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Original article

THE POTENTIAL RISK OF TYPE 2 DIABETES MELLITUS AND COAGULOPATHIES ASSOCIATED WITH CONSUMPTION OF ENERGY DRINKS

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Abstract

Introduction: The consumption of energy drinks (EDs) has risen in recent years and is associated with numerous health concerns. This study aims to evaluate the effects of EDs consumption on blood glucose, HbA1c, hematological, coagulation parameters, and liver markers in mice.

Materials and methods: Twenty mice were equally divided into two groups; one group served as a control and the other was given EDs. After four months, blood glucose, HbA1c, hematological, coagulation profile, and liver markers were analyzed using an unpaired t-test.

Results: Results showed that mice fed EDs had higher blood glucose and HbA1c. In addition, platelet count was lower, whereas levels of prothrombin time, activated partial thromboplastin time, international normalized ratio and D-dimer were higher in ED-treated group, with reduced levels of fibrinogen. Results showed that levels of alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase were significantly higher in ED-treated mice, while total protein and albumin were reduced.

Conclusion: These data suggest that the intake of EDs potentially increases the risk of type 2 diabetes mellitus and coagulation abnormalities.

Key words: energy drinks, type 2 diabetes mellitus, coagulation, blood glucose

Resumen

El riesgo potencial de diabetes mellitus tipo 2 y coagulopatías asociadas al consumo de bebidas energéticas

Introducción: El consumo de bebidas energizantes (BE) ha aumentado en los últimos años y se asocia a numerosos problemas de salud. Este estudio tiene como objetivo evaluar los efectos del consumo de BE sobre la glucemia, la HbA1c, los parámetros hematológicos, de coagulación y los marcadores hepáticos en ratones.

Materiales y métodos: Veinte ratones se dividieron en dos grupos iguales: un grupo que sirvió de control y el otro grupo que recibió BE. Después de cuatro meses, se analizaron la glucemia, la HbA1c, el perfil hematológico, de coagulación y los marcadores hepáticos mediante una prueba t para datos no pareados.

Resultados: Los resultados mostraron que los ratones a los que se suministró BE presentaron niveles más altos de glucemia y de HbA1c. Además, el recuento de plaquetas fue menor, mientras que los niveles de tiempo de protrombina, tiempo de tromboplastina parcial activada, razón internacional normalizada y dímero D fueron mayores en el grupo tratado con BE, con niveles reducidos de fibrinógeno. Los resultados mostraron que los niveles de alanina aminotransferasa, aspartato aminotransferasa y fosfatasa alcalina fueron significativamente mayores en los ratones tratados con BE, mientras que

los niveles de proteína total y albúmina se redujeron.

Conclusión: Estos datos sugieren que la ingesta de BE podría aumentar el riesgo de diabetes mellitus tipo 2 y anomalías de la coagulación.

Palabras clave: bebidas energizantes, diabetes mellitus tipo 2, coagulación, glucosa en sangre

KEY POINTS
Current knowledge

- The consumption of energy drinks (EDs) has risen in recent years and is associated with numerous health concerns. However, few studies have comprehensively conducted to assess the effects of ED consumption on blood glucose, HbA1C, hematological and coagulation parameters in mice.

Contribution of the article to current knowledge

- Mice given EDs had lower body weight, higher blood glucose, and HbA1C, but they had lower levels of platelet and fibrinogen. Also, the levels of PT, aPTT, INR, and D-dimer were higher in the ED-treated group. The intake of EDs was compatible with type 2 diabetes mellitus and coagulation abnormalities.

The twenty-first century has seen a surge in synthetic and caffeinated energy beverages[1]. Energy drinks (EDs) are relatively new products that are similar to soft drinks but contain additional additives. They typically include caffeine, the primary stimulant, along with carbohydrates, taurine, vitamins, and amino acids[2]. Numerous brands are currently available, with caffeine concentrations ranging from a modest 50 mg to a concerning 505 mg per can or bottle[3]. These drinks are particularly popular among young adults and adolescents due to their ability to increase energy and improve alertness[2]. In 2006, global ED consumption rose by 17% over the previous year, totaling 906 million gallons[3]. Approximately 51% of college students in the United States consume at least one ED per month[4]. The lack of government oversight has led to the aggressive promotion of EDs, primarily to young males, for their psychotropic, performance-enhancing, and stimulant properties[3]. The safety of EDs is controversial given numerous cases of adverse health effects[5].

