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Sugar-Sweetened Beverages and the Rising Burden of NAFLD: Pathophysiology, Clinical Evidence, and Preventive Strategies

Preeti Arya[1*], Vir Vikram[1], Benu Chaudhary[2]

[1]School of Pharmaceutical Sciences, CT university, Ludhiana, Punjab, India

[2]Department of Pharmacy, Panipat Institute of Engineering and Technology, Samalkha, Panipat, Haryana

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ABSTRACT:

A new worldwide health crisis, non-alcoholic fatty liver disease (NAFLD) is directly linked to food and way of life. One of the most important factors in the development and worsening of many diseases is the regular use of sugary drinks, especially energy drinks and cold drinks. An important factor in the development of non-alcoholic fatty liver disease (NAFLD) is fructose, a sugar found in many soft drinks. Fructose stimulates hepatic de novo lipogenesis, triglyceride buildup, oxidative stress, and also insulin resistance by evading normal glycolytic control. Metabolic dysfunction is worsened by the high fructose content and other ingredients found in cold beverages and energy drinks, such as caffeine, artificial flavors, preservatives, and sugar. In order to shed light on the pathophysiology of fatty liver and provide solutions for prevention and treatment, this review will examine the components of cold drinks and energy drinks, as well as fructose metabolism, and will draw from current clinical and experimental findings. Hepatic steatosis, inflammatory conditions, and the development of non-alcoholic steatohepatitis are all positively correlated with regular consumption of sugar-sweetened beverages, according to recent research. In a similar vein, increased enzymes in the liver, mitochondrial dysfunction, and decreased hepatic metabolism have all been associated with long-term energy drink use. These results show that sugary and caffeinated drinks are bad for your liver, therefore people should cut less on them. This is why public health campaigns and dietary changes are needed.

1. Introduction

Hepatic steatosis, the abnormal accumulation of triglyceride-rich droplets in liver cells, is a hallmark of non-alcoholic fatty liver disease (NAFLD), one of the leading causes of chronic liver impairment worldwide and ultrasounds with contrast dyes, MRI scans, CT scans, and even histological analyses can all identify it (1). Non-alcoholic steatohepatitis (NASH) is characterized by hepatic steatosis, lobular inflammation, and hepatocyte injuries, and it ultimately leads to liver cirrhosis and hepatocellular cancer (*2*).

NAFLD is frequently linked to a wide variety of other diseases, such as metabolic disorders, diabetes, obesity, and cardiovascular disease (*3*). NAFLD is rapidly becoming the leading cause of abnormal liver functioning in developed countries, right along with the epidemics of overweight (obesity) and metabolic illnesses. NASH will overtake alcoholic liver disease and hepatocellular carcinoma as the primary cause of liver failure and cancer in the next two decades (*4,5*). Ludwig and colleagues first postulated NAFLD in 1980 during their study on patients having many histopathological similarities to fatty liver disease without a previous alcohol usage (*6*).

The growth and advancement of NAFLD may be influenced by a person's lifestyle and dietary habits, which may have a beneficial or harmful role (*7*). Over the past twenty years, people's eating habits have shifted dramatically over the world in large part because people are eating and drinking more things that are high in sugar and saturated fat. Diabetes mellitus, obesity, cardiovascular problems, as well as nonalcoholic fatty liver disease (NAFLD) are just some of the metabolic problems that have been linked to an overconsumption of processed foods and a lack of physical activity (*8*). Although nutrition regulates metabolism, accumulating data suggest that numerous dietary components are directly involved in NAFLD development.

2. Beverages and fatty liver disease

Over the last twenty years, beverages have emerged as almost a substantial part of the western diet and contributed to an increase in liquid energy intake (*9*).

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Alcoholic beverages, sugary carbonated drinks, low-calorie beverages (diet drinks), high-caffeine beverages (energy drinks), and coffee or tea-based beverages are all considered beverages. The impact of beverages on public health is still unclear, despite the numerous studies conducted on the topic. Studies show that drinking some dense calorie liquids not reduces the eating behavior of food to maintain energy. Positive energy equilibrium without calorie compensation is hypothesized to promote weight gain when a person consumes a lot of sugary drinks (*9*). Many animal and human research have linked NAFLD to the long-term use of processed carbohydrates, particularly fructose (*10*). Some evidence suggests that a high-fructose diet may accelerate the development of obesity and NAFLD by raising the risk factors for these diseases (*11*). Coffee and tea are the most popular non-alcoholic drinks, used largely for flavor and taste, although typically regarded as favorable for various organs, such as the liver, because of their low sugar content (*12*). Despite the harmful effects of soft drinks and energy drinks, tea, coffee, and alcohol may help liver ailments (*13,15*). The most frequently consumed beverages are sweetened, hot, and alcoholic. This review set out to comprehensively assess existing observational studies of the influence of these drinks on NAFLD development and management (Fig.1). Before discussing the potential involvement of these drinks in the progression and prevention of NAFLD, we will also demonstrate the pathogenesis of NAFLD. The nutritional treatment methods to NAFLD prevention will be discussed also.

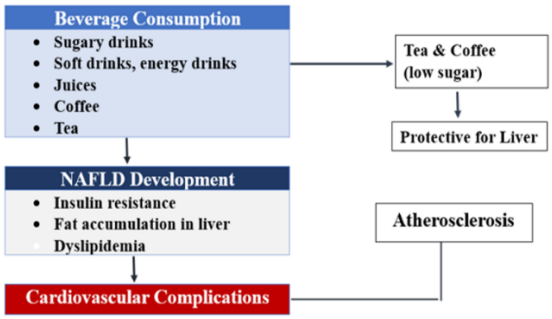

Fig.1 The intricate relationship between beverage consumption and the rising burden of fatty liver disease

NAFLD is highly related to increased cardiovascular disorders due to higher fasting triglycerides and reduced HDL (*16*). Dyslipidemia in children and teens raises the cardiovascular events, which is concerning (*17*). Dyslipidemia in children having NAFLD has not been studied well to determine whether it predicts cardiovascular complications at a later age. Despite metabolic syndrome, NAFLD patients have higher rates of cardiac mortality. NAFLD increases atherosclerosis independently of obesity and abdominal obesity (*18*).

