Original Article

Effect of Different Doses of Energy Drink on The Pancreas of Adult Male Albino Rats and The Effects of Its Withdrawal. Light and Electron Microscopic Study

Marium Romany, Sara Abdel Gawad Elsebay, Abeer A. Abd El Samad and Nagwa Kostandy Kalleny

Department of Histology, Faculty of Medicine, Ain Shams University, Egypt

ABSTRACT

Introduction: Energy drinks are non-alcoholic beverages containing mainly caffeine which recently became frequently used as concentrate boosting agents. Recent findings indicate that caffeinated energy drinks can affect the structure and function of many organs.

Aim of the Work: Studying the influence of different doses of Red Bull -as an example of caffeinated energy drinks- on the pancreas of adult male albino rats and the effects of its withdrawal using microscopic techniques.

Materials and Methods: 63 adult male albino rats were separated into three groups. Group I (control group), Group II (energy drink administered) was divided into IIA and IIB that were orally administrated Red Bull 7.5 ml/kg/day and 15 ml/kg/day respectively once daily for 4 weeks, and Group III (energy drink withdrawal) was divided into IIIA and IIIB that were administrated Red Bull as IIA and IIB respectively, then were left for 15 days without any treatment. Light and transmission electron microscopic studies were performed.

Results: Red Bull administration demonstrated focally disrupted architecture in both the exocrine and endocrine pancreas. These changes were dose dependent, as they were more noticeable and extended to include wide areas of pancreatic lobules in the subgroup administered high dose energy drink. Discontinuing the intake of Red Bull partially improved these alterations particularly in the low dose subgroup.

Conclusion: Consumption of energy drinks caused dose-dependent histological changes in the pancreas structure, and its withdrawal partially improved these alterations, particularly in rats administered lower consumption.

Received: 07 April 2025, **Accepted:** 30 April 2025

Key Words: Energy drink, histology, islets of langerhans, pancreas, red Bull, transmission electron microscopy.

Corresponding Author: Marium Romany, MD, Department of Histology, Faculty of Medicine, Ain Shams University, Egypt, **Tel.**: +20 10 3228 8154, **E-mail:** Marium@med.asu.edu.eg

ISSN: 1110-0559, Vol. 48, No. 2

INTRODUCTION

Energy beverages are well-known alcohol-free beverages that contain different concentrations of caffeine and other stimulants, including sugars, amino acids, and several herbal supplements[1]. Nowadays energy drinks usage has increased dramatically, so concerns regarding their health impacts were studied to define their side effects and to how far they are safe or dangerous[2].

There is a growing interest in performing experimental research to examine and understand the influence of energy drinks on various bodily organs[3]. Energy drinks contain excessive amounts of sugar, ranging from 10-13 % of their volumes. This causes obesity and type 2 diabetes[4].

Given the previous data, this study intended to evaluate the influence of different doses of Red Bull -as an example of caffeinated energy drinks- on the pancreas of adult male albino rats and the effects of its withdrawal histologically.

MATERIALS AND METHODOLOGY

Substance administered

Red Bull®, as an example of caffeinated energy drinks, was purchased at the local Egyptian souq, and was used in this study. Hulail *et al*.[5] mentioned that 100ml of Red Bull contains taurine (400 mg), sucrose and glucose (11.3 g), caffeine (32 mg), gluconolactone (240 mg), niacin (7.2 mg), B6 (0.8 mg), pantenol (2.4 mg), inositol (20 mg), B2 (0.64 mg).

The consumption of 3.57 ml/kg/day of Red Bull in rats is matching to one can of energy drink (250 ml) oral in humans[6], which means that the present work studied the effect of 2 cans daily in the group that received a low dose, and the effect of 4 cans daily in the group that received a high dose.

Animals

Sixty-three adult male albino rats weighing an average of 200 grammes each were used in this study. Animals

DOI: 10.21608/ejh.2025.371784.2241

were kept at Medical Ain Shams Research Institution (MASRI). We kept the animals in clean plastic cages with wire mesh tops and possessed free food and water during this trial. They were kept at the proper light, temperature, and humidity levels.

Ethical approval

Every animal experiment was conducted in accordance with Federal Wide Assurance No. FMASU MD 161/2021, ethical approval was granted by the Faculty of Medicine's Ethical Committee (FMASU REC) for animal research at Ain Shams University.

Experimental design

Three groups were randomly assigned to the animals following a seven-day acclimatisation period:

Group I (Control): contained 27 rats that were separated into three equal subgroups; all were sacrificed on the 30th day of the experiment. Subgroup IA: did not receive treatment. Subgroup IB: were given 7.5 ml/kg/day of water orally using gastric tube daily. Subgroup IC: were given 15 ml/kg /day of water by mouth by gastric tube daily.

Group II (Red Bull administered group): Contained 18 rats which were equally split into two subgroups: Subgroup IIA (Low Dose Red Bull): Rats were given 7.5 ml/kg/day of Red Bull orally using gastric tube once daily and were sacrificed on the 30th day of the experiment[7]. Subgroup IIB (High Dose Red Bull): were given 15 ml/kg/day of Red Bull orally using gastric tube once daily and were sacrificed on the 30th day of the experiment[7].

Group III (Red Bull Withdrawal): contained 18 rats which were equally split into 2 subgroups: Subgroup IIIA (Red Bull Withdrawal Low Dose): rats were given Red Bull as in subgroup IIA, Subgroup IIIB (Red Bull Withdrawal High Dose): Rats were given Red Bull as in subgroup IIB. Rats of both subgroups (IIIA & IIIB) were left for another fifteen days without any interference and were sacrificed on the 45th day of the experiment[5-7].