Recently, there has been a significant increase in emergency reports related to EDs, which have been identified as a cause of cardiovascular issues, including cardiac arrhythmias. These cardiovascular alterations can lead to morbidity and mortality[5]. The sudden increase in blood pressure caused by caffeine is thought to stress the cardiovascular system, thereby increasing the risk of arrhythmia. This increase in blood pressure is more pronounced in the elderly and those with preexisting hypertension[6]. A few studies, reviews, and meta-analyses have examined the correlation between sugar-sweetened beverages and weight gain, overweight, and obesity in childhood and adolescence, with the majority identifying a positive correlation between ED intake and obesity[7,8]. One study found that increased consumption of sugar-sweetened beverages correlates with the onset of metabolic syndrome and type 2 diabetes[9]. Histopathological examination of kidney tissue has revealed a significant difference in the extent of injury in mice given EDs[10].

Lifestyle factors, such as physical inactivity and obesity, are significantly associated with deep vein thrombosis (DVT); however, the specific influence of dietary components, particularly caffeinated beverage consumption, on DVT remains ambiguous[11]. Increased platelet aggregation and impaired endothelial function have been reported in young individuals who consume EDs[4]. However, few studies have comprehensively investigated the effect of ED intake on diabetes induction, hematological, and coagulation parameters. This study aims to assess the effects of ED consumption on blood glucose, HbA1c, hematological, and coagulation parameters in mice.

Materials and methods

Experiment design

The study received an ethical approval from the ethics committee at Taif University (Approval number: 46-224). An institutional and national protocol for the care and utilization of laboratory animals has been followed. The experiments were conducted from December 2024

to March 2025. Twenty BALB/c male mice, weighing 40 g and aged four months, were acquired from the animal shelter at Umm Al-Qura University. The mice were confined in standard rodent enclosures with woodchip bedding in a spacious, well-ventilated room maintained at 25 °C and subjected to a 12-hour light/dark cycle. Standard rodent food and tap water were provided to all mice throughout the investigation. After acclimatizing to their new surroundings, the mice were randomly assigned to two groups of 10 mice each:

(1) Control group: Mice in this group drank water but did not receive any treatment.

(2) Treated group: Mice in this group received an energy drink for four months. The main ingredients of these energy drinks are taurine (30 mg), caffeine (30 mg), and added sugar (14.5 g). Mice were given 9.5 mL/kg of body weight per day of energy drinks (equivalent to two bottles for an adult human daily) by oral intragastric gavage.

Both groups received the same quantity of ordinary mice diet for four months.

Body-weight measurement

The body weight of each mouse was recorded over a period of four months utilizing a digital balance (OHAUS, Model: Scout Pro SPU601, Shanghai, China).

Estimation of blood glucose and HbA1c

After four months, blood glucose concentrations were assessed via a colorimetric technique. Furthermore, HbA1c levels for all subjects in the trial were assessed after the four-month experiment utilizing a glycohemoglobin kit (POINTE Scientific Inc., Canton, MI, USA).

Estimation of complete blood count (CBC)

After the four-month trial, a blood sample was obtained by puncturing the heart with a 23–25 gauge needle while the mouse was anesthetized. The mice were administered intraperitoneal anesthesia using a 2 % sodium pentobarbital solution at a dosage of 50 mg/kg. Following anesthesia, the mice were terminated via spinal dislocation. Blood samples were collected in ethylenediaminetetraacetic acid (EDTA) from each mouse in both groups to assess various parameters of complete blood count using the Beckman Coulter UniCel® D×H 500.

Estimation of coagulation profile

Blood samples were collected in sodium citrate tubes. Prothrombin time (PT), activated partial thromboplastin time (aPTT), international normalized ratio (INR), fibrinogen, and D-dimer levels were quantified in each animal using a Sysmex CS5100 automatic coagulation analyzer.

Assessment of liver markers

Liver function tests, comprising serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), albumin, and total protein were evaluated using a colorimetric assay (Dimension EXL 200, SIEMENS).