3. Role of fructose in NAFLD

NAFLD and hepatic steatosis are linked to diets heavy in carbohydrates, simple sugars, and saturated fats, especially in adults. Sugar intake is linked to abdominal ultrasound-diagnosed fatty liver in obese teenagers (*18*). The WHO and SACN (Scientific Advisory Committee on Nutrition) attempted to establish the daily sugar consumption guidelines in 2014. A meta-analysis looked at even more than 1700 articles on the subject and found that sugar intake is a strong predictor of weight gain or loss (*19*). SACN also suggested that sugars account for no more than 10% of daily basis calorie consumption [20]. Hence, plain sugar should provide no more than 5% of food energy and sweetened beverages must be avoided (*20*). When compared to the American guidelines, which recommended no more than 25% of total energy come from sugar, these current guidelines are significantly more stringent (*21*). There has been a dramatic rise in recent decades in the use of soft drinks among children, and this trend needs to be taken into account when assessing the risk for metabolic disorders like NAFLD in children.

In the United States, fructose makes up around 11% of an average teen's caloric intake (22), and its consumption has been associated with worsening of NAFLD in adults (*23,25*). Fructose, unlike glucose, bypasses the rate-limiting step of phosphofructokinase cascade during first pass hepatic metabolism. Consuming a lot of fructose can enhance gene expression of lipid synthesis in as little as eight hours in the liver (*26*) and boosts hepatocellular synthesis of TG by boosting glycerol production and also saturated fatty acid production, particularly palmitate (*27*). The markers like gene expression of lipogenesis, obesity, hepatic fat accumulation, plasma triglycerides, as well as oxidative stress have all been shown to rise in animal

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experiments when a high-sugar diet is used. Studies of fructose in place of glucose in short-term adult feeding indicate greater fasting and postprandial plasma TG amounts (*28,29*).

Throughout the past century, fructose intake has climbed fivefold, and in the past thirty years, it has increased by more than twice. High fruit juice consumption has been linked to obesity in children, despite high fructose corn syrup in soft drinks receiving most of the attention (*30,31*). Americans currently consume a total of 66.8 kilograms of sugar annually, with half of it coming from fructose.

Fructose has been linked to the development of the metabolic syndrome according to, a number of researchers and because of its GLUT5 fructose transporter, the liver is the primary organ for processing fructose (*32,33*). The GLUT5 mRNA as well as protein are present in adipocytes, although the amount of that kind of carrier in adipose tissue is comparatively low. GLUT5 transporters in the kidney and small intestine carry fructose molecules across their lumina to urinary excretion or release through the hepatic portal vein (Fig. 2), which goes to the liver directly. Fructose is insulin-independent, skips glycolysis, and stimulates lipogenesis more than glucose. Fructose metabolism is more similar to metabolism of ethanol in liver. Fructose, like ethanol, causes metabolic disorder symptoms (*34,35*).

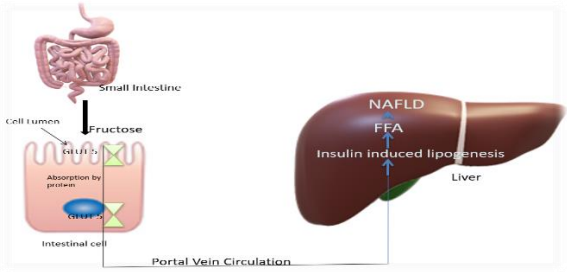

Fig.2 Fructose sneaks past the body's normal metabolic checkpoints, flooding the liver with fat and fueling the silent progression of NAFLD

Fatty liver disease results from fructose consumption. GLUT5 facilitates passive transfer of fructose from the intestinal lumen to the blood circulation. It enters the liver via the portal vein of the liver. Fructose is converted to glucose within the liver, which causes lipid production, steatosis, and obesity. Under normal circumstances, the brain combines signals affecting feeding, metabolism, insulin secretion, fatty acid metabolism, and hepatic glucose synthesis, keeping everything in check and finely tuned (*36,37*).

4. Energy drinks and its Ingredients

In the last few decades, energy drinks have exploded in both the mainstream and the worlds of athletics. There is a development of the basic concept of sugary drinks which led to "soda drinks" so widespread in the modern era (*38,39*). They contain different amino acids, ginseng, and also caffeine with sugary syrup, which distinguish them from sodas. There is some evidence that sugary drinks may contribute to the development of nonalcoholic fatty liver disease, metabolic obesity, and dysmetabolic syndrome while there has been a rise in awareness and concern for dysglycemic conditions, particularly among children, significantly less attention has been paid to the safety of energy drinks (*40,41*). Caffeine excess has been identified as a key contributor to the toxidromes commonly seen with these drinks yet, energy drinks contain a wide variety of ingredients, not just sugar and caffeine (*42-44*). Some beverages may be toxidromes that are multifaceted and hazardous.

The consumption of energy drinks is extremely common and are promoted as safe energy enhancers that can be used everywhere, including at work, in sports, or at home. They often contain a high concentration of monosaccharides, as well as caffeine, along with a variety of additional items, such as ginseng. There has been no thorough investigation of the potential hazards associated with their utilization, and the documentation supporting its asserted capacity for raising energy production is limited. After four months of beginning his daily use of two to three cans of Monster Energy, a 46-year-old Caucasian male with type 2 diabetes as well as nonalcoholic fatty liver disease developed a deadly triumvirate illness comprising pancreatitis, gastritis, and hepatitis. We share this case as a cautionary tale in the hopes that it will help others avoid the same fate. By avoiding the drink, he quickly experienced a resolution of his clinical signs and biochemical anomalies (*45*).

Taurine

A naturally occurring amino acid that is ingested via meat and seafood, taurine helps prevent exercise-related

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injuries (*46*). Despite claims to the contrary, no evidence was found linking taurine's interaction with coffee to improved focus and reaction time. There is insufficient data from scientific studies to determine whether or not the amount of taurine in energy drinks is safe. Researchers at New York's Weill Cornell Medical College revealed that in addition to its rejuvenating effects, taurine also acts as a tranquilizer. Furthermore, it has been speculated that extraneous manufactured taurine may lead to high blood pressure and cardiovascular disease (*47*).

