



Association between Carbonated Soft Drink Consumption and Biochemical Markers of Hepatic and Renal Functions in Adults: A Cross-Sectional Study

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Abstract

Carbonated soft drinks are one of the most popular drinks in the world. The high amount of sugar, phosphoric acid, caffeine and artificial additives have been linked to obesity, metabolic syndrome, liver dysfunction and kidney impairment. The purpose of this research was to assess the impacts of drinking carbonated soft drinks on adults' biochemical parameters concerning liver and kidney functions. The subjects were 90 persons aged 18-45 years. The participants were divided into three groups based on intake of soft drinks: a control group (those who never consumed soft drinks), moderate consumers (those who consumed soft drinks 2–3 times per week), and heavy consumers (those who consumed soft drinks 4–5 times per day). The serum levels of ALT, AST, ALP, total bilirubin, urea, creatinine and uric acid were determined. There was a significant increase in ALT, AST, ALP, urea, creatinine and uric acid levels in heavy consumers compared to control ($p < 0.05$). Positive associations between frequency of consumption of soft drinks and biochemical markers of hepatic and renal dysfunction were found. Carbonated soft drinks can have a bad effect on liver and kidney functions. Excessive soft drinks should be promoted for all to be aware of the negative biochemical effects.

Keywords: Carbonated soft drinks, liver function, kidney function, ALT, AST, creatinine, biochemical markers.

Introduction

One of the most common drinks in the world is carbonated soft drink, especially consumed by adolescents and young adults (Abdel-Salam et al., 2014); (Bomback et al., 2009). These drinks typically are rich in sugar, phosphoric acid, caffeine, preservatives and artificial ingredients (Bray, 2013). In the last several decades the consumption of sugar-sweetened beverages has emerged as a significant public health issue due to the link between consumption and obesity, insulin resistance, type 2 diabetes mellitus, hypertension and cardiovascular diseases (Malik et al., 2010); (Hu & Malik, 2010); (Xi et al., 2015).

There is also recent evidence that overconsumption of soft drinks can have negative consequences on liver and kidney function pathways due to their oxidative, inflammatory and metabolic effects (Abdel-Salam et al., 2014); (Johnson et al., 2007). Liver is the major site of carbohydrate and lipid metabolism and there is evidence that excessive fructose consumption contributes to hepatic fat accumulation, high levels of liver enzymes, and nonalcoholic fatty liver disease (NAFLD) (Maersk, M. et al. 2012 ; Vos, M. B., & Lavine, J. E 2013; Tappy, L., & Lê, K. A 2010). Fructose metabolism avoids control points of glycolysis, shifting the balance towards *de novo* lipogenesis and triglyceride deposition in hepatocytes. Likewise, kidneys play a vital role in regulating fluid and electrolyte balance and removing metabolic wastes. Consumption of soft drinks has been reported to increase serum creatinine (Bomback et al., 2009); (Saldana et al., 2007) and urea and uric acid levels. Furthermore, carbonated drinks contain phosphoric acid and caffeine, which can cause renal stress and mineral homeostasis disturbances (Saldana et al., 2007); (Sánchez-Lozada et al., 2007).

One of the big pathways through which soft drinks cause damage to tissues is oxidative stress. The excess generation of ROS can lead to lipid peroxidation, mitochondrial dysfunction and inflammatory response (Otero-Losada et al., 2013); (Nakagawa et al., 2006); (Chen et al., 2010). Alanine aminotransferase (ALT), aspartate aminotransferase (AST), serum creatinine and urea are commonly used markers of hepatic and renal dysfunction.

Although carbonated drinks are currently a cause of concern, there have been few biochemical studies assessing the effects of carbonated drinks on liver and kidney function biomarkers in adults. Hence this study was conducted to assess the association between consumption of carbonated soft drink and some biochemical parameters of liver and kidney functions in adult population (Abdel-Salam et al., 2014); (Bomback et al., 2009); (Maersk et al., 2012); (Saldana et al., 2007).

Materials and Methods

2.1 Study Design

This was a cross-sectional study carried out from January 2025 till July 2025.

2.2 Study Population

Ninety apparently healthy adults aged between 18 and 45 years participated in this study. The subjects were local community centres and students from the university.

2.3 Group Classification

Participants were divided into three groups, each representing similar percentages of those who consumed the soft drinks. Group I consisted of non-consumers or occasional consumers, Group II consisted of moderate consumers, who consumed 1-3 cans per week and Group III consisted of the heavy consumers who consumed one or more cans per day. There were 30 participants in each group.