Sample Collection

When the experiment was over, the rats were sacrificed according to the study grouping, by intraperitoneal injection of anesthesia by Thiopental Sodium (40 mg/kg)[8]. Specimens of the pancreas were obtained from the tail that contains a higher number of islets[9], and then were subjected to both light (LM) and transmission electron microscopic (TEM) studies.

Preparation of Tissue

Every specimen was split into two parts. The first part was fixed in 10% formalin for LM study. The second part was cut into small pieces of 1 mm3 and fixed in 2.5% phosphate buffered glutaraldehyde for TEM study.

For LM study, pancreatic specimens were fixed in 10% formalin, then dehydrated, cleared, and finally embedded in paraffin. Serial sections of 5-6 µm thick were stained by hematoxylin & eosin (H&E)[10].

For TEM study, small sections of pancreatic specimens were taken off and fixed in 2.5% phosphate buffered glutaraldehyde1% osmium tetroxide post-fixation, followed by ethanol dehydration in increasing grades. The specimens were submerged in propylene oxide before being imbedded in a mixture of epoxy resin. To choose appropriate regions for ultrathin sectioning, semithin sections (1µm) were stained with 1% toluidine blue and viewed under the LM[10]. Uranyl acetate and lead citrate were used to stain ultrathin sections (60 nm), which were then inspected using a JEOL-JEM-1200EX II electron microscope at the Electron Microscopic Unit, Faculty of Science, Ain Shams University, Egypt.

RESULTS

No mortality occurred throughout the duration of the study.

Results of control subgroups were almost similar in all histological findings.

Results of light microscopy

The H&E-stained sections of the control grouped showed that the exocrine part consisted of tightly packed serous acini separated from each other by vascular connective tissue septa, dividing the gland into lobules. Each acinus was formed of pyramidal serous cells surrounding a narrow lumen. The cytoplasm of the acinar cells showed intense basal basophilia and apical acidophilia. The endocrine part of pancreas was formed of the islets of Langerhans, which appeared lightly stained in between the more deeply stained exocrine pancreatic acini. The cytoplasm of the islets' cells appeared pale acidophilic, and the nuclei were vesicular (Figure 1 A).

Compared to control group, H&E sections of Red Bull administered subgroups IIA and IIB showed noticeable focal structural alterations in the exocrine and endocrine tissues of the pancreas.

Examination of H&E-stained sections of the exocrine part of subgroup IIA showed altered pancreatic architecture that appeared as wide interlobular spaces and apparent decrease or even loss in the basal basophilia and apical acidophilia in the affected pancreatic serous acinar cells as compared to control group. Also, some affected acinar cells showed vacuolation of the cytoplasm. Moreover, some nuclei of the affected acinar cells appeared shrunken and darkly stained. The pancreatic ducts appeared dilated and lined by squamous epithelial cells, and some showed retained secretion. Dilated congested blood vessels and homogenous acidophilic material were detected between the pancreatic acini (Figures 1 B-C). The endocrine part of the pancreas (islets of Langerhans) of subgroup IIA showed up with irregular outlines and showed disturbed cellular architecture as compared to the control group. Some endocrine islets' cells showed vacuolated cytoplasm.

The nuclei of the affected islets' cells appeared small and darkly stained (Figure 1 D).

LM examined sections of the exocrine and endocrine tissues of the pancreas of subgroup IIB showed more profound and noticeable structural changes that extended to include wider areas of pancreatic tissue in this subgroup as compared to subgroup IIA that administered low dose of Red Bull.

Examination of H&E-stained sections of the exocrine pancreatic part of subgroup IIB showed that most of the serous acini were distorted with variation in size and shape up to complete loss of architecture with loss of lobulation of pancreatic tissue and wide separation between acini. Some acinar cells showed loss of basal basophilia and apical acidophilia. Others showed vacuolation of the cytoplasm and pyknotic nuclei (Figures 1E-G). In addition, loss of cellular details in some acini could be encountered, in which some acinar cells appeared as homogenous acidophilic substance giving a glassy appearance as compared to subgroup IIA (Figures 1 E-F). The pancreatic ducts were dilated, lined by flattened epithelial cells, and some showed retained secretion (Figure 1 F). In some areas, different sizes of unilocular fat cells appeared accumulated in between the distorted acini (Figure 1 G). Homogenous acidophilic material, multiple dilated congested blood vessels and mononuclear cellular infiltration were noticed in between the distorted acini (Figures 1 E-G). The endocrine part of the pancreas of subgroup IIB showed that the islets of Langerhans appeared with irregular outlines, widely separated from the exocrine part, and showed disturbed cellular architecture as compared to the control group. The affected islets' cells showed vacuolated cytoplasm, and their nuclei were small and darkly stained. In addition, dilated congested blood capillaries were observed in between the islets' cells (Figure 1 H).

LM examined sections of the exocrine and endocrine pancreatic tissues of subgroup IIIA showed marked regression of many of the altered microscopic architecture as compared to subgroup IIA. However, there was persistence of some structural alterations as compared to the control group.

The examined H&E-stained sections obtained from the exocrine part of the pancreas of subgroup IIIA showed obvious lobulation by vascular connective tissue septa. The lobules contained closely packed serous acini. Most of the serous pancreatic acinar cells resembled the usual pancreatic structure revealing vesicular nuclei with prominent nucleoli, well apparent basal basophilia and apical acidophilia apparently similar to the control. However, few acinar cells showed small darkly stained nuclei with perinuclear halo (Figure 2 A). The endocrine part of the pancreas of subgroup IIIA nearly resembled the usual pancreatic structure, showing that the islet of Langerhans was regular, well-defined, and surrounded by delicate vascular connective tissue separating it from the more deeply stained exocrine pancreatic acini. The endocrine cells of the islets of Langerhans appeared nