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Are soft drink consumption and fatty liver in any way connected?

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Summary

Objective of the present study was to elucidate the effects of soft drink consumption on the prevalence of hepatic steatosis in a randomly selected population-based collective.

A total of 1,435 subjects (54.6 % women, 45.4 % men) underwent upper abdominal sonographic examination and were questioned regarding their soft drink consumption and their body-mass index (BMI), waist-to-hip ratio (WHR); laboratory parameters and anthropometric data were documented.

The prevalence of hepatic steatosis in the overall collective stood at 26.1 % (n = 374; women 39.0 %; men 61.0 %). Consumption of soft drinks was reported by 20.8 % of subjects (n = 298); hepatic steatosis was diagnosed in 23.2 % (n = 69). The prevalence stood at 26.8 % among non-consumers. Male sex, greater age, higher BMI and WHR increased the risk of developing hepatic steatosis. A higher prevalence of hepatic steatosis could not be demonstrated for consumers of soft drinks (p = 0.3715).

Keywords: Fatty liver, NAFLD, soft drinks, ultrasound

Introduction

The increase in overweight, type-2 diabetes mellitus and dyslipidemia has reached epidemic proportions. Closely associated with it is the development of non-alcoholic fatty liver disease (NAFLD) [1]. NAFLD is increasingly being recognized as a serious health problem, especially in Western industrial nations [1–3]. The spectrum of NAFLD extends from simple deposits of fat in the liver to non-alcoholic steatohepatitis (NASH), fibrosis and, ultimately, cirrhosis [1–3]. In simple steatosis, histology reveals triglyceride-containing fat vesicles within the hepatic parenchyma; NASH, by contrast, is characterized by inflammatory infiltrates and cell destruction. In cirrhosis, scar tissue replaces the degenerated hepatic parenchyma [2].

Soft drinks are defined as non-alcoholic carbonated beverages sweetened either with non-caloric sweeteners such as aspartame ("diet soft drinks") or with sugar, especially fructose ("regular soft drinks"). Since the 1960s, high-fructose corn syrup (HFCS) which contains a high percentage of fructose (HFCS-42 and HCFS-55 with 42 % and 55 % of fructose, respectively), has been used as a sweetener [4]. Because it is inexpensive to produce, HFCS is widely used in manufacturing soft drinks [5, 6]. It is, therefore, not surprising that the consumption of fructose has climbed by 25–30 % over the past three decades [5]. Over the past fifty years, the per capita consumption of soft drinks has grown by nearly 500 % in the USA [7].

Soft drink consumption is considered a risk factor for obesity, especially in adolescents [7, 8]. Alimentary fructose reaches the liver where, thanks to a specific enzyme, the regulation of glycolysis is evaded: as a result, there is an uncontrolled synthesis and accumulation of substrates used for triglyceride production and stimulation of de novo lipogenesis in the liver [6].

Because of the high lipogenicity and energy content of fructose, a correlation between soft drink consumption and the development of hepatic steatosis has also been suspected [5, 8]. Ultrasonography is a non-invasive diagnostic method for assessing

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the presence of NAFLD with a sensitivity of 60–94 % and a specificity of 73–93 % [1, 3].

Study objective

The objective of the present study was to investigate the correlation between the prevalence of hepatic and consumption of soft drinks and other metabolic parameters in a randomly selected urban population-based sample.

Material and Methods

Study population/Participation rate

During the period of 4 November 2002 to 7 December 2002 a total of 4,000 inhabitants aged 10–65 years randomly selected from the population of a town in the southeastern part of the State of Baden-Württemberg, Germany were invited to participate in an epidemiological survey. Of these, 107 were excluded due to lack of current mailing address or other reasons. A total 2,445 subjects ultimately participated in the study, corresponding to a participation rate of 62.8 % [9].

In order to better assess the effect of soft drink consumption on the prevalence of hepatic steatosis, subjects with known or suspected hepatitis B or C (n = 31), known hemochromatosis (n = 1), elevated alcohol consumption (n = 240), and subjects less than 18 years of age (n = 258) were excluded from the study collective. Also excluded were subjects with incomplete data. Thus, data from a total 1,435 subjects was included in the statistical analysis.

The study proposal was approved by the ethics commission of the State Medical Board of Baden-Württemberg and was conducted according to the guidelines of the Helsinki Declaration. All subjects gave their written informed consent to participation in the study [9].

Interview and anthropometric data

Subjects' historical data was documented using a questionnaire. Subjects were requested to quantify their frequency of consuming sweets, caffeinated beverages (e. g. cola beverages) and coffee according to "several times daily", "daily", "several times per week", "several times per month", and "rarely/never". For coffee, subjects were asked to quantify the number of cups consumed according to "1–3 cups daily", and "> 3 cups daily" and whether subjects preferred their coffee with or without cream [9]. With respect to beverages consumed in social situations, subjects were presented the choice of "alcoholic beverages", "juice", "water", "coffee/tea", "soft drinks", and "other". Body height, body weight and waist-to-hip ratio (WHR) were documented according to WHO recommendations [10]. Men and women were assigned to common BMI classes. Normal weight, overweight and obesity corresponded to BMI levels of 18.5–25 kg/m^2, 25–30 kg/m^2 and > 30 kg/m^2, respectively [9].

Ultrasonographic examination and hepatic steatosis

Ultrasonographic examinations were performed using four identical HDI 5000 units (ATL Ultrasound, Philips Medical Systems, Bothell, WA, USA) [9]. Criteria applied to interpretation of ultrasonographic liver findings were ability to assess parenchymal structure ("good", "poor", "only intercostal"), presence of normal echogenic parenchyma ("yes/no"), liver size (largest craniocaudal diameter in millimeters in the right medioclavicular line). The diagnosis of hepatic steatosis was made according to the criteria of HAMAGUCHI [3] and SAVERYMUTTU [11], which are based on comparison with the renal and hepatic parenchyma. The severity of steatosis was rated according to "no steatosis", "grade I steatosis (mild)", "grade II steatosis (moderate)", and "grade III steatosis (severe)".

Laboratory testing

About 25 ml of whole blood was obtained from each study participant through the cubital vein. Serum iron, transferrin, triglycerides, glucose, cholesterol, HDL, AP, AST, ALT and GGT concentrations were determined using a Dimension XL (Dade Behring Inc., Newark, DE 19714, USA) [9]. A blood count was performed using a fully automated hematological analysis unit (CellDyn 3500, Abbott), while a AIA-21 immunological analysis unit (TOSOH BIOSCIENCE) provided data on ferritin and the soluble transferritin receptor. Regular precision controls were performed to assure proper functioning of all units.

Statistical analysis

Statistical calculations were performed using the SAS 9.2 statistics software (SAS Institute Inc., Cary, North Carolina, USA). Data were first analyzed descriptively. Median and standard deviation were determined for constant values. Categorical data were presented with absolute and relative frequencies. In order to detect differences between consumers and non-consumers of soft drinks, the Wilcoxon rank-sum test was used for constant variables, while, for categorical variables, the χ^2 test or, when the number of cases was too small, FISHER'S exact test were used. Bivariate logistical regression was used to test a potential correlation between hepatic steatosis and soft drink consumption and other potential factors. Vari-

Glossary

hemochromatosis = also siderophilie, is an inherited disease that leades to increased iron absorption and iron deposition among others in the liver parenchyma.

ables that showed an association in the bivariate analysis were included in the multivariate logistical regression analysis. All tests were two-tailed. Statistical significance was set at $\alpha = 5\,\%$.

Results

The collective showed an average age of 42.3 ± 12.8 years and consisted of 54.6 % (n = 783) women and 45.4 % (n = 652) men. Ultrasonographic examination revealed evidence of hepatic steatosis in 26.1 % (n = 374) of subjects, among whom there was a predominance of males (p < 0.0001; 61.0 % men vs. 39.0 % women).

