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Research Article

The Effect of Chronic Caffeinated Energy Drink Consumption on Insulin Sensitivity and Blood Glucose Regulation: A Case–Control Study

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ABSTRACT

Caffeinated energy drinks are rich in sugar and caffeine, which can disrupt the normal glycemic metabolism, elevate insulin resistance, and increase the risk of diabetes and other metabolic disorders, among habitual consumers. The current case–control study examined the effect of long-term caffeinated energy drink consumption on glucose regulation and insulin sensitivity. It involved 110 participants (60 cases and 50 controls) recruited from young college students; chronic energy drink consumers were considered cases. After informed consent, whole blood and serum samples were analyzed for hemoglobin A1C (HbA1c), fasting glucose, insulin, and later homeostatic model assessment for insulin resistance (HOMA-IR) was calculated. There were no significant differences in sex, age, fasting blood sugar (FBS), or HbA1c between groups ($P > 0.005$). However, fasting insulin and HOMA-IR levels were significantly higher in cases, indicating greater insulin resistance ($P < 0.001$). There was a significant positive correlation between HOMA-IR and fasting insulin, FBS, and HbA1c. These associations show that insulin resistance is linked to elevated glucose parameters ($P < 0.001$). This study concludes that chronic energy drink consumption is associated with increased insulin resistance. Future studies should explore long-term effects on pancreatic function, glucose tolerance, and diabetic risk in larger, diverse populations, with particular emphasis on longitudinal assessments to delineate the trajectory from insulin resistance to beta-cell dysfunction and the potential for synergistic effects with other dietary and lifestyle factors.

Keywords: Caffeinated energy drinks, insulin resistance, glucose regulation, homeostatic model assessment for insulin resistance, hemoglobin A1C

INTRODUCTION

Caffeinated energy drinks have gained significant popularity, particularly among young athletes and adults; nevertheless, there are major concerns about how they may disrupt blood glucose regulation.[1] These beverages often contain high amounts of caffeine and sugar, which alter metabolic processes. As a central nervous system stimulant, caffeine influences glucose metabolism by boosting catecholamine secretion, which accelerates hepatic glycogenolysis while decreasing insulin sensitivity.[2] This process can cause a temporary increase in fasting blood sugar (FBS) levels. Furthermore, the considerable sugar content of energy beverages leads to postprandial hyperglycemia, which results in frequent insulin spikes. With long-term consumption, the risk of decreased glucose tolerance and insulin resistance, hence diabetes, increases due to their disruptive effect on pancreatic beta cells.[3]

Insulin resistance is a condition that develops when the cells become less responsive and less sensitive to insulin, and the body requires higher insulin levels to preserve the normal glucose homeostasis. The increased blood glucose levels (hyperglycemia) develop from prolonged caffeine-induced hepatic glycogenolysis along with insulin resistance, and this eventually may lead to a vicious cycle of metabolic dysregulation.[4] The above-mentioned phenomenon develops as caffeine antagonizes adenosine receptors due to their structural similarities; adenosine typically induces insulin sensitivity. Consequently, the body would require more insulin

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in order to preserve glucose homeostasis. Caffeine and sugar also worsen metabolic dysfunction, which makes it more difficult to properly control blood glucose. Individuals with pre-diabetes should be particularly concerned about this, as it can induce the development of type 2 diabetes.[5,6]

The chronic hyperglycemia and the resulting hyperinsulinemia can raise the risk of cardiovascular disease, metabolic syndrome, and other complications over time.[7,8] Furthermore, excessive consumption of caffeine may interfere with sleep cycles, which is another reason for increased insulin resistance and glucose dysregulation.[8] Sleep deprivation is associated with elevated cortisol levels, which disrupts glucose metabolism. This hormonal imbalance results in sustained hyperglycemia, reduced insulin sensitivity, and decreased glucose uptake by the cells in peripheral tissues over time.[9]

It has been reported that frequent consumption of caffeinated energy drinks can elevate hemoglobin A1C (HbA1c) values, a parameter used to represent the average blood glucose levels in the previous 2–3 months, which provides an overall view of glucose control.[4] The present study was conducted to investigate the effect of long-term consumption of caffeinated energy drinks on glucose regulation and insulin sensitivity among habitual consumers. It was done by assessing fasting blood glucose levels, HbA1c, and homeostatic model assessment for insulin resistance (HOMA-IR) and comparing them with normal controls.