Statistical analysis

Statistical analysis was conducted using GraphPad Prism software. An unpaired *t*-test was employed for all comparisons between the ED group and the controls, with data expressed as mean ± standard deviation (SD). The significance threshold was set at $p < 0.05$.

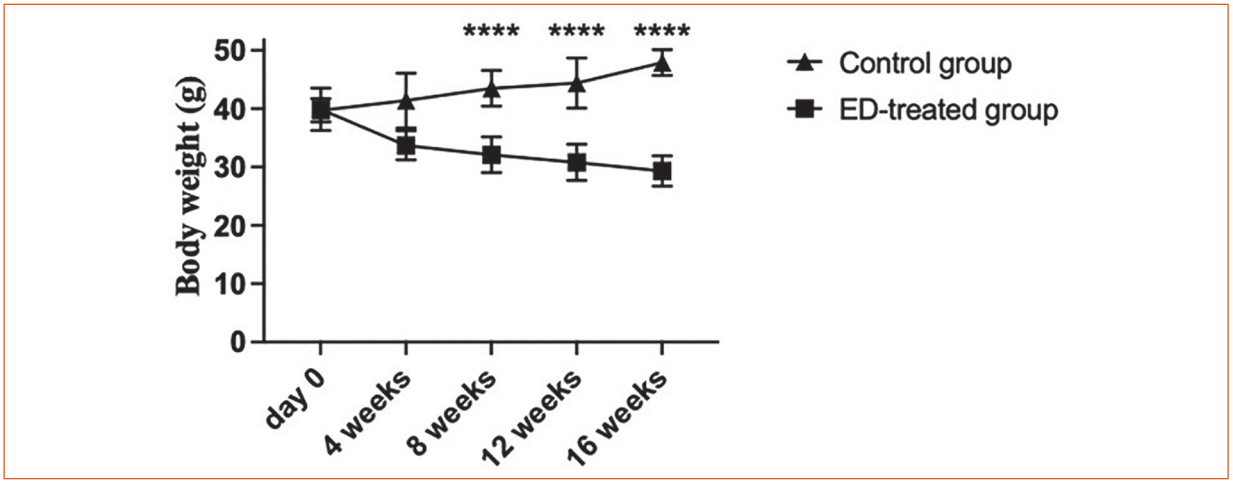
Results

The results showed that mice that received EDs had lower body weights compared to the control group over four months, with a significant difference at 8, 12, and 16 weeks ($p < 0.0001$) (Fig. 1). In addition, blood glucose levels were significantly higher (218.2 ± 23.12, $p < 0.0001$) in ED-treated mice compared to controls (89.96 ± 11.52, Fig. 2). The percentage of HbA1c was also significantly increased (9.95 ± 1.31, $p < 0.0001$) in ED-treated mice compared to the control group (5.26 ± 1.70, Fig. 2).

We evaluated the effect of EDs on several hematological parameters using a complete blood count. Data indicated no significant difference in RBCS: red blood cells (RBC) counts (9.67 ± 1.59 vs 10.40 ± 1.05), hemoglobin (12.32 ± 1.29 vs 14.05 ± 1.75), or hematocrit levels (44.80 ± 3.96 vs 47.18 ± 3.11) between mice given EDs and controls. Furthermore, EDs had no significant effect on WBC counts (6.75 ± 4.38 vs 8.54 ± 3.28). However, platelet counts were lower in mice treated with EDs (838.8 ± 30.19) compared to the control group (1157 ± 27.42, $p < 0.0001$) (Fig.3).