	Hepatic steatosis not diagnosed Mean ± SD	Hepatic steatosis diagnosed Mean ± SD	p
Demographics			
age	39.9 ± 12.4	49.0 ± 11.5	< 0.0001
BMI	24.1 ± 3.8	29.7 ± 4.7	< 0.0001
WHR	0.8 ± 0.7	0.9 ± 0.7	< 0.0001
Laboratory findings			
ALT (U/l)	13.3 ± 5.6	20.1 ± 10.7	< 0.0001
AST (U/l)	8.9 ± 2.4	11.0 ± 5.2	< 0.0001
GGT (U/l)	11.1 ± 11.3	19.2 ± 20.5	< 0.0001
AP (U/l)	80.0 ± 22.8	87.6 ± 22.5	< 0.0001
cholesterol (mmol/l)	5.4 ± 1.0	5.8 ± 1.1	< 0.0001
LDL (mmol/l)	3.2 ± 0.9	3.5 ± 1.0	< 0.0001
HDL (mmol/l)	1.6 ± 0.4	1.4 ± 0.4	< 0.0001
triglycerides (mmol/l)	1.3 ± 0.7	2.0 ± 1.0	< 0.0001
iron (µmol/l)	12.5 ± 6.3	12.0 ± 5.5	0.4221
CRP (mg/l)	2.2 ± 4.9	3.2 ± 4.7	< 0.0001
albumin (mg/l)	41.7 ± 4.3	41.3 ± 4.4	0.2181
fibrinogen (g/l)	3.1 ± 0.7	3.4 ± 0.8	< 0.0001
coeruloplasmin (g/l)	0.3 ± 0.1	0.3 ± 0.1	< 0.0001
Historical data	n (%)	n (%)	
soft drink consumption			
daily	39 (3.7)	10 (2.7)	0.3715
serveral times per week	87 (8.2)	27 (7.2)	
serveral times per month	103 (9.7)	32 (8.6)	
rarely/never	832 (78.4)	305 (81.6)	
coffee			
serveral times daily	203 (19.1)	70 (18.7)	0.0314
daily	583 (55.0)	233 (62.3)	
serveral times per week	88 (8.3)	26 (7.0)	
serveral times per month	45 (4.2)	6 (1.6)	
rarely/never	142 (13.4)	39 (10.4)	
sweets			
serveral times daily	57 (5.4)	11 (2.9)	< 0.0001
daily	313 (29.5)	76 (20.3)	
serveral times per week	345 (32.5)	136 (36.7)	
serveral times per month	200 (18.9)	72 (19.3)	
rarely/never	146 (13.8)	79 (21.1)	
diabetes mellitus			
no	1049 (98.9)	353 (94.4)	< 0.0001
yes	12 (1.1)	21 (5.6)	
hypertension			
no	953 (89.8)	268 (71.7)	< 0.0001
yes	108 (10.2)	106 (28.3)	
metabolic syndrome			
no	1040 (98.0)	312 (83.4)	< 0.0001
yes	21 (2.0)	62 (16.6)	

ALT: Alanine transaminase; AP: Alkaline phosphatase; AST: Aspartate transaminase; BMI: body-mass index; CRP: C-reactive protein; GGT: Gamma-glutamyltransferase; HDL: high-density lipoprotein; LDL: low-density lipoprotein; SD: standard deviation; WHR: waist-to-hip ratio

Table 1: **Demographics and other characteristics of subjects with and without hepatic steatosis**

Factors associated with hepatic steatosis included greater age ($p < 0.0001$), elevated BMI and WHR ($p < 0.0001$), concomitant diabetes mellitus, hypertension and metabolic syndrome ($p < 0.0001$; ◆ Table 1).

In the overall collective, 20.8 % (n = 298) reported consumption of soft drinks; of these, 23.1 % were diagnosed with hepatic steatosis (n = 69). The average age of soft drink consumers was significantly lower than the average age of those who reported never or only rarely consuming soft drinks ($p < 0.0001$). Young adults report the highest consumption of soft drinks; this consumption then declines with increasing age, and experiences a drastic drop around age 40 years (◆ Figure 1).

Among both sexes, soft drink consumption is reported by most respondents as "rarely/never"; still, there is a significant gender difference among consumers, with males predominating (◆ Figure 2). The average BMI figures for those reporting soft drink consumption differ only slightly and non-significantly from those of non-consumers ($p = 0.9399$). Values for WHR show no correlation to subjects' soft drink consumption ($p = 0.2375$).