MATERIALS AND METHODS

The current case–control study was done by recruiting a total of 110 subjects who were divided into two groups: The cases, 60 (49 males and 11 females) who were consumers of energy drinks, and controls, 50 (41 males and 9 females) who were non-consumers. The cases were enrolled based on the duration of their energy drink consumption; they were required to be consumers for at least 1 year. The subjects were all matched based on their body mass index and physical activity, and were informed about the aim of the study, and were assured that their personal and medical information would remain confidential. Consents were obtained from them, and they were given the right to withdraw. The cases were enrolled based on the criteria that they were chronic (>6 months) consumers of caffeinated energy drinks on a daily basis. Control subjects were selected from non-consumers. To minimize the influence of genetic factors on glucose regulation, individuals with a family history of diabetes, as well as those taking medications that affect blood glucose levels, were excluded from both groups.

The majority of the subjects were college students and were recruited from Cihan University-Erbil and Salahaddin University-Erbil, Iraq. They were given systematic questionnaires and interviewed to record their personal and medical information. The control subjects were enrolled following the cases to make sure they were age- and gender-matched. The participants were required to fast, blood samples were obtained from them, and were collected into ethylenediaminetetraacetic acid (EDTA) and gel tubes. The samples were promptly transferred to a private laboratory to be tested. The whole blood samples from the EDTA tubes were used to assess HbA1c values. The gel tubes were used to get serum following centrifugation, and they were used to test fasting blood glucose (mg/dL) and fasting insulin levels (μU/mL). The HOMA-IR was used to assess insulin sensitivity; it was calculated as below, and values >2.7 were considered abnormal: [10]

HOMA-IR% = Fasting insulin (μU/mL) × Fasting blood sugar (mg/dL)/405 × 100

Statistical analysis was done using the Statistical Package for the Social Sciences (SPSS), and an independent t-test and correlations were used for data analysis. $P < 0.05$ was considered statistically significant, whereas $P \geq 0.05$ was considered statistically insignificant. Statistical analysis was done using SPSS. The independent samples t-tests were applied to assess differences in continuous variables between the two tested groups. At the same time, Pearson correlation (PC) analysis was used to investigate the potential associations between selected glycemic metabolic parameters. *P*-value threshold of lower than 0.05 was considered to indicate statistical significance, suggesting a <5% probability that the observed results occurred by chance. Conversely, $P > 0.05$ was interpreted as statistically insignificant, suggesting insufficient evidence to reject the null hypothesis. All statistical tests were two-tailed unless otherwise specified.

RESULTS

The present case–control study was done on a total of 110 subjects who were divided into two groups. The case group consisted of 49 (81.7%) males and 11 (18.3%) females. The control group was composed of 41 (82%) males and 9 (18%) females. There was no significant difference between the two groups regarding sex distribution ($P < 0.001$). The mean ± standard deviation (SD) age of the subjects was (22.367 ± 1.178) and (22.180 ± 1.257) years for the cases and controls, respectively. Statistical analysis showed no significant difference in the age of the subjects between the two groups ($P = 0.681$).

Table 1 shows that the mean with SD of FBS level (mg/dL) was (96.535 ± 6.535) among the cases, whereas it was (86.218 ± 6.385) in the control group. The higher mean FBS in the case group indicates slightly elevated blood sugar levels compared to the control group. Statistical analysis revealed that the difference in FBS between cases and controls was not significant ($P = 0.578$).

Regarding the fasting insulin level (μU/mL), the mean and SD were (14.724 ± 5.252) among the cases, whereas it was (9.570 ± 2.519) in the control group. The cases had significantly higher fasting insulin levels ($P < 0.001$). As for the HOMA-IR (%), which is the parameter for insulin resistance, the mean and SD were (3.543 ± 1.366) among the cases, whereas it was (2.036 ± 0.562) in the control group. The higher mean HOMA-IR among the cases suggests a greater insulin resistance compared to controls ($P < 0.001$).

Regarding the HbA1c (%), the parameter of sugar control over the past 2–3 months, the mean and SD were (5.434 ± 0.411) among the cases, whereas it was (5.059 ± 0.479) in the control group. Despite the higher mean of HbA1c values among the cases, the statistical analysis showed no significant difference ($P < 0.055$).

Table 1: Glycemic metabolic parameters

Parameter	Cases (n=60)	Controls (n=50)	P-value	Interpretation
FBS (mg/dL)	96.535±6.535	86.218±6.385	0.578	Not significant (P>0.05). No significant difference in FBS between cases and controls.
Insulin (μU/mL)	14.724±5.252	9.570±2.519	<0.001	Highly significant (P<0.001). Cases have significantly higher fasting insulin, suggesting insulin resistance.
HOMA-IR (%)	3.543±1.366	2.036±0.562	<0.001	Highly significant (P<0.001). Cases have significantly higher insulin resistance.
HbA1c (%)	5.434±0.411	5.059±0.479	0.055	Not significant (P>0.05). Slightly higher HbA1c in cases, but not statistically significant.