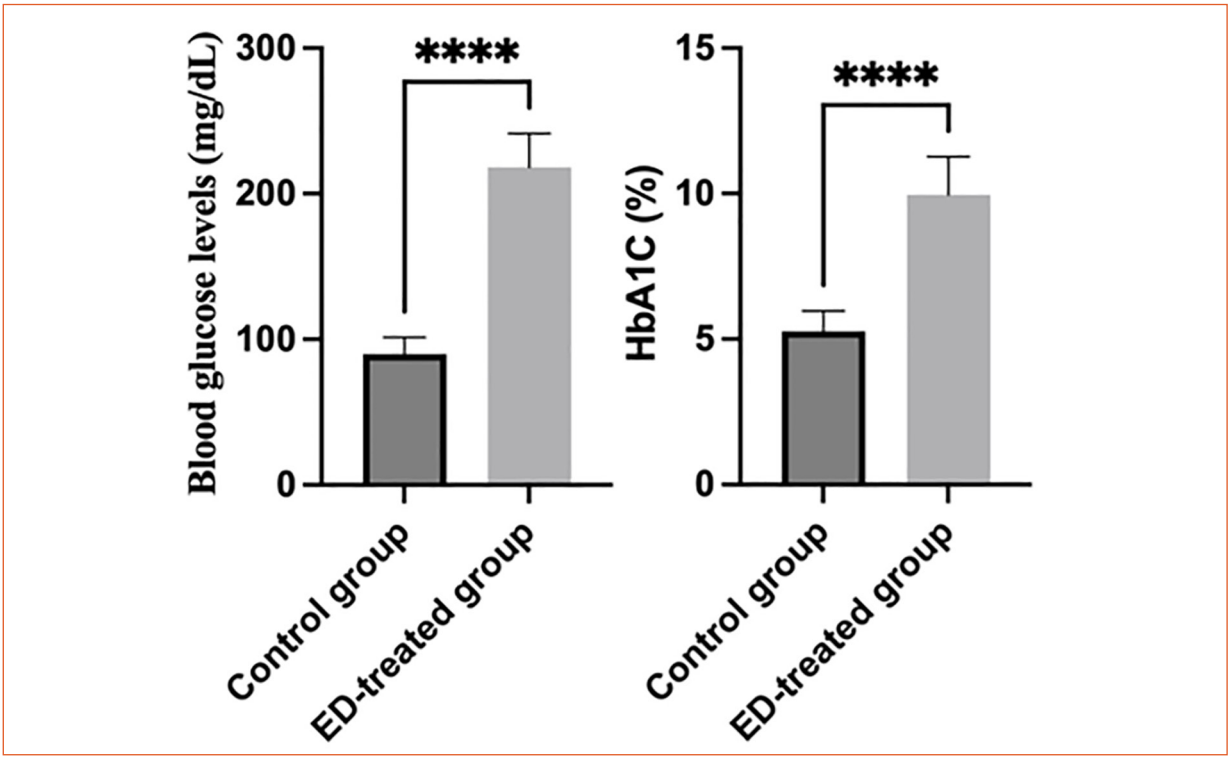
Next, we assessed the coagulation profile in the ED-treated group and the controls (Figure 4). The results showed that the levels of PT ($p < 0.0001$) and aPTT ($p < 0.0001$) were significantly elevated in the ED-treated group compared to the controls (32.70 ± 6.41 vs 9.16 ± 0.93) and (77.40 ± 8.96 vs 36.40 ± 2.88), respectively. Moreover, consumption of ED caused an elevation in the INR (3.85 ± 0.51, $p < 0.0001$) compared

Figure 1 | Measurement of body weight. The body weight in mice treated with energy drinks and controls were measured after 4 months