Bile and its derivatives, including taurine, are essential for the conversion of bile acid to bile salt. In the process of the conjugation of bile acids with taurine that takes place in the liver, with various alterations in the hepatocytes. Bile salt hydrolases (BSHs), a group of enzymes metabolize the link of C24 N-acylamide, causing the deconjugation of taurine and glycine bile acids. BSHs promote intestinal bile acid deconjugation, lipid metabolism, fluid retention, and alters signaling. Diseases like hypercholesterolemia, overweight, and disruptions in circadian rhythm have been linked to BSH dysregulation (*48*).

Niacin

In NAFLD, diacylglycerol acyltransferase 2, a crucial enzyme that catalyzes the final stage in TG production, is inhibited by therapeutic dosages of niacin, which is a water-soluble vitamin (B3). Niacin, on the other hand, has been shown in some research to be responsible for the development of fatty liver in rats that were fed a high-fat diet (HFD). For this reason, "*Fang et al.* (2020)" tested the efficacy of niacin in curing NAFLD using two mouse strains, B6129SF2/J (B6129) and C57BL/6J (B6) mice, after they were fed an HFD for 5 months. Beginning in week 5 and continuing through the remainder of the research, animals received niacin. In HFD-fed B6129 mice, therapy with niacin increased normal liver weight, TG level, and also improved NAFLD, while having no effect on B6 strain. In B6129 mice, metabolomics showed no alteration in the functions of 4-hydroxyphenylpyruvic acid, which itself is involved in fatty acid oxidation, but it was dramatically reduced after niacin administration. Lipidomics showed that HFD-fed B6129 treated with niacin had a lesser amount of phosphocholine, which is essential for synthesis and release of VLDL–TG (*49*).

A healthy twenty-three-year-old lady was diagnosed with acute hepatitis, characterized by jaundice and malfunctioning of the liver, after consuming 10 cans of an energy drink daily (*50*). That lady refused to take any other medications, and there was no measurable quantity of acetaminophen in her system. She was able to get better with just supportive therapy. The investigators suspected that niacin from the energy drink caused the severe hepatitis, despite the fact that the victim had only ingested 300 mg per day. The lowest quantity documented to cause hepatotoxicity is 1 gram [50]. After receiving a whole liver transplant, a 16-year-old male because of repeated cholestatic hepatitis, was reported by *Apestegui et al.* (51). The patient experienced two separate bouts of jaundice that coincided with his ingestion of multiple Red Bull energy drinks. The use of niacin has been blamed for the hepatitis once more.

Caffeine

The main psychoactive component in energy drinks is caffeine, but the dosage varies greatly across different brands (Table 1) Taurine, niacin, guarana, ginkgo biloba, ginseng, milk thistle, levocarnitine, and others are some of the ingredients that are commonly found in energy beverages (52-60) and are said to improve physical and cognitive performance (*59*). As the world's highest caffeinated plant, guarana serves as a convenient delivery system for caffeine in energy beverages (*61,62*).

Table 1 Several popular beverage brands contain significant amounts of caffeine, including soft drinks, energy drinks, and ready-to-drink coffees, all of which contribute to rising health concerns when consumed excessively.

Amount of caffeine in different brands			
Brand Name	Volume(ml)	Caffeine content (mg)	Reference
Coke	222	18.43	[63]
Diet Coke	222	23.87	[63]
Mountain Dew	355	45.4	[63]
Thumbs up	100	17.8	[66]

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Pepsi	355	31.7	[63]
Sting	200	112	[64]
Red bull	245.46	66.7	[63]
Starbucks Coffee	280.9	63.8	[63]
Rockstar	400	80	[65]
Monster	236	80	[67]
Burn	250	97.5	[68]

5. Artificial sweeteners

Many people who have problems with their metabolism or who are overweight use artificial sweeteners. Yet, there is mounting evidence that they have a role in the onset of intolerance of glucose through altering the microbiota's makeup and function. A study found that eating saccharin, sucralose, or aspartame made people less able to handle glucose. This has been linked to changes in the gut bacteria, such as more *Bacteroides vulgates* and fewer beneficial bacteria. Antibiotics prevented these effects in germ-free animals [69]. It's possible that this impact is caused by an increase in GLUT2 activity in the small bowel [70]. In response to β-adrenergic agonists and gut hormones like glucagon-like-peptide 1 and 2, as well as the amount of leptin, GLUT2 transporters at apex, change both transmembrane entry and functioning [70]. The hepatic GLUT2 transporter fully extracts fructose from portal blood and oxidizes it into glucose and lactate. Then both are either metabolized to glycogen that can be stored or towards the de novo lipogenesis pathway [*70*). There is evidence that artificial sweeteners are associated with a number of health problems, including cardiovascular disease, diabetes, obesity, metabolic disorders, and cognitive impairments (*71*).

Soft Drinks

Soft Drinks (SD) are nonalcoholic carbonated beverages including soda, pop, soda pop, Pepsi™, Coke™, etc. There are two main kinds of SD on the market today: the traditional kind, which uses sugars like fructose, and the diet kind, which uses artificial sweeteners to save calories like aspartame. Prior to the 1980s, the majority of the calories in a can of SD came from processed cane sugar (*72*). As a result of its lower price, HFCS (high-fructose corn syrup) has replaced sugar as the primary sweetener in the Americas as well as other nations. Table 1 provides an overview of the amounts of calories and sugar that can be found in a selection of popular soft drinks.

Table 2 Summarized list of popular cold (soft) drinks, their approximate calorie content per can, and the diseases or health risks linked to regular or high consumption.

Cold Drink	Calorie per can	Associated health risks
Cola (e.g., Coke)	~156 kcal	Weight gain, visceral (belly) fat, metabolic syndrome, type 2 diabetes, heart disease, NAFLD, tooth decay, gout, dementia, some cancers (*73*)
Sugar-Sweetened Beverages (general)	Varies (~150 kcal)	Obesity, type 2 diabetes, cardiovascular disease, chronic kidney disease, cancers linked to obesity (e.g., colorectal, pancreatic) (*73*)
Generic Sugary Soda	~150–200 kcal	Empty calories devoid of nutrients, obesity, insulin resistance, type 2 diabetes, fatty liver disease (NAFLD) (*74*)
Soft Drinks (frequency impact)	Varies	Increased risk of all-cause mortality, cardiovascular diseases and tooth decay with ≥2 drinks/day. (*75*)
HFCS-	Caloric	May promote

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Containing Drinks	similar to sugar (~4 kcal/g)	hepatic de novo lipogenesis (fat creation), increase triglycerides, uric acid, contribute to NAFLD, NASH, fibrosis. (*76*)

In addition to sugar, aspartame and caramel (a coloring agent) are commonly employed as substitutes in the soda market, especially for diet sodas (*77*). Artificial sweeteners may reduce hunger and calorie consumption because they stimulate the release of glucagon-like peptide 1 through the digestive system, as demonstrated by Brown et al. (*78*). Caramel is produced through a process known as caramelization, which involves the processing of polysaccharides with heat in a cautious and regulated manner, commonly in presence of both acids and strong bases. Caramel coloring is included in soft drinks, and is known to be high in advanced glycation end products, which are known to induce diabetes as well as inflammatory reactions (*79,80*). The Food and Drug Administration has determined that an intake of 200 milligrams of caramel per kilogram of body weight daily basis is permissible. Rats as well as ducks that were fed diets high in fructose developed fatty liver disease (*81*). These diets have also been shown to be responsible for increases in lipid peroxidation in liver as well as for triggering the signals of inflammatory cascade in rodents and cause metabolic diseases (Fig. 3) (*82*).

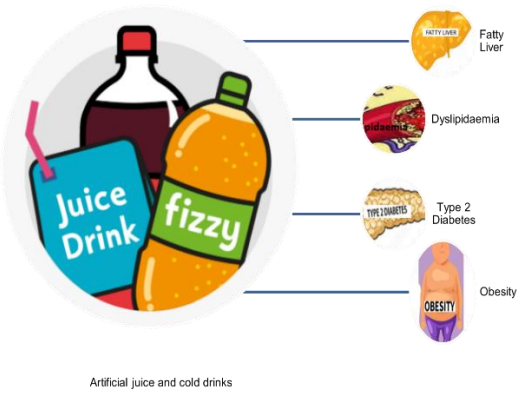

Fig.3 The Hidden Harm of Cold Drinks

6. Existing research indicates that cold drinks affect liver health

The rising global prevalence of childhood obesity has led to a rise in the prevalence of nonalcoholic fatty liver disease (NAFLD), a highly persistent liver condition affecting young people. Non-alcoholic steatohepatitis (NASH) is currently sufficiently linked to diets heavy in carbs and sweeteners. Consumption of sugary beverages, especially those with a lot of calories and fructose, plays a significant effect in the development of nonalcoholic fatty liver disease (NAFLD) and its gradual development to NASH. Furthermore, it appears that extra elements in the drinks, such as xanthine, play a significant involvement in the development of NAFLD. In order to combat youth obesity with nonalcoholic fatty liver disease, it is recommended that they cut down significantly on their intake of sugary and energetic beverages (*83*).

Sam, a middle-aged white male worker with a lengthy healthcare history that includes type-2-diabetes, high lipid levels, high blood pressure, non-alcoholic fatty liver disease (NAFLD), and a previous kidney cancer operation, developed worsening upper abdomen discomfort, cramping, and vomiting. Metformin, liraglutide, and repaglinide were his regular diabetic treatment medicines, and he kept his blood sugar levels well-controlled.

Omeprazole provided short relief from symptoms, but the pain remained when stopped taking atorvastatin and liraglutide. An important discovery was made when he confessed to drinking two or three cans of Energy drinks every day for four months to fight off exhaustion from work. After avoiding energy drinks and drinking more water, his symptoms disappeared within twelve weeks, even when restart liraglutide as well as atorvastatin. Radiographs showed no signs of pancreatitis or any biliary illness; instead, they showed postoperative kidney alterations and widespread steatosis of the liver. Individuals with preexisting metabolic disorders and renal diseases are more likely to experience upper gastrointestinal symptoms if they consume a large quantity of energy drinks, as this instance shows (*84*).

In a new study, investigators looked at the impact on metabolic processes and the function of the liver of combining high-fat diets (HFD) with energy beverage

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consumption in both sex of Wistar rats. The 64 rats were split into four groups for the 12-week experiment: control, control + ED, HFD, and HFD + ED. Body composition, cholesterol level, antioxidant capacity, blood sugar, insulin hormone, leptin, and immunohistochemistry of the liver were among the many biochemical as well as physiological markers assessed. Higher body mass indexes and blood levels of leptin were seen in the HFD + ED group of female rats, suggesting more adiposity and possibly leptin resistance. Muscle mass increased in male rats without an increase in body mass index (BMI), indicating metabolic responses specific to sex. In reaction to stress caused by oxidation, the levels of the antioxidant enzymes superoxide dismutase and catalase were shown to be raised in both female and male rats.

There was a drop in overall protein levels with increase in ALP and ALT in liver function tests, indicating a compromised liver and slow down the synthesis of protein. These findings were most pronounced in female group. The combined consumption of HFD plus energy drinks was associated with more severe liver injury, as shown by histological examination. The study found that female rats were more severely affected by the metabolic disruptions along with liver damage (*85*).

In a cross-sectional study conducted in Thailand, researchers looked at persons experiencing a metabolic condition and their beverage intake in connection to the prevalence of non-alcoholic fatty liver disease (NAFLD). Out of 505 subjects enrolled in the investigation, 67.5% were found to have NAFLD by ultrasonography. The research took place at Siriraj Hospital from 2011 to 2013. Coffee, and tea, chocolate or cocoa, and soft drink/energy drink patterns were explored in depth in the respondents' questionnaires, with follow-up in-person interviews providing further clarification. Overall, the results demonstrated that individuals, irrespective of NAFLD status, consumed beverages in a similar manner after controlling for confounding variables such as body measurements, age, laboratory findings, multiple medical conditions, and overall energy beverage intake. Patients with NAFLD, on the other hand, drank tea at far higher rates. The adjusted chance ratio for the prevalence of nonalcoholic fatty liver disease (NAFLD) was 0.35, which is very low but is associated with drinking a minimum of three cups of coffee per day. Although there was no significant correlation between NAFLD and the majority of the beverages examined, a high coffee consumption seemed to offer some protection (*86*).

Soft drink consumption and the development of nonalcoholic fatty liver disease (NAFLD) were the subject of a recent study that aimed to determine if there was a correlation between the two in asymptomatic individuals who did not suffer from those common risk factors. The 36-months study compared 310 individuals with an ultrasound-confirmed NAFLD diagnosis to 30 normal/healthy subjects along with 31 subjects without traditional metabolic susceptibility markers. Throughout the whole trial, participants were monitored for metabolic indicators including oxidative stress as well as insulin resistance in addition to their regular exercise and food habits. Eighty percent of NAFLD patients, in contrast to twenty percent of healthy subjects, drank soft drinks with a large quantity of added sugar (described as 50gm/day). Coca-Cola as well as Diet Coke were the drinks that people consumed the most. Patients' levels of fatty liver disease varied from moderate to profound. In particular, levels of MDA AND HOMA-IRI were considerably higher in patients having NAFLD compared to controls, suggesting increased sensitivity to insulin and a state of oxidative stress. Soft drink use remained the sole reliable determinant of fatty liver when accounting for nutritional and physical activity factors. It accurately identified 82.5% overall incidences with both good specificity and sensitivity. The results highlight the significance of cutting back on sugary drinks to avoid destruction of the liver, and they also provide compelling evidence that NAFLD can develop in those who do not have traditional risk factors for metabolic disease (*87*).

The purpose of this descriptive study was to examine the association between the intake of soft drinks and the onset of nonalcoholic fatty liver disease (NAFLD) in people residing in China. This particular demographic is known for its comparatively low use of soft drinks. In order to assess for NAFLD and to recognize individuals with high amounts of liver biomarkers, 26,790 adults participated in a research project that was carried out in Tianjin, China. The participants received hepatic ultrasonography as well as plasma alanine aminotransferase (ALT) screening. There were three levels of participants' weekly soft drink consumption

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assessed using a standardized dietary habits assessment: those who drank not more than one cup, those who drank at least three cups, along with those who drank extra than one cup.

After controlling for potential confounding variables, such as metabolic syndrome (MetS) outlined by the American Heart link, the link among soft drink intake and nonalcoholic fatty liver disease (NAFLD) was assessed using multivariate regression models. Among the participants in this research, 27.1% had non-alcoholic fatty liver disease (NAFLD), while 6.5% had NAFLD together with high amount of ALT. Utilization of soft drinks was positively associated with the incidence of nonalcoholic fatty liver disease (NAFLD). The odds of developing nonalcoholic fatty liver disease (NAFLD) were 1.14 for those who drank a minimum of one cup of soft beverages per week versus those who drank nearly no soft beverages at all, and 1.26 for those who drank at least two cups per week. The link was much greater for NAFLD patients with increased ALT, with odds ratios of 1.16 for the group drinking less than one cup per week and 1.32 in the group drinking one cup or more per week. Even after controlling for the metabolic disorder, these results showed that drinking soft drinks increases the incidence of nonalcoholic fatty liver disease (NAFLD). This study is the first to demonstrate such a link in a non-Caucasian population with relatively low soft drink consumption, highlighting the potential benefits of reducing soft drink intake as a preventive measure against NAFLD (*88*).

The Israeli National Health and Nutrition Survey included 375 participants who were subsamples for the purpose of studying their eating habits related to primary nonalcoholic fatty liver disease. Following the application of criteria for exclusion, 349 participants underwent evaluation and primary NAFLD was confirmed in 30.9% of them. Researchers discovered that people with NAFLD ate 27% greater quantities of meat and almost double the amount of soft beverages as people with no disease. A trend toward less consumption of seafood high in omega-3 fatty acids was also seen. A higher incidence of nonalcoholic fatty liver disease (NAFLD) was correlated with a diet that included meat and drinking alcoholic beverages even after controlling for age, gender, body mass index (BMI), and total consumed calories. These results provide more evidence indicating a diet heavy in meat and beverage consumption may increase the possibility of nonalcoholic fatty liver disease (NAFLD), but a diet strong in omega-3 fatty fish could mitigate this risk (*89*).

During the current research, experts examined four European cohorts comprising 42,024 people to see if there was a correlation between drinking sweetened drinks /low-or no-calorie beverages /fruit juice and the development of non-alcoholic fatty liver disease. A non-alcoholic fatty liver disease (NAFLD) diagnosis was made when the Fatty Liver Index score was 60 or above. The results showed that the number of cases of NAFLD increased by 7% for every extra serving of sweetened drinks consumed daily. The prevalence of nonalcoholic fatty liver disease (NAFLD) was found to be greater at lower consumption levels of low-or no-calorie beverages, and this connection stagnated at greater amounts of drinking.

Those taking more than two LNCB servings each week showed dramatically higher probabilities of NAFLD. On the other hand, modest fruit juice consumption more than 2 serving in a week was related with a decreased probability of NAFLD, while more servings did not have beneficial response. A higher incidence of NAFLD was associated with substituting LNCB for SSB, which is an interesting finding. In contrast, substituting fruit juice for SSB had no discernible benefit. While reasonable usage of fruit juices may have a protective effect in NAFLD, the investigation found that SSB and LNCB usage were positively correlated with NAFLD (*90*).

Adolescent males who do not have obesity were the subjects of the current investigation, which sought to identify metabolic risk indicators for NAFLD by analyzing their exercise habits, soft drink use, and clinical markers of metabolic disorders. During a routine health screening, 134 male college students underwent assessment and were classified as either having non-alcoholic fatty liver disease (NAFLD) or not (non-NAFLD) according to the findings of gastrointestinal ultrasonography. Metabolic indicators including high blood pressure, belly fat, damaged livers, dyslipidemia, and reduced tolerance to glucose were more prevalent in the nonobese group than in the obese group. On the other hand, people with NAFLD were far more likely to have these characteristics, as well as to

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consume carbonated beverages. Students who did not have obesity were more likely to have NAFLD if they had higher cholesterol levels and consumed soft beverages often, according to both univariate as well as multivariate studies. Among the most striking associations was the elevated odds ratio for soft drink intake. In nonobese teenagers (male), these results point to increased cholesterol levels and frequent soda consumption as potential risk factors for nonalcoholic fatty liver disease (*91*).

