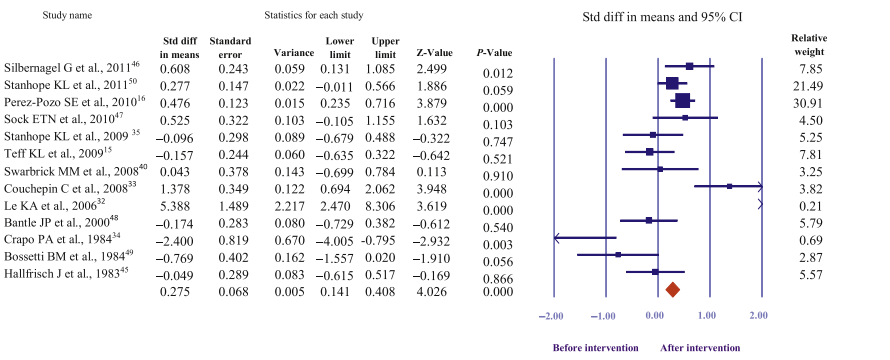

Fig. 5. Meta-analysis of the effect of fructose consumption on triglycerides in published clinical trials.The sizes of the data set indicate the weight of each study in the analysis

free fructose depends on the activity of intestinal brush-border enzymes to hydrolyze sucrose. Thus, fructose contained in sucrose is absorbed more slowly than fructose ingested as a monosaccharide [3]. Because of these differences in metabolism, we excluded those studies in which fructose was administered as sucrose when isolating and determining the effect of fructose on the components of MetS.

It was documented that the source of fructose might be important in determining the risk for MetS. Natural fructose, for example, the fructose in fruits and vegetables, improved some components of MetS such as elevated BP, blood lipids, insulin resistance, and BMI [44]. Likewise, these beneficial effects were seen with a reduction of diet fructose to $<$ 20 g/d [20]. Fruit consumption enhanced the antioxidant system [13] and decreased the ratio of malondialdehyde to antioxidant capacity [44]. Given that consumption of natural fructose could lead to heterogeneity in our study results, we excluded studies conducted on this type of fructose.

Conclusion

According to our meta-analysis, fructose consumption from industrialized foods, as sweetened beverages, is one of the causes of some chronic disorders as MetS in healthy adults. It provides further support to decrease consumption of fructose-sweetened beverages for the prevention of chronic disorders in a healthy population. However, several studies had investigated the effect of fructose in short-term. Long-term trials in the future about the effect of fructose consumption on chronic diseases can help us to make better decision.

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