HbA1c: Hemoglobin A1C, HOMA-IR: Homeostatic model assessment of insulin resistance, FBS: Fasting blood sugar

The scatter plot shows the association between fasting insulin levels (μU/mL) and HOMA-IR (%) with a strong positive correlation (PC: 0.982). Statistical analysis revealed a significant association (P < 0.001).

The scatter plot shows the relationship between FBS levels (mg/dL) and HOMA-IR (%) with a positive correlation (PC: 0.646). Statistical analysis was highly significant (P < 0.001).

The scatter plot illustrates the link between HbA1c (%) and HOMA-IR (%) with a positive correlation (PC: 0.406). Statistical analysis revealed a highly significant association (P < 0.001).

The scatter plot shows there was a positive correlation between HbA1c (%) and FBS (mg/dL) with (PC: 0.456). HbA1c values increased with elevation of FBS levels, indicating their positive association. Statistical analysis revealed a significant association (P < 0.001).

DISCUSSION

This case–control study was conducted to investigate the effect of long-term consumption of caffeinated energy drinks on glucose regulation and insulin resistance among chronic consumers. It was done by assessing fasting blood glucose levels, HbA1c, and HOMA-IR and comparing them with normal controls. The findings of this study indicate that the mean ± SD fasting insulin levels were significantly elevated in the cases compared to the control group (P < 0.001). This indicates a heightened insulin secretion response in these cases, potentially indicating an underlying metabolic dysregulation or compensatory mechanism in response to insulin resistance. This finding is in line with other studies done, which indicate the negative effect of caffeine on insulin levels.[3,11,12]

In addition, the HOMA-IR values, which are used as a measure of insulin resistance, were shown to be significantly higher in the cases compared to the controls (P < 0.001). This difference supports the existence of developing insulin resistance among the cases, as indicated by the elevated HOMA-IR values, which reflect a decreased responsiveness of the cells to insulin, hence glucose metabolism dysfunction. These results align with previous studies that indicate the association between increased fasting hyperinsulinemia and insulin resistance.[5,13]

The mean HbA1c values were slightly greater in the cases compared to the controls, with a P = 0.055. Although this difference was not statistically significant, the upward trend possibly indicates early alterations in glycemic regulation among the case group, which may reflect the initial phase of metabolic dysregulation. Furthermore, the mean FBS levels were higher in the cases in comparison to the controls, with a P = 0.578, revealing no significant difference between the groups. These results suggest that standard glycemic markers such as FBS and HbA1c may be insufficiently sensitive to detect early metabolic abnormalities, particularly in non-diabetic patients. This emphasizes the significance of including additional measures, such as fasting insulin and HOMA-IR values, to obtain a greater picture of underlying hyperinsulinemia and metabolic dysfunction risk. Our results are in line with another study that reported no significant effect of caffeine on HbA1c.[14,15] In contacts to our results, other studies have reported a significant association between elevated HbA1c and habitual caffeine consumption.[16]

While HbA1c and FBS values were elevated in cases compared to controls, the differences were not statistically significant (P = 0.055 and P = 0.578, respectively). This indicates that traditional glycemic markers may be insufficiently sensitive for identifying early insulin resistance in non-diabetic individuals. Consequently, the inclusion of fasting insulin and HOMA-IR measurements offers a more nuanced evaluation of metabolic dysfunction. Comparable results were documented in studies indicating no substantial impact of caffeine on HbA1c,[14,15] whereas other studies.[16]

Based on current work, there was a high association between fasting insulin levels and HOMA-IR (with a strong positive correlation (PC: 0.982) [Figure 1]. HOMA-IR values grew in tandem with fasting insulin levels, indicating a clear link between insulin resistance and insulin levels. This association is expected because HOMA-IR is calculated using fasting insulin and glucose levels, serving as an indicator of insulin sensitivity (the calculation above).[10] The trendline's closely packed data points correspond to a steady and predictable pattern, supporting the theory that increasing insulin resistance is linked to higher fasting insulin levels.[17] Statistical analysis revealed a significant association (P < 0.001). This is in accordance with other studies showing a significant association between insulin levels and HOMA-IR.[18]

There was a statistically significant association (P < 0.001) between FBS levels (mg/dL) and HOMA-IR (%), with a positive correlation (PC: 0.646) [Figure 2]. HOMA-IR values grow in the same manner as fasting sugar levels, suggesting a possible link between insulin resistance and blood sugar levels. This association is predictable as HOMA-IR is calculated

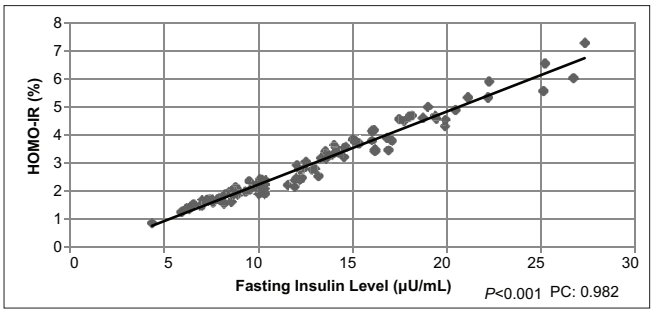

Figure 1: Correlation between fasting insulin level and homeostatic model assessment of insulin resistanc

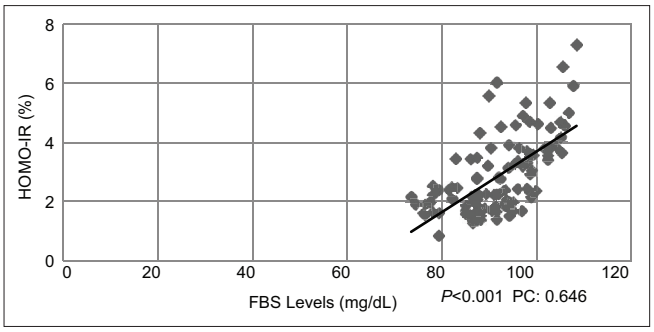

Figure 2: Correlation between fasting blood sugar level and homeostatic model assessment of insulin resistance

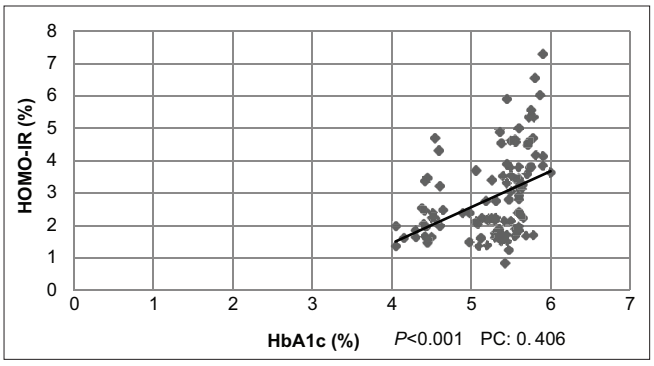

Figure 3: Correlation between hemoglobin A1C and homeostatic model assessment of insulin resistance

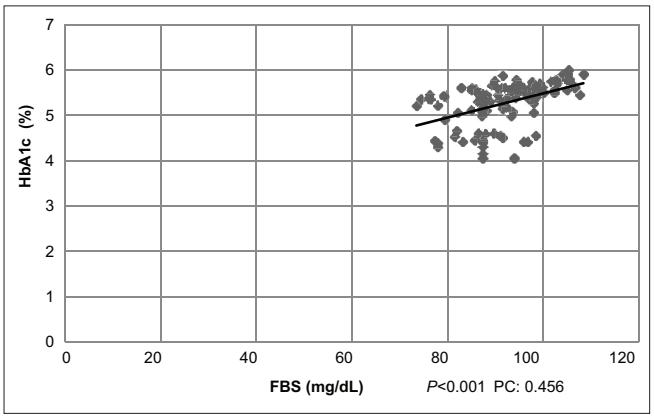

Figure 4: Correlation between fasting blood sugar level and hemoglobin A1C

using fasting insulin and glucose levels, serving as an indicator of insulin sensitivity.[10] The trendline's data points support the theory that increasing FBS levels is linked to higher fasting insulin resistance.[17] Statistical analysis revealed a significant association ($P < 0.00$). The trendline's data points support the theory that increasing FBS levels is linked to higher fasting insulin resistance.[19]

The current work illustrated the link between HbA1c (%) and HOMA-IR (%) with a positive correlation (PC: 0.406) [Figure 3]. HOMA-IR values increased with HbA1c, indicating their direct association. This relationship is expected since HbA1c represents the average blood sugar control over the 2–3 months, and HOMA-IR is calculated using fasting insulin and glucose levels.[20] Statistical analysis revealed a significant association ($P < 0.001$). Other studies have reported the association between these two glycemic parameters.[21,22] Another study showed a non-significant association.[23] There was also a positive correlation between HbA1c (%) and FBS (mg/dL) [Figure 4], indicating their positive association ($P < 0.001$).

CONCLUSION

This study investigated that long-term consumption of caffeinated energy beverages significantly raises the fasting insulin and HOMA-IR levels, indicating early stages of insulin resistance. While HbA1c and FBS did not show significant differences, their upward trends may indicate early metabolic disruption. Further studies are required to explore the long-term impacts of these drinks on glycemic control and diabetes progression.

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