People with a diagnosis of NAFLD were the subjects of this research, which aimed to determine how caffeine consumption affected metabolic markers of liver. Forty individuals, consisting of 20 men and 20 women ranging in age from 19 to 64, were divided into three different groups according to their routine daily consumption of caffeine that was 150 mg or less, 150 mg to 250 mg, and 250 mg or more every day. Biochemical indicators, such as levels of protein and hepatic enzyme in the blood, were measured in addition to anthropometric data. The study found that people who consumed the least amount of caffeine reported a higher percentage of extra fat on their bodies, whereas those who consumed 250 mg or more of caffeine per day showed greater concentrations of SGOT and SGPT, suggesting possible stress to the liver.

This group also exhibited reduced levels of high-density lipoprotein cholesterol. The total amount of caffeine consumed was positively correlated with SGPT levels. There was no association among caffeine consumption and blood protein concentration. The results indicate that individuals with NAFLD may experience adverse effects on the liver enzyme level and high-density lipoprotein cholesterol due to daily caffeine use of 250 mg or more (*92)*.

The purpose of the randomized controlled study was to examine the impact of physical exercise and individualized dietary modifications for NAFLD in metabolic individuals who were overweight. One hundred fifty-five people, a diagnosis of NAFLD were assigned to a traditional meal diet. A Mediterranean-style diet that has a high meal frequency (MD-HMF), with organized physical activity (MD-PA) were assigned at random to one of the three groups. Increasing the use of energy and decreasing calorie intake by 25-30% was the goal of all strategies. Although there were no notable variations across the groups, it was shown that higher energy expenditure was linked to lower levels of insulin resistance, glomerular hyperfiltration, and fatty liver tissue. These results provide credence to the idea that people with a metabolic disorder can effectively manage their NAFLD with reduced calories and exercise (*93*).

Caffeine use, the frequency and extent of hepatocyte fibrosis in patients with nonalcoholic fatty liver disease were the subjects of a meta-study and comprehensive review designed to examine this relationship. Three research studies examined the frequency of NAFLD in 18,990 subjects while three studies examined hepatocyte fibrosis in 1,074 subjects; an entire sample of 20,064 people was included in the analysis. Neither the occurrence of NAFLD nor the extent of hepatocyte fibrosis was shown to be substantially linked with total daily caffeine/day from all sources.

There were not any substantial connections found in analysis of subgroups based on research design or geographical context. Caffeine from ordinary coffee was related with less fibrosis in NAFLD participants; however, this association was not found when data was categorized by source of caffeine. This provides more evidence that the beverage contains bioactive chemicals that could have beneficial effects on liver function beyond just caffeine. Consistent coffee use may help reduce the impairment of the liver in people with NAFLD, even though overall caffeine intake doesn't seem to increase NAFLD or advanced fibrosis (*94*).

A meta-analysis and comprehensive review looked at surveys to see whether there was a connection between drinking sugar-sweetened beverages (SSBs) and NAFLD. A total of six research studies with 6,326 individuals and 1,361 instances of NAFLD were incorporated in the systematic literature review, which followed an examination of 1,015 papers. The review of the literature included four cross-sectional research studies. Even after controlling for important associated variables, the results showed that the likelihood for acquiring NAFLD was 40% greater in people with the greatest consumption of SSBs as compared to those with the least consumption. Analyses of sensitivity verified a continuous positive correlation. The results show that both men and women who consume a lot of sugar-sweetened beverages are more likely to develop

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nonalcoholic fatty liver disease (NAFLD), which lends credence to the idea that limiting SSB consumption is a good way to lessen the impact of the condition on the general population (*95*).

According to Vancells Lujan., *et al*. 2021, the most prevalent persistent liver condition worldwide is non-alcoholic fatty liver disease (NAFLD), that strongly associated with the increasing prevalence of both Type 2 diabetes and obesity. Hepatic buildup is a hallmark of this condition, which can advance to inflammation, cirrhosis, fibrosis, and non-alcoholic steatohepatitis (NASH). The foundation of management is alterations to routine eating habits, especially the nutrition and level of workouts, because there is presently no proven pharmaceutical remedy. An unhealthy diet rich in calories, fatty and trans fats, omega-6 polyunsaturated fats, along with added sweets is deleterious to the onset and advancement of non-alcoholic fatty liver disease (NAFLD). Alternatively, there is some evidence that lees-calorie vegetarian and vegan diets, like the Mediterranean diet, can improve the state of the liver. More research studies are needed to discover the best nutrition methods for NAFLD in both prevention and therapy, considering that the latest recommendations support such dietary practices. In order to manage or avoid NAFLD, it is crucial to adopt more nutritious eating habits and cut back on dangerous food additives (*96*).

College students (aged 18–35) were the subjects of an observational study which looked at the implications of consuming energy beverages on hepatic activity. There were two groups of fifty students each, one group consumed energy beverages and other that did not. The study analyzed serum concentrations of SGOT, SGPT (liver enzyme), anthropometric markers and vitamin B12, which were all the parameter analyses in study. Researchers found the elevated levels of all parameters in serum than who did not consume any energy drinks, particularly in males rather than females. The use of energy drinks was also associated with marked rises in both BMI and systolic blood pressure. Results showed that people who drank too many energy drinks had worse metabolic conditions, higher levels of liver markers, and were at risk for liver damage. Potentially contributing to these unfavorable results are essential components like taurine, caffeine, B vitamins and guarana (*97*).

In the Health Professionals Prospective Study, 1,759 adults from Mexico were studied to determine if there was a correlation between the intake of soft beverages/carbonated beverages and possibility of fatty liver disease. To determine NAFLD, the Hepatic Steatosis Index (HSI) was used, and patients were grouped according to their daily consumption of beverages employing a standardized questionnaire. After accounting for potential confounding variables, the results demonstrated that consuming moderate amount (not more than 3.5 serving in a week) and high amount (more than 3.5 servings per week) substantially raised the likelihood of NAFLD. The correlation seemed to be more pronounced in males than females. These results add to the mounting confirmation that consistent intake of cold drinks, probably because of its elevated sugar content, contributes to the onset of NAFLD. Public health initiatives aimed at preventing NAFLD should prioritize lowering SD consumption, according to the study (*98)*.