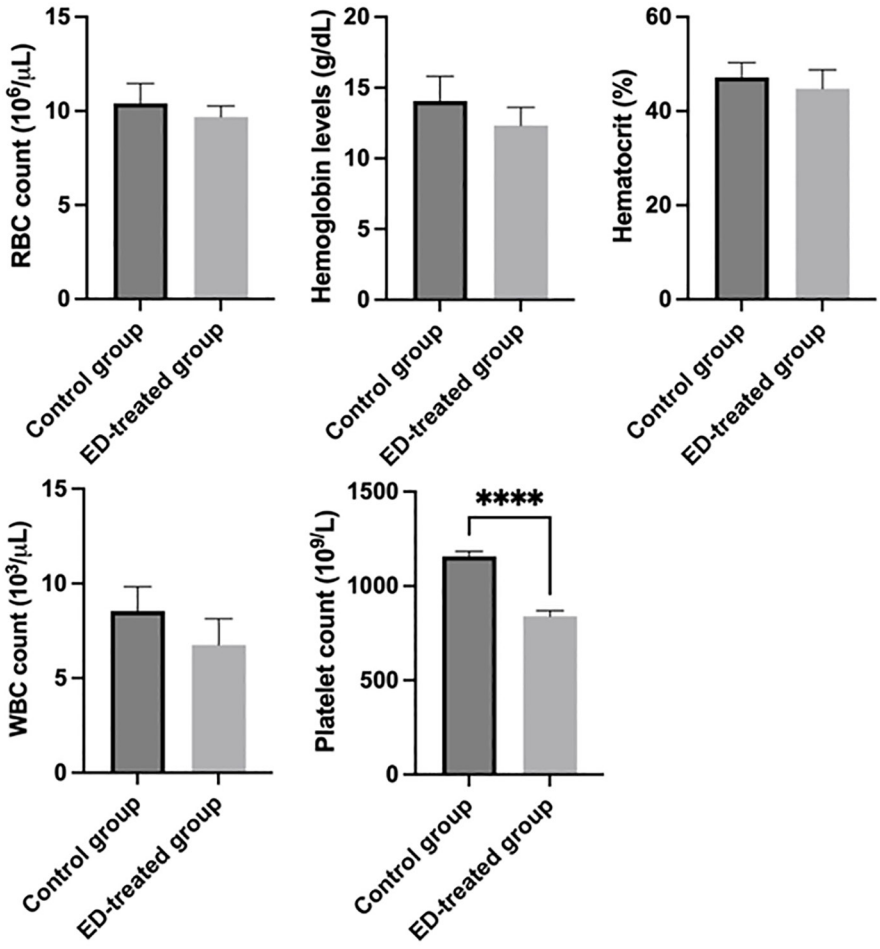
ED: energy drinks
*****p ≤ 0.0001*

Figure 2 | Measurement of blood glucose levels and HbA1c. Both the blood glucose levels and HbA1c were measured in mice treated with energy drinks and controls after 4 months. The t test was used to evaluate differences between groups



ED: energy drinks
*****p ≤ 0.0001*

Figure 3 | Estimation of complete blood count. Different parameters of complete blood count were measured in mice treated with energy drinks and controls after 4 months. The t test was used to evaluate differences between groups



ED: energy drinks; RBCs: red blood cells; WBCs: white blood cells
*****p ≤ 0.0001*

to the controls (0.95 ± 0.22). Compared with the control group, the ED-treated group had higher levels of D-dimer (1.60 ± 0.43 vs 0.90 ± 0.21) ($p < 0.05$). However, ED-treated mice had significantly lower levels of fibrinogen (135.2 ± 8.04 vs 166.6 ± 9.15) ($p < 0.001$) (Fig. 4).