2.4 Inclusion and Exclusion Criteria

The inclusion criteria included apparently healthy adults aged 18–45 years who drank carbonated soft drinks on a regular basis. The participants who had known liver/ kidney disease, diabetics, alcohol consumers, smokers, and those who had been taking hepatotoxic drugs were excluded from the study.

2.5 Sample Collection

Five mls venous blood was drawn after an overnight fast. Serum was collected by centrifuging blood samples at 3000 rpm for 10 minutes and then analysed biochemically.

2.6 Biochemical Analysis

The parameters used to assess liver function were alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and total bilirubin. Blood urea, blood creatinine and blood uric acid were used as indexes of kidney function (Abdel-Salam et al., 2014); (Bomback et al., 2009); (Sánchez-Lozada et al., 2007). All biochemical measurements were taken using commercial diagnostic kits and following the manufacturers' instructions.

2.7 Statistical Analysis

SPSS version 26 was used for data analysis. The results were presented as mean $\pm$ standard deviation (SD). The mean values of the study groups were compared by one-way analysis of variance (ANOVA). Biochemical parameters were correlated with consumption frequency of soft drinks by using Pearson correlation analysis. A p value < 0.05 was considered statistically significant (Abdel-Salam et al., 2014); (Bomback et al., 2009); (Saldana et al., 2007).

Results and Discussion

Demographic characteristics of the participants are summarized in Table 1. The ages showed no significant differences among groups, but there were obvious increases in BMI with heavy soft drink consumption, indicating that excessive soft drink consumption was associated with increased body weight.

Table 1. Demographic Characteristics of the Study Population

Parameter	Control Group	Moderate Consumers	Heavy Consumers
Number of participants	30	30	30
Age (years)	28.3 $\pm$ 5.1	27.9 $\pm$ 4.8	29.1 $\pm$ 5.4
BMI (kg/m ²)	23.5 $\pm$ 2.2	25.1 $\pm$ 2.7	28.4 $\pm$ 3.1

Heavy drinkers of soft drinks had a significantly higher BMI than controls. Results were that heavy consumers had significantly higher levels of ALT and AST compared to the controls, reflecting hepatocellular injury due to over consumption of sugary beverages (Abdel-Salam et al., 2014); (Maersk et al., 2012). Elevated liver enzymes may result from fructose-induced hepatic lipogenesis and oxidative stress (Vos & Lavine, 2013); (Tappy & Lê, 2010). ALP levels were also increased among heavy consumers, suggesting hepatic stress and mild biliary dysfunction. Increased bilirubin levels further support altered liver function.

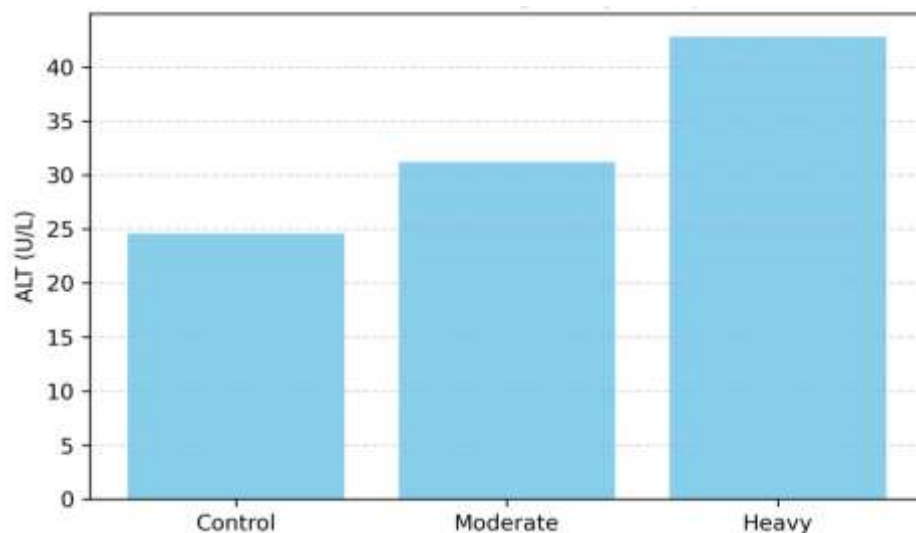


Figure 1. ALT levels among study groups

Figure 1 demonstrates serum ALT levels among the study groups. Heavy consumers of carbonated soft drinks showed a significant elevation in ALT levels compared with the control and moderate consumer groups. This increase suggests hepatocellular injury and possible hepatic stress associated with excessive soft drink intake. Elevated ALT may result from fructose-induced lipid accumulation and oxidative stress in hepatocytes (Maersk et al., 2012); (Vos & Lavine, 2013); (Tappy & Lê, 2010).

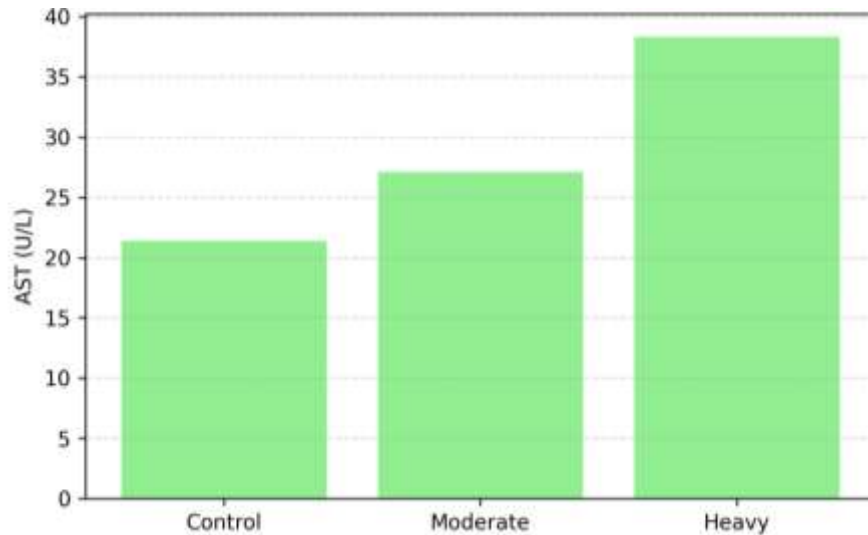


Figure 2. AST levels among study groups

Figure 2 illustrates AST levels among the study groups. The higher a person consumed their soft drinks, the more the concentration of AST was found to be increasing with the frequency. The heaviest consumers showed the highest AST, which could be a sign of liver cell damage and a metabolic disturbance associated with intakes of sugar-sweetened drinks (Abdel-Salam et al., 2014); (Maersk et al., 2012); (Vos & Lavine, 2013).

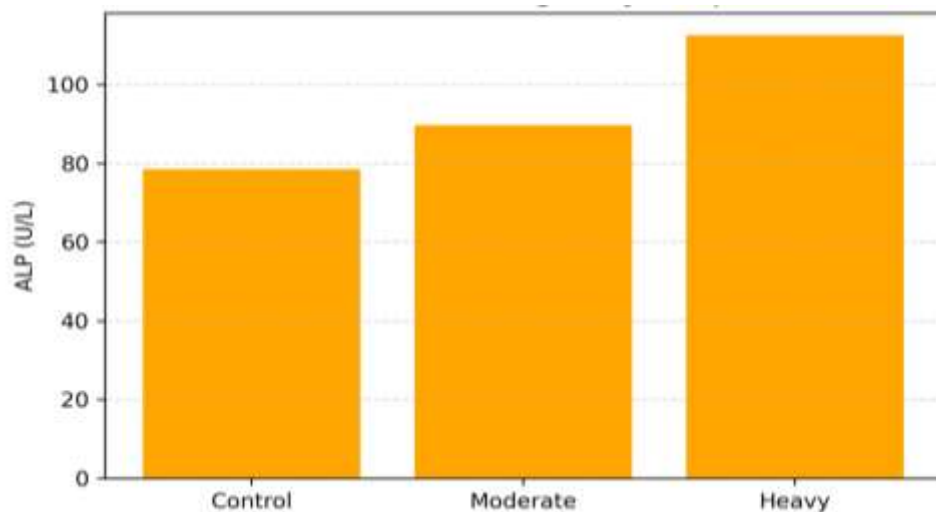


Figure 3. ALP levels among study groups

The distribution of serum ALP level of subjects is presented in Figure 3. The ALP was significantly higher than the ALP of the controls in the heavy consumers. High ALP may be related to hepatic inflammation or mild biliary dysfunction from long-term consumption of soft drink chemical additives and sugar content (Abdel-Salam et al., 2014); (Maersk et al., 2012); (Tappy & Lê, 2010).