An increasing number of people around the world are turning to energy drinks that contain caffeine. These drinks are usually made up of basic sugars with significant amounts of caffeine, but they may also contain minerals, vitamins, or extracts of herbs. Despite claims to the contrary, the effects of these drinks on metabolic processes, digestive system, particularly liver functions are a cause for concern. Metabolic control is mostly dependent on the gastrointestinal tract and the liver, which are the initial organs to come into contact with these substances.

These beverages have wildly varying amounts of sugar (6–29 gm/serving) and caffeine (300% more than conventional colas in some cases). The review emphasized the importance of investigating short-term as well as persistent consumption throughout different populations, and it advocates for studies to take into account the cumulative impact of all constituents instead of evaluating them separately. Furthermore, it acknowledges that caffeine sensitivity is likely to influence how people react to such beverages. The intricate interactions and possible negative effects of such beverages, which are introduced to the digestive tract promptly and have a significant impact on the metabolic processes of liver, need to be further investigated in subsequent research (*99*).

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Employing information pertaining to 136,277 persons from the United Kingdom Biobank, the current research investigated the correlations between the possibilities of non-alcohol-related fatty liver disease (NAFLD) with consumption of artificially sweetened beverages (ASBs), pure fruit juice, and sugar-sweetened beverages (SSBs). With comparatively lower consumption of ASBs, the largest association seen with higher chances of occurring NAFLD. The link between PJ and NAFLD incidence was weak and unreliable, nevertheless, with reduced intake frequencies suggesting some protection and greater usage suggesting a little increase. Consumption of ASBs exceeding 2L/week can elevate the chance of hospitalized NAFLD up to 35% in a time period of 10.2 Years. Having said that, there was no discernible correlation between the consumption of SSB or PJ and the likelihood of NAFLD hospitalization. The results indicate that despite all three kinds of drinks being associated with nonalcoholic fatty liver disease (NAFLD), the likelihood of serious liver damage might be highest with artificially sweetened beverages, suggesting that people should drink them with caution and indicating that more research is needed in this area (*100*).

An extensive prospective group of research was conducted in China to examine the link among the use of soft drinks and the likelihood of developing nonalcoholic fatty liver disease (NAFLD) in adults. Using gastrointestinal ultrasound, NAFLD was identified throughout the average follow-up period of 4.2 years in 14,845 subjects who did not have any history of serious chronic liver diseases. Baseline measurements of soft drinks use were taken. The risk of developing nonalcoholic fatty liver disease (NAFLD) was found to be dose-dependently related to soft drink consumption. The incidence ratios for nonalcoholic fatty liver disease (NAFLD) were 1.18, 1.23, and 1.47, respectively, for those who consumed less than one serving per week, two to three servings per week, and four or more servings per week. For greater intake indices in particular, sensitivity studies employing the hepatic steatosis index (HSI) revealed comparable tendencies. The results of this study corroborate recommendations from the public health community to reduce sugar-sweetened drink usage in order to improve the condition of the liver, and they offer compelling potential data suggesting that regular drinking of soft drinks raises the risk of nonalcoholic fatty liver disease (NAFLD) (*101)*.

A prospective analysis of the Framingham Heart Study focused on the association of drinking low-calorie sodas or sugary drinks and the onset of fatty liver disease along with abnormal distribution of fat in liver with time. Hepatic fat was evaluated using computed tomography, and volunteers were grouped into Second and Subsequent Generation groups according to their consumption of sugar-sweetened beverages and low-calorie drinks. The findings demonstrated a dose dependence connection in the elder Offspring group, having probability of acquiring NAFLD being over doubled for frequent sugary beverages drinkers when compared with those who did not consume Furthermore, there was a significant rise in fat in the liver in both casual and regular SSB users. In contrast, in the younger third or subsequent generation group, there was no discernible correlation between drinking soft drinks and NAFLD, as well as the same held true for low-fat soda. It is necessary to implement focused community health initiatives to decrease SSB utilization, especially in groups that are at risk, because these results indicate that increased SSB drinking is a substantial risk determinant in NAFLD among older individuals, regardless of fluctuations in bodyweight (*102*).

Research compared children with NAFLD to their healthy counterparts in order to determine the effects of fructose on the metabolism of lipids among children. Over the course of two days, nine children confirmed to have NAFLD and ten healthy subjects participated in this randomized, supervised clinical trial. Each group received three identical meals. Every single meal during the experimental crossover trial was accompanied by either glucose or fructose-rich drinks. Tests were conducted to evaluate glucose, HDL cholesterol levels, apolipoprotein B, insulin, non-esterified fatty acids and triglycerides.

Among both normal and NAFLD-afflicted children, the results showed that fructose significantly raised the level of triglycerides in the plasma relative to glucose. Fructose also caused a fall in the amounts of HDL cholesterol among all participants, especially after meals as well as throughout the night, although glucose had no comparable impact. Children having NAFLD

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consistently had greater amounts of non-esterified form of fatty acids, suggesting a malfunction in metabolism.

The study concludes that even a short-term ingestion of fructose from food causes unfavorable lipid alterations, with greater severity for children with NAFLD. The importance of dietary therapies aimed at reducing fructose levels is highlighted by these findings, particularly in pediatric populations that are susceptible for NAFLD (*103*).

The purpose of this study was to examine the effects of energy drinks on the health of youth by reviewing the most current research in the field. These drinks are popular beverages without alcohol that are packed with sugar, caffeine, and a variety of additional ingredients like ginseng, taurine, creatine, and guarana. According to the research, drinking these drinks on a frequent basis is associated with a host of negative health impacts, most notably on the neurological health and heart disease. Caffeine raises blood pressure and the risk of high blood pressure and stroke by causing irregular heartbeats, tachycardia, and other cardiac rhythm problems. Not to mention that energy drinks have a reputation for making people anxious, hyperactive, and unable to sleep. Type 2 diabetes, obesity, and dental problems are all exacerbated by the substantial amounts of sugar in these drinks. Additional scientific investigation in ethical way, is needed in order to comprehend the long-lasting medical repercussions of energy drink intake in young people, according to the comprehensive analysis that was conducted using Google Scholar and PubMed employing specified illness-related keywords (*104*).