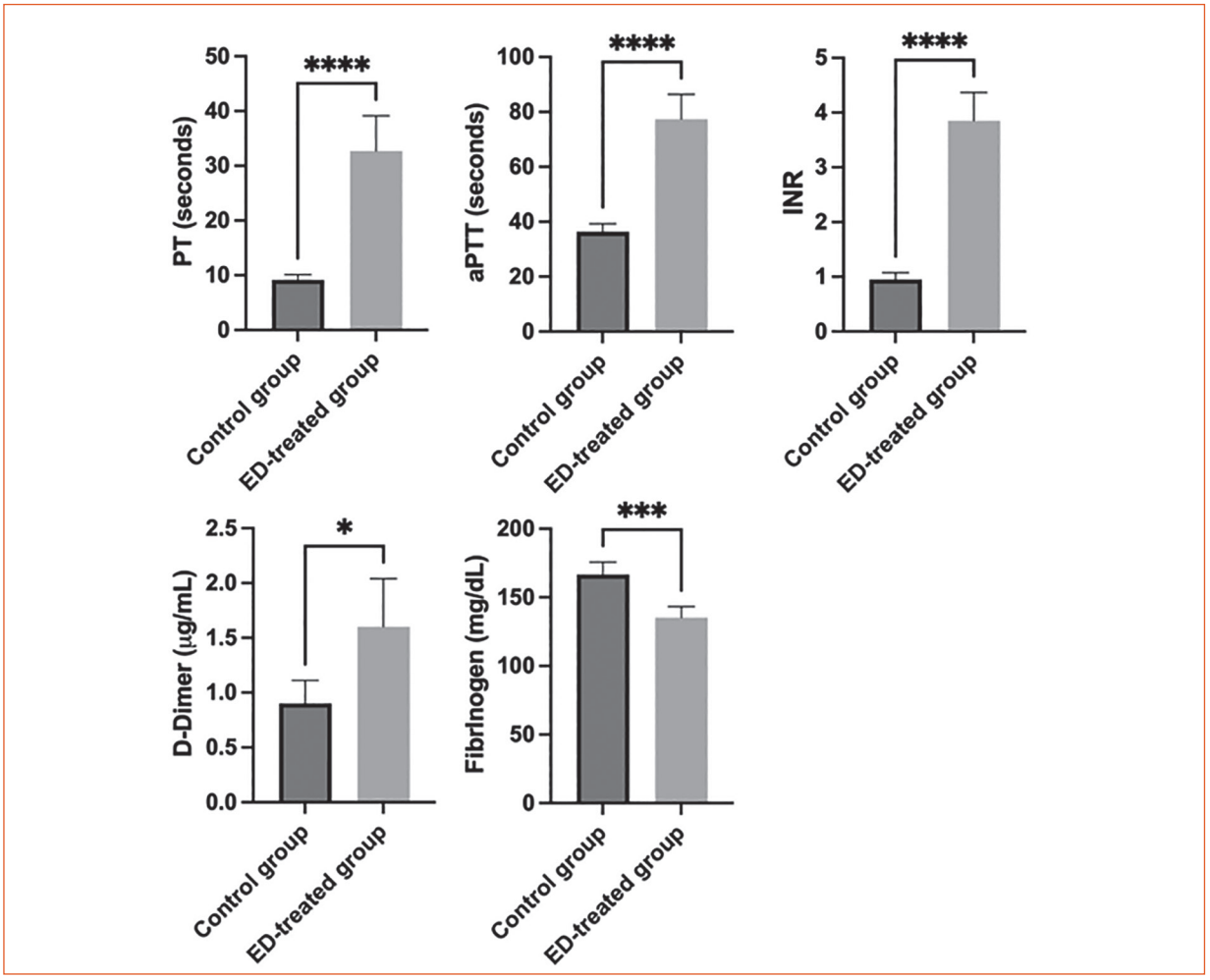
Results showed that the levels of ALT, AST, and ALP were significantly higher in ED-treated mice compared to controls ($p < 0.0001$, Table 1). Furthermore, data revealed a significant decrease in the levels of total protein ($p < 0.01$) and albumin ($p < 0.001$) in the ED-treated group compared to the control group (Table 1).

Discussion

The consumption of EDs has grown globally, becoming a societal issue[12]. Many reports have found that these drinks can cause many side effect and organ toxicities[1, 12]. However, the association between excessive consumption of EDs with type 2 diabetes mellitus and coagulopathy are limited. Thus, we aimed to explore the potential effect of ED ingestion on blood glucose levels, HbA1c, hematological, and coagulation parameters.

It should be noted that the primary constituents of these beverages include caffeine, tau-

Figure 4 | Estimation of coagulation profile after 4 months. The coagulation profile was estimated in mice treated with energy drinks and controls. The t test was used to evaluate differences between groups.



ED: energy drinks; PT: prothrombin time; aPTT: activated partial thromboplastin time; INR: international normalized ratio
*$p \leq 0.05$, ***$p \leq 0.001$ and ****$p \leq 0.0001$*

Table 1 | Liver function tests after 4 months

Parameter	Control group	ED-treated group	P value
ALT (U/L)	22.96±4.49	131.80±7.50	<0.0001
AST (U/L)	26.48±4.99	219.40±6.54	<0.0001
ALP (U/L)	67.76±3.97	252.40±4.66	<0.0001
Total protein (g/L)	71.70±5.32	56.60±5.96	<0.01
Albumin (g/L)	55.54 ±5.93	24.04 ±5.55	<0.001