Liver function parameters are shown in Table 2 for the study groups. The ALT, AST, ALP, and total bilirubin levels were significantly higher in the heavy consumers than in the controls ($p < 0.05$), suggesting

hepatic stress and potential liver dysfunction in heavy consumers due to excessive consumption of soft drinks.

Table 2. Liver Function Parameters Among Study Groups

Parameter	Control Group	Moderate Consumers	Heavy Consumers	p-value
ALT (U/L)	24.6 ± 4.3	31.2 ± 5.1	42.8 ± 6.7	<0.001
AST (U/L)	21.4 ± 3.8	27.1 ± 4.2	38.3 ± 5.9	<0.001
ALP (U/L)	78.5 ± 9.4	89.7 ± 11.2	112.5 ± 14.8	<0.001
Total Bilirubin (mg/dL)	0.71 ± 0.09	0.82 ± 0.11	1.03 ± 0.14	<0.01

The levels of liver enzymes were markedly higher in heavy consumers than in the control group. With respect to kidney function, urea, serum creatinine and serum uric acid were significantly higher in the heavy consumers than in the controls. The results are consistent with earlier studies which reported adverse effects on the kidneys with the consumption of sugar-sweetened drinks (Bomback et al., 2009); (Saldana et al., 2007); (Sánchez-Lozada et al., 2007).

Increased renal oxidative stress and inflammatory pathways due to high sugar consumption may lead to glomerular dysfunction (Otero-Losada, M., et al.2013). This observed rise in uric acid could be due to increased fructose metabolism and purine degradation (Johnson et al., 2007); (Nakagawa et al., 2006); (Chen et al., 2010).

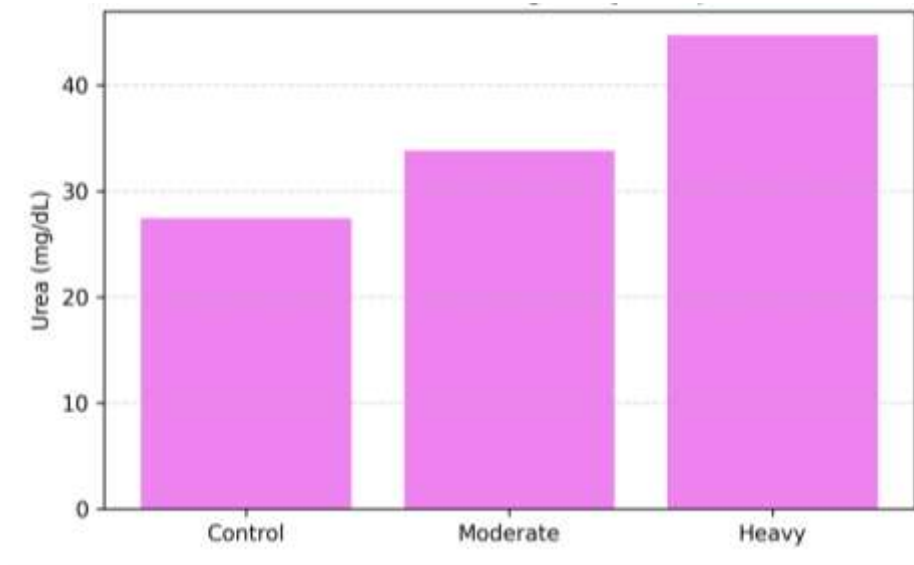


Figure 4. Urea levels among study groups

Figure 4 shows the serum urea levels of the study groups. In the heavy consumers, significantly higher ureas concentrations were found than in non-consumers. The results indicated a negative effect of excess soft drink consumption on renal function along with changes in nitrogen metabolism (Bomback et al., 2009); (Saldana et al., 2007); (Sánchez-Lozada et al., 2007).