The bivariate analysis revealed no significant association between soft drink consumption and an increased prevalence of hepatic steatosis. For males, however, a 2.4-fold increased risk of developing hepatic steatosis was identified. Factors such as advancing age, high BMI and WHR, and disorders such as diabetes mellitus, metabolic syndrome and hypertension also showed a significant association with the development of hepatic steatosis (◆ Table 2).

Considering the effect of soft drink consumption in a subgroup of individuals below 40 years, the results show also no significant association between soft drink consumption and hepatic steatosis for each age group (age 18–25, $p = 0.7980$; age 26–30, $p = 0.6886$; age 31–35, $p = 0.8418$; age 36–40, $p = 0.0714$).

Multivariate analysis confirmed a significant correlation between hepatic steatosis and sex, age, BMI and WHR. No significant association between consumption of sweets and soft drinks and hepatic steatosis was observed (◆ Table 3).

Discussion

In comparison with the relatively small collectives in the few available studies, which frequently were characterized by a predominance of males and attempted to study hepatic steatosis in patients treated in outpatient departments of hepatology or hospitals [8, 12], our collective consisted of 1,435 subjects (women, 54.6 %, n = 783; men, 45.4 %, n = 652; aged 18–65 years). Studies to date have revealed that men generally consume more soft drinks than do women [13]. The majority of current studies examined a variety of questions such as the relationship between weight gain and