Dietary fructose influences hepatic gene expression and metabolic regulation, which in turn contributes to the onset and progression of nonalcoholic fatty liver disease (NAFLD), as this review demonstrates. Utilized as a component of sucrose as well, fructose disrupts the breakdown of lipids, increases de-novo lipid synthesis and fatty acids absorption, and reduces fatty acid expenditure; all of which are linked to steatosis of the liver. Fructose consumption increases expression of genes involved in lipid production and decreases transcription of genes involved in lipogenic processes, according to a number of cell strains and experimental animal research. Additionally, there is substantial upregulation of inflammatory mediators including IL6, TNFα, and CCL2, after fructose consumption.

Chrebp and Fgf21 are important transcription factors involved in lipid production and circulating metabolic signaling; fructose also changes their expression. Elevated fructose consumption leads to NAFLD progression through a basic mechanism, as these genetic modifications are conserved across organisms, particularly humans. The results show that fructose in the meals affects the state of the liver, and they also show that it plays a role in leading to metabolic abnormalities causing NAFLD and modifying the expression of genes (*105*).

7. Future with Regard to Dietary and Life style Changes

At the moment, there is no pharmacologic medication that has been granted approval that can treat NAFLD in every patient. Nonetheless, appropriate specific dietary recommendations for persons suffering from NAFLD include things like having coffee without sugar and attempting to avoid corn syrup in their diet (*106*). NAFLD has been demonstrated to be mitigated by, or at least weakly associated with, a diet rich in fruits, vegetables, olive oil, seafood, grains, and nuts. The onset of NAFLD is instead favored by a Western diet heavy in red meat, soft drinks, fatty acids that are saturated, and sugar, (*107*). Potentially beneficial and detrimental effects can be seen as a result of short-term therapies that involve adjusting macronutrients. As such, limiting intake of carbs and saturated form of fatty acids by macronutrient alterations has been linked to positive health outcomes (*108*).

Based on a comprehensive literature analysis of existing research and theory, we have identified five primary nutritional guidelines, which are as follows:

(i) Conventional eating plans like the Mediterranean diet,

(ii) Restriction of fructose-rich processed meals and drinks,

(iii) Diets low in saturated fat and high in unsaturated fats,

(iv) Suitable replacement of fast food with fruits, processed food with high fiber food and bakery products and sweets, nuts, as well as seeds,

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(v) Decrease of overindulgence in alcohol (*109*).

Hence, enhancing the quality of diet while adhering to these rules will considerably cut the incidence of NAFLD as well as its progression (Fig. 4). Dietary patterns are thought to provide these benefits rather than one particular parameter (*110*).

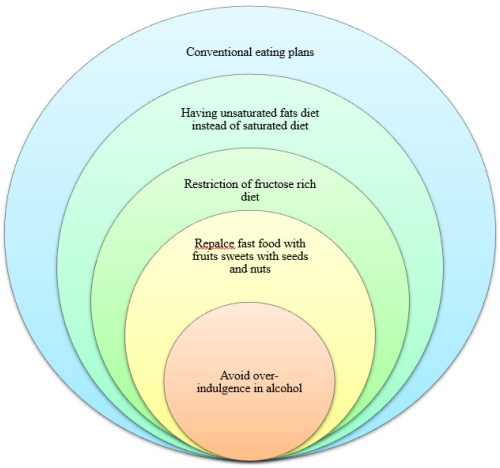

Fig. 4 Nutritional Guidelines to overcome NAFLD

Nutritional therapies reduce the biological and metabolic impact that medications exert upon the body. It is frequently advised to lose weight in conjunction with lifestyle therapies for NAFLD that could also reduce inflammation and damage in the liver, liver steatosis, irregular lipid profiles, and cardiac disorders (*111*). The metabolic syndromes are largely caused by soft or energy drinks which also add excess fat to meal. To foster an appropriate atmosphere, it is crucial to tackle these carbonated drinks. Many natural sweeteners, including stevia, hoodia, trehalose, dexorotary tagatose, have just been discovered that help in losing weight. They protect against NAFLD, insulin resistance, and that can replace sugar in SSBs to satisfy sweet cravings without excess energy utilization (*112*). Green and Syn (2019) summarized the clinical and laboratory evidence linking low consumption of noncaloric sugar substitutes with minimum rates of metabolic syndrome, including NAFLD (*113*). However, definitive results are urgently required from treatment studies involving individuals.

Furthermore, consistent physical activity has been shown to lower lipid levels of the liver. Particularly, various studies have demonstrated a negative correlation between degree of severity in NAFLD and high-intensity exercise. One study with more than 5,700 participants showed a dose-response relationship between exercise and the development of nonalcoholic fatty liver disease (NAFLD). Short-term exercise, when done on a regular basis, can reduce death of liver cell in obese NAFLD patients through enhancing responsiveness to insulin and bolstering antioxidant capacity, implying that exercise regimens as well as dieting can reduce histomorphological markers of fatty liver in these patients (*114*).

8. Conclusion

Several studies have linked fructose consumption to increased risk of obesity and related diseases like nonalcoholic fatty liver disease. We present a large body of research showing that fructose also inhibits the body's ability to burn fat from food consumed. Despite the weight and volume of corroboration, the full scope of metabolic activities of fructose has received comparatively little attention and investigation. More research is needed, but these results have huge implications for people whose health has been negatively impacted by eating a Western diet. There have been a number of studies showing a correlation between the increasing popularity of simple sugars and the prevalence of obesity, type 2 diabetes, and nonalcoholic fatty liver disease. To date, carbohydrate rich but also sugary components of soft as well as energy drinks seem to play the biggest role in NAFLD's onset and development into NASH. Keep in mind that other components of these drinks appear to have a significant influence on the etiopathogenesis of NAFLD. Obesity and other metabolic illnesses for example NAFLD can be prevented by massively reducing these kinds of sodas. It appears that lifestyle modification is the only proven treatment for NAFLD at the present time. Individual or community-based interventions aimed at reducing exposure to unhealthy food and drink may be most effective. The identification of molecular therapies again for the intervention of NAFLD especially due to beverages should also be facilitated by a greater awareness of the function of each ingredient involved in the pathogenesis of such conditions.

Conflict of Interest

The authors declare no competing interest.

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AUTHORS' CONTRIBUTION

All authors participated in the formation, writing and corrections of this review.

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