ED: energy drinks; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase

rine, sugar, and B-vitamins. EDs may contain double or triple the FDA's established safe caffeine concentration limit of 71 milligrams (mg) per 12-ounce beverage[12]. For example, Red Bull, contain approximately 32 mg of caffeine and 11 grams of sugar per 100 mL[12]. Our data have shown that ED induced low body weight, high blood glucose levels, and higher percentage of HbA1c. It is well-known that EDs have many ingredients such as sugar and other sweeteners[12]. Consistent with our results, it was suggested that the consumption of sugar-sweetened beverages is associated with the development of metabolic syndrome and type 2 diabetes[8]. It was also reported that prolonged intake of these energy-dense foods with high levels of simple sugars may result in obesity and insulin resistance[12]. It should be noted that the lower body weight observed in our study could be related to the potential organ toxicities associated with consumption of energy drinks[1, 12]. The lower insulin sensitivity prompts pancreatic beta cells to augment insulin production. Over time, beta cells become incapable of secreting adequate insulin to sustain normal blood glucose levels, resulting in diabetes[12]. In contrast, it was suggested that taurine may enhance the efficacy of insulin and its receptors, potentially benefiting the blood glucose levels of diabetes patients[13].

There are very limited data on the potential effect of ED intake on the hemostasis system. However, it was reported that there is an association between caffeine intake and cardiovascular disorders and blood pressure[14]. In the current study, we have reported that treatment of mice with EDs has no effect of RBC and WBC counts, while the number of platelets were significantly reduced causing thrombocytopenia. In addition, we showed that consuming EDs caused prolongation of PT, aPTT, INR, D-dimer, whereas the levels of fibrinogen were reduced. In agreement with our findings, it was shown that EDs rich in taurine is associated with the induction of thrombocytopenia[15]. Another study found that endothelial dysfunction and platelet aggregation were observed in participants following the consumption of EDs[16]. Furthermore, a different study observed a reduction in thrombocytopenia, platelet aggregation, and endothelial function in mice administered EDs for almost two weeks[16]. In a human study, it was reported that taurine reduces platelet function and may have a crucial role in mitigating the risk of thrombosis[17]. Taurine has consistently been shown to suppress platelet activation and aggregation in both animals and humans[18]. In light of the anticipated effects of ED, a thorough assessment of its influence on the coagulation system is recommended, with supplementary platelet function testing.

It is well-documented that such abnormalities in these coagulation profile correlates with an augmented propensity for bleeding tendency and those abnormal coagulation parameters are frequently seen in many disorders, including liver diseases[17]. Thus, the abnormal coagulation profile observed post treatment with EDs suggest that the liver is potentially damaged. In the current study, it was shown that the levels of ALT, AST, ALP were higher in ED-treated mice, while a significant reduction in the levels of total protein and albumin was noted. In agreement with our findings, a study demonstrated that the blood levels of ALT and AST were markedly elevated in the ED groups relative to the control group[19]. Additional severe and potentially fatal outcomes associated with ED intake include acute hepatitis[12]. The liver is a central organ in the homeostasis of the hemostatic system. The liver synthesizes the majority of plasma proteins involved in hemostasis including pro- and anticoagulant factors[20]. The liver is also responsible for producing thrombopoietin hormone (stimulant of platelet production) and its levels was reduced in individuals with liver diseases[21]. It was also reported that alterations in coagulation markers have been a defining characteristic of advanced liver disease[22]. Additionally, it was demonstrated that thrombocytopenia is a prevalent hematological consequence in individuals with acute and chronic liver diseases[23, 24]. Furthermore, individuals with hepatic disorders exhibit a tendency for hemorrhage, characterized by prolonged results of coagulation screening tests, especially prothrombin time[25].

This study has some limitations. For example, the impact of various duration and doses of the EDs could be further investigated. Also, the potential impact of EDs on the liver must be further verified through HE staining of the liver to con-

firm its harmful effect. In addition, the potential impact of EDs on diabetes and hemostasis system was demonstrated using limited markers.

In conclusion, the consumption of EDs has demonstrated to affect blood glucose levels, HbA1c, coagulation, and liver markers, suggesting they could increase the risk of type 2 diabetes mellitus and coagulation abnormalities in a mice model. The identified coagulopathies may be associated with the hepatic damage caused by EDs. Thus, a thorough examination and additional markers of diabetes and hemostasis is necessary to validate these findings. However, awareness must be raised regarding the significant negative effects that these beverages may induce.

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Conflicts of interest: None to declare

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