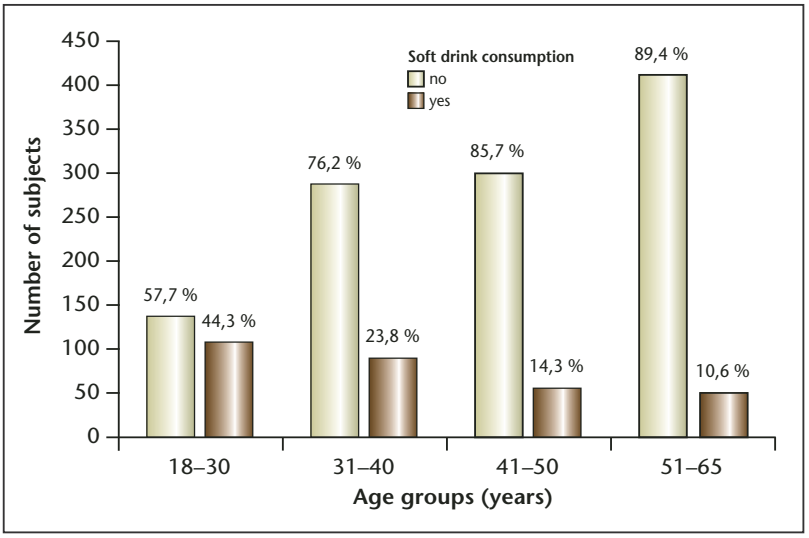

Figure 1: **Soft drink consumption of the study participants by age**

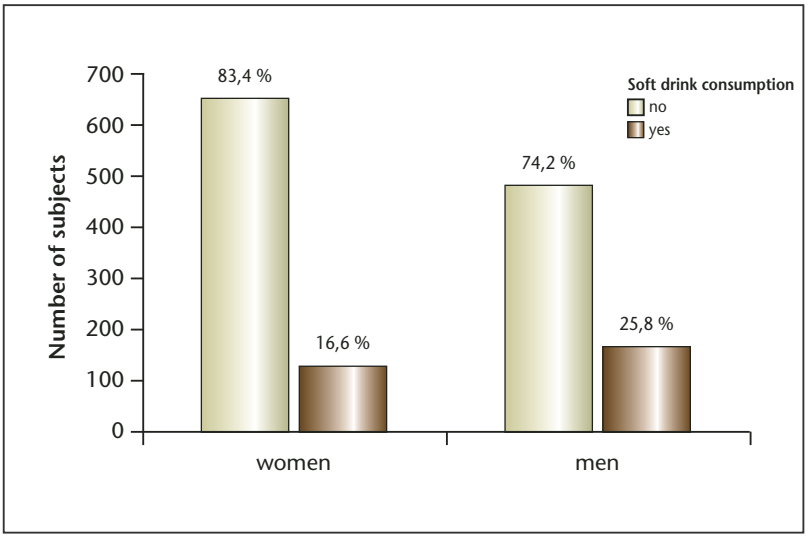

Figure 2: **Soft drink consumption of the study participants by sex**

soft drink consumption [14], soft drinks and childhood obesity [7], and soft drinks and general health [15]. Investigations into the prevalence of hepatic steatosis primarily focused on establishing an association with nutrition [16] and fructose consumption [17]. To our knowledge, the present study is the first to investigate the association between hepatic steatosis and soft drink consumption in a randomly selected population sample in Germany.

Corresponding to the findings of other population-based surveys, the prevalence of hepatic steatosis in the present study was 26.2 % [1, 16]. Of subjects with the sonographic diagnosis of hepatic steatosis, 18.4 % reported regular soft drink consumption (n = 69), compared with 21.6 % in those without evidence of steatosis. Neither the bivariate nor multivariate analysis revealed a significant correlation between soft drink consumption and an increased prevalence of hepatic steatosis.

Because soft drink consumption continues to rise in young adulthood and experiences a drastic drop around the age of 40 years, our average age does not fall within the age group with the highest consumption of soft drinks [18]. Data of the National Consumption Study II (NVS II) also show that males aged 14–18 years report the highest consumption of soft drinks [19].

	OR (95-%-CI)	p
sex		
female	ref.	ref.
male	2.346 (1.843–2.987)	< 0.0001
age (years)		
18–30	ref.	ref.
31–40	2.879 (1.668–4.967)	< 0.0001
41–50	3.632 (2.113–6.245)	< 0.0001
51–65	10.414 (6.230–17.407)	< 0.0001
BMI (m²/kg)		
< 25	ref.	ref.
25–30	10.224 (7.070–14.784)	< 0.0001
> 30	35.721 (23.441–54.435)	< 0.0001
WHR		
normal	ref.	ref.
elevated	7.271 (5.501–9.610)	< 0.0001
soft drink consumption		
daily	0.700 (0.345–1.419)	0.3219
several times/week	0.847 (0.539–1.329)	0.4695
several times/month	0.847 (0.558–1.287)	0.4376
rarely/never	ref.	ref.
metabolic syndrome		
no	ref.	ref.
yes	9.835 (5.902–16.390)	< 0.0001
hypertension		
no	ref.	ref.
yes	3.490 (2.585–4.713)	< 0.0001
diabetes		
no	ref.	ref.
yes	5.200 (2.533–10.677)	< 0.0001

95–%–CI = 95 %–confidence interval; BMI: body–mass index, OR = Odds Ratio; ref. = reference group; WHR: waist–to–hip ratio

Table 2: **Bivariate association between hepatic steatosis and other variables**

Compared with our study, which consisted of a single survey and examination, other studies have included longer observation periods, such as 1.5 years [5] or two periods of seven days over six months [12]. Seasonal differences with higher soft drink consumption in the summer and lower consumption in the winter may have impacted the findings of Assy et al. [8], Abid et al. [12] and our own data collected in late fall/early winter in different ways.

The impact of age on the development of hepatic steatosis was demonstrated in both bi- and multivariate analyses. Other studies have also shown that NAFLD is age-dependent and occurs less frequently in younger years [1]. For example, Kuhla et al. have shown that, with increasing age, the balance between lipolysis and lipogenesis shifts toward the latter, which may explain the increased prevalence of NAFLD among older persons [20]. Other studies confirm our findings of an age-dependent pattern of soft drink consumption, with a maximum in younger years and a decline with age [13, 18].

Both bi- and multivariate analysis revealed a significant association between elevated BMI and WHR and an increased risk of hepatic steatosis. However, as borne out by findings of Berkey et al. [21], there was no significant effect of soft drink consumption on BMI.