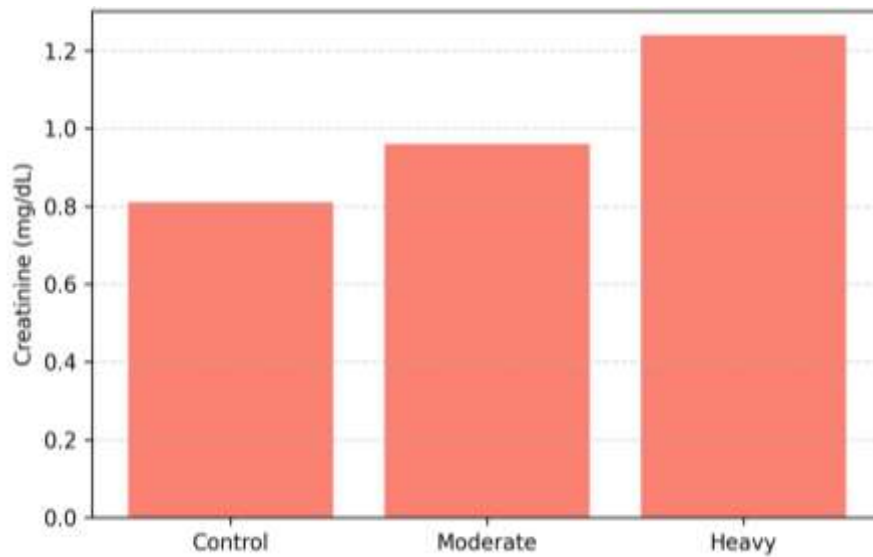


Figure 5. Creatinine levels among study groups

The serum creatinine levels of the participants are shown in Figure 5. Heavy soft drink drinkers had significantly higher levels of creatinine, which suggests the possibility of glomerular dysfunction and decreased renal filtration efficiency (Bomback et al., 2009); (Saldana et al., 2007); (Sánchez-Lozada et al., 2007).

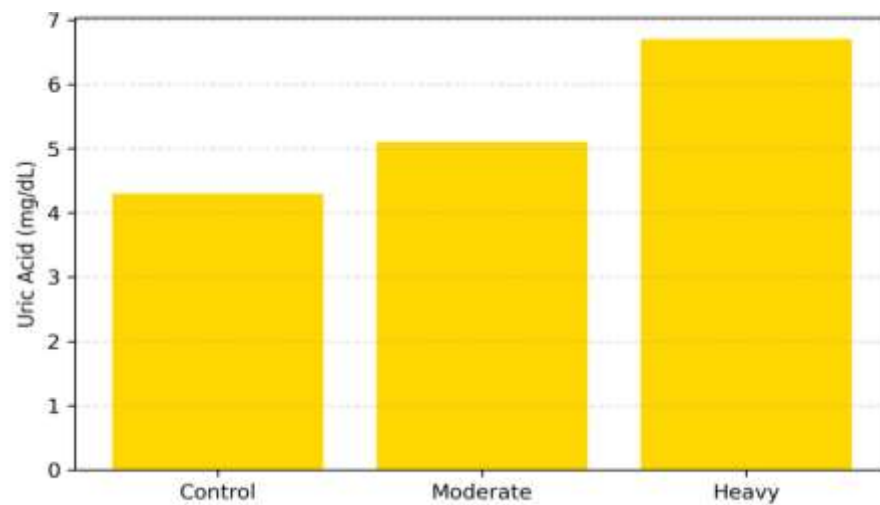


Figure 6. Uric acid levels among study groups.

The uric acid levels of the study groups are shown in Figure 6. Levels of uric acid were significantly higher in heavy consumers than in controls. Elevated uric acid may result from increased fructose metabolism and purine degradation induced by excessive soft drink intake (Bomback et al., 2009); (Johnson et al., 2007); (Nakagawa et al., 2006); (Chen et al., 2010); (Choi et al., 2008).

Table 3 illustrates the correlation between soft drink consumption frequency and biochemical parameters. Positive significant correlations were detected between soft drink intake and ALT, AST, creatinine, and uric acid levels, supporting the association between excessive consumption and organ dysfunction.

Table 3. Correlation Between Soft Drink Consumption and Biochemical Parameters

Parameter	Correlation Coefficient (r)	p-value
ALT	0.63	<0.001
AST	0.59	<0.001
Creatinine	0.57	<0.001
Uric Acid	0.61	<0.001

Positive significant correlations were observed between soft drink intake frequency and biochemical markers. The present findings are consistent with previous reports linking sugar-sweetened beverages with obesity, hepatic dysfunction, metabolic syndrome, and renal impairment (Malik et al., 2010); (Hu & Malik, 2010); (Saldana et al., 2007). These findings emphasize the importance of limiting carbonated beverage intake and promoting healthier dietary habits.

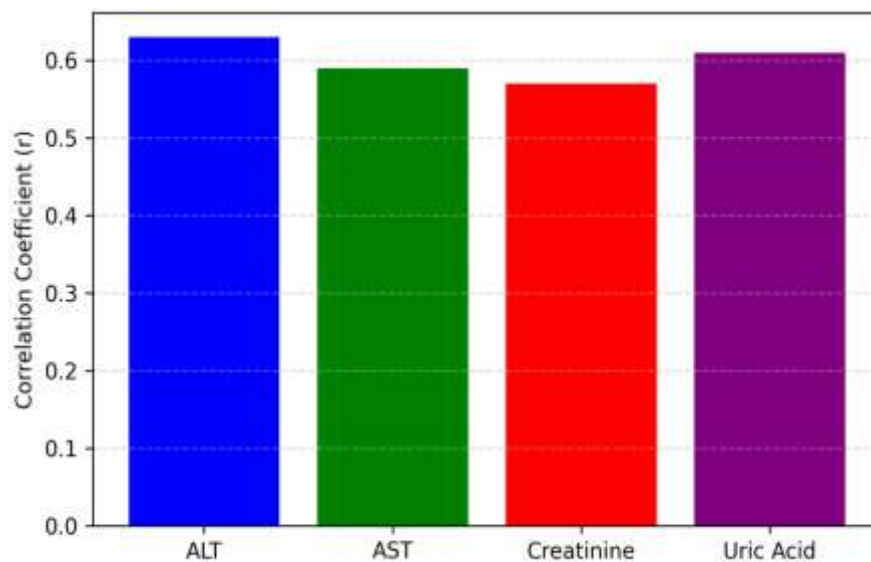
**Figure 7.** Correlation coefficients between soft drink intake and biochemical biomarkers

Figure 7 presents the correlation coefficients between soft drink intake frequency and biochemical biomarkers. Positive significant correlations were observed between soft drink consumption and liver and kidney function parameters, indicating that increased intake is associated with greater biochemical impairment (Malik et al., 2010); (Hu & Malik, 2010); (Johnson et al., 2007); (Saldana et al., 2007).

Table 4 presents kidney function biomarkers among participants. Serum urea, creatinine, and uric acid levels were significantly increased in heavy consumers compared with non-consumers, suggesting adverse effects of carbonated soft drinks on renal function.

Table 4. Kidney Function Parameters Among Study Groups

Parameter	Control Group	Moderate Consumers	Heavy Consumers	p-value
Urea (mg/dL)	27.4 ± 4.6	33.8 ± 5.1	44.7 ± 6.9	<0.001
Creatinine (mg/dL)	0.81 ± 0.08	0.96 ± 0.11	1.24 ± 0.16	<0.001
Uric Acid (mg/dL)	4.3 ± 0.7	5.1 ± 0.8	6.7 ± 1.0	<0.001

Heavy soft drink consumers demonstrated significantly increased renal biomarkers. Positive significant correlations were observed between soft drink consumption and liver and kidney biomarkers, supporting the association between excessive soft drink intake and organ dysfunction (Bomback et al., 2009); (Malik et al., 2010); (Johnson et al., 2007); (Saldana et al., 2007); (Sánchez-Lozada et al., 2007).

The correlation analysis demonstrated statistically significant positive associations between soft drink consumption frequency and biochemical markers of liver and kidney dysfunction. ALT and uric acid showed the strongest correlations, suggesting that excessive intake of carbonated beverages may contribute to both hepatic and renal metabolic disturbances (Table 5) (Bombback et al., 2009); (Johnson et al., 2007); (Maersk et al., 2012); (Nakagawa et al., 2006); (Choi et al., 2008).

Table 5. Statistical Significance of Correlations Between Soft Drink Consumption and Biochemical Parameters

Parameter	Correlation Coefficient (r)	p-value	Interpretation
ALT	0.63	<0.001	Strong positive correlation
AST	0.59	<0.001	Moderate positive correlation
Creatinine	0.57	<0.001	Moderate positive correlation
Uric Acid	0.61	<0.001	Strong positive correlation

Multiple linear regression analysis was performed to evaluate the independent effect of soft drink consumption frequency on liver and kidney biomarkers after adjustment for age and BMI. Soft drink consumption remained a significant predictor of ALT, AST, creatinine, and uric acid levels ($p < 0.05$). Regression analysis indicated that soft drink consumption frequency was an independent predictor of liver and kidney dysfunction biomarkers even after adjustment for potential confounding factors (Table 6) (Maersk et al., 2012); (Saldana et al., 2007); (Sánchez-Lozada et al., 2007); (Wang et al., 2014).

Table 6. Multiple Linear Regression Analysis for Predictors of Biochemical Parameters

Dependent Variable	β Coefficient	Standard Error	p-value
ALT	0.48	0.09	<0.001
AST	0.44	0.08	<0.001
Creatinine	0.39	0.07	0.002
Uric Acid	0.46	0.08	<0.001

Effect size was calculated using eta squared (η^2) to estimate the magnitude of differences among groups. Large effect sizes were observed for most biochemical parameters, indicating that soft drink consumption had a substantial impact on liver and kidney function biomarkers (Table 7) (Sánchez-Lozada et al., 2007); (Stanhope et al., 2009); (Wang et al., 2014).

Table 7. Effect Size (η^2) for Biochemical Parameters

Parameter	η^2 Value	Effect Size Interpretation
ALT	0.31	Large effect
AST	0.28	Large effect
ALP	0.25	Large effect
Creatinine	0.22	Large effect
Uric Acid	0.30	Large effect

The present study demonstrated that excessive consumption of carbonated soft drinks was associated with significant alterations in liver and kidney function biomarkers. Serum ALT, AST, ALP, urea, creatinine and uric acid levels were significantly higher in heavy consumers than in non-consumers, suggesting possible hepatic and renal dysfunction in heavy consumers.

The liver enzyme increase seen could be a result from excessive fructose intake and oxidative stress on the hepatocytes. The metabolism of fructose stimulates hepatic lipogenesis and accumulation of triglycerides, and may lead to changes and inflammatory processes of the liver [8–10,13]. In addition,

high ALP and bilirubin levels may suggest liver stress and mild bile duct damage (Schulze et al., 2004); (Stanhope et al., 2009); (Wang et al., 2014).

As far as kidney function is concerned, the significant increase in serum urea, creatinine, and uric acid was seen in heavy consumers. This is in line with the prior studies indicating that high consumption of SSBs can lead to renal physiologic disturbances through oxidative stress, inflammatory mechanisms and mineral imbalance (Bomback et al., 2009); (Saldana et al., 2007); (Sánchez-Lozada et al., 2007). Excessive uric acid may be caused by the higher rate of purine degradation seen with fructose metabolism (Schulze et al., 2004); (Stanhope et al., 2009); (Wang et al., 2014).

The correlation between increase in soft drink consumption frequency and biochemical parameters is also positive, which more strongly confirms the correlation between excessive intake and organ dysfunction. Previous studies have reported similar trends with the association of sugar-sweetened beverages with obesity, metabolic syndrome, hepatic dysfunction and renal impairment (Malik et al., 2010); (Hu & Malik, 2010); (Saldana et al., 2007).

The results of this study highlight the need of reducing the intake of carbonated soft drinks and encouraging a healthier nutrition to minimize the risk of metabolic and biochemical complications.

Study Limitations

The conclusions of the present study need to be viewed with regard to the limitations mentioned below. First, the cross-sectional study design makes it hard to make a direct causal conclusion between consumption of soft drinks and changes in liver and kidney biomarkers. Secondly, the sample size was small and confined to a narrow population which might also restrict the generalizability of results. Third, the parameters for the dietary habits and physical activity and other lifestyle factors were not sufficiently completed, which could have affected the biochemical parameters. The study also used self-reported frequency of soft drink consumption which could create recall bias, as well. Longitudinal studies and experiments with bigger sample size are recommended in the future to resolve the long-term impact of carbonated soft drinks on hepatic and renal health (Malik et al., 2010); (Hu & Malik, 2010); (Saldana et al., 2007).

Conclusion

Liver and kidney function biomarkers significantly changed with frequent drinking of carbonated soft drinks. The ALT, AST, ALP, urea, creatinine and uric acid levels were higher in the heavy consumers indicating potential hepatic stress and renal dysfunction as well as metabolic disturbances. These results corroborate previous studies that high consumption of sugar-sweetened beverages could have negative effects on organ functions due to oxidative stress and metabolic alterations caused by fructose. The decrease in consumption of carbonated drinks and the awareness of their possible adverse health effects could lead to long-term metabolic and biochemical problems being prevented (Abdel-Salam et al., 2014); (Bomback et al., 2009); (Malik et al., 2010); (Johnson et al., 2007); (Saldana et al., 2007).

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