A possible explanation for the low soft drink consumption in our study collective may relate to the site of the survey which, based on its geographical situation and number of inhabitants, could be described as more rural in nature. Other studies have found differences between urban and rural populations in terms of nutrition, with and increased consumption of soft drinks and fast food [22] and of sugar, fat

		OR (95-%-CI)	p
sex			
	female	ref.	ref.
	male	2.558 (1.873–3.493)	< 0.0001
age (years)			
	18–30	ref.	ref.
	31–40	1.955 (1.035–3.694)	0.0390
	41–50	2.493 (1.305–4.763)	0.0057
	51–65	4.842 (2.585–9.068)	< 0.0001
BMI (m²/kg)			
	< 25	ref.	ref.
	25–30	6.109 (4.116–9.067)	< 0.0001
	> 30	20.797 (13.150–32.891)	< 0.0001
WHR			
	normal	ref.	ref.
	elevated	2.861 (1.914–3.757)	< 0.0001
sweets			
	daily	0.826 (0.524–1.304)	0.4118
	several times/week	0.823 (0.535–1.266)	0.3760
	several times/month	0.721 (0.442–1.176)	0.1903
	rarely/never	ref.	ref.
softdrinks			
	daily	0.578 (0.238–1.401)	0.2248
	several times/week	1.171 (0.638–2.149)	0.6098
	several times/month	0.984 (0.578–1.674)	0.9523
	rarely/never	ref.	ref.

95–%–CI = 95 %–confidence interval; BM = body–mass–index; OR = Odds Ratio; ref. = reference group; WHR = waist–to–hip–ratio

Table 3: **Multivariate association between hepatic steatosis and other variables**

and calories [23]. Rural populations tend to consume increased amounts of milk, fish and vegetables [22]. As these studies were conducted in Croatia and Pakistan, however, their findings may be difficult to apply to conditions in Germany. Traditional nutrition low in fast food [24], healthier eating [25] and generally healthier behaviors with three meals per day, adequate sleep and avoidance of excessive alcohol consumption [26] have been found in other studies to characterize rural life.

A strength of the present prospectively conducted cross-sectional study consists in its population representativeness: all subjects were randomly selected and all age classes from 10 to 65 years were included. In addition, the large number of participants and the balanced representation of men and women contribute to the importance of the study.

A weakness of the study with respect to soft drinks is that subjects were questioned regarding their frequency of soft drink consumption but the amount consumed in liters was not assessed. Also, there was no attempt to differentiate between diet and regular soft drinks or between different brands or manufacturers with different sugar contents. In addition, the fact that the study was conducted in the autumn and winter months may have impacted the study's findings secondary to a seasonal drop in soft drink consumption. A further weakness of our study relates to the low number of children, adolescents and young adults in the study collective. Soft drink consumption is highest in these groups; overweight in older subjects is not due to increased soft drink consumption.

Liver biopsy represents the gold standard for diagnosis of NAFLD, but this procedure was not suitable on both ethical and practical grounds for use in a population-based cross-sectional study [1].

Outlook

In conclusion, the findings of the present study show that the consumption of soft drinks is not associated with an increased prevalence of hepatic steatosis. The majority of subjects with and without the diagnosis of hepatic steatosis reported drinking soft drinks either rarely or never. Soft drinks are most frequently consumed by younger persons and become much less important beginning around the age of 40 years [18, 19]. Further prospective studies exactly quantifying the amount, frequency and type of consumed soft drinks will be required in order to better assess whether and, if so, to what degree soft drink consumption can be implicated in the development or prevalence of hepatic steatosis.

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Conflict of Interest
The authors declare no conflict of interest according to the guidelines of the International Committee of Medical Journal Editors.

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References

1. *Bellentani S, Marino M (2009) Epidemiology and natural history of non-alcoholic fatty liver disease (NAFLD). An Hepatol 1: 4–8*
2. *Cohen JC, Horton JD, Hobbs HH (2011) Human fatty liver disease: old questions and new insights. Science 332: 1519–1523*
3. *Hamaguchi M, Kojima T, Itoh Y, et al. (2007) The severity of ultrasonographic findings in nonalcoholic fatty liver disease reflects the metabolic syndrome and visceral fat accumulation. Am J Gastroenterol 102: 2708–2715*
4. *Vuilleumier S (1993) Worldwide production of high-fructose syrup and crystalline fructose. Am J Clin Nutr 58: 733–736*
5. *Nseir W, Nassar F, Assy N (2010) Soft drinks consumption and nonalcoholic fatty liver disease. World J Gastroenterol 16: 2579–2588*
6. *Havel PJ (2005): Dietary fructose implications for dysregulation of energy homeostasis and lipid/carbohydrate metabolism. Nutr Rev 63: 133–157*
7. *Ludwig DS, Peterson KE, Gortmaker SL (2001) Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. Lancet 357: 505–508*
8. *Assy N, Nasser G, Kamayse I et al. (1993) Soft drink consumption linked with fatty liver in the absence of traditional risk factors. Can J Gastroenterol 22: 811–816*
9. *Haenle MM, Brockmann SO, Kron M et al. (2006) Overweight, physical activity, tobacco and alcohol consumption in a cross-sectional random sample of German adults. BMC Public Health 6: 233*
10. *WHO (1995) Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. World Health Organ Tech Rep Ser 854: 1–4*
11. *Saverymuttu SH, Joseph AE, Maxwell JD (1986) Ultrasound scanning in the detection of hepatic fibrosis and steatosis. Br Med J (Clin Res Ed) 292: 13–15*
12. *Abid A, Taha O, Nseir W et al. (2009) Soft drink consumption is associated with fatty liver disease independent of metabolic syndrome. J Hepatol 51: 918–924*
13. *Rombaldi AJ, Neutzling MB, Silva MC et al. (2011) Factors associated with regular non-diet soft drink intake among adults in Pelotas, Southern Brazil. Rev Saude Publica 45: 382–390*
14. *Schulze MB, Manson JE, Ludwig DS et al. (2004) Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. JAMA 292: 927–934*
15. *Vartanian LR, Schwartz MB, Brownell KD (2007) Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. Am J Public Health 97: 667–675*
16. *Leclercq IA, Horsmans Y (2008) Nonalcoholic fatty liver disease: the potential role of nutritional management. Curr Opin Clin Nutr Metab Care 11: 766–773*
17. *Thuy S, Ladurner R, Volynets V et al. (2008) Nonalcoholic fatty liver disease in humans is associated with increased plasma endotoxin and plasminogen activator inhibitor 1 concentrations and with fructose intake. J Nutr 138: 1452–1455*
18. *Nasreddine L, Hwalla N, Sibai A et al. (2006) Food consumption patterns in an adult urban population in Beirut, Lebanon. Public Health Nutr 9: 194–203*
19. *Max Rubner-Institut Bundesforschungsinstitut für Ernährung und Lebensmittel (Hg). Nationale Verzehrstudie II (2009), S. 52–57*
20. *Kuhla A, Blei T, Jaster R et al. (2011) Aging is associated with a shift of fatty metabolism toward lipogenesis. J Gerontol A Biol Sci Med Sci 66: 1192–1200*
21. *Berkey CS, Rockett HR, Field AE et al. (2004) Sugar-added beverages and adolescent weight change. Obes Res 12: 778–788*
22. *Colic-Baric I, Kajfez R, Satalic Z et al. (2004) Comparison of dietary habits in the urban and rural Croatian schoolchildren. Eur J Nutr 43: 169–174*
23. *Hakeem R, Thomas J, Badruddin SH (2002) Food habits and nutrient density of diets of Pakistani children living in different urban and rural settings. J Health Popul Nut 20: 255–263*
24. *Lazarou C, Kalavana T (2009) Urbanization influences dietary habits of Cypriot children: the CYKIDS study. Int J Public Health 54: 69–77*
25. *Morgan K, Armstrong GK, Huppert FA et al. (2000) Healthy ageing in urban and rural Britain: a comparison of exercise and diet. Age Ageing 29: 341–348*
26. *Johnson JL, Ratner PA, Bottorff JL (1995) Urban-rural differences in the health-promoting behaviours of Albertans. Can J Public Health 86: 103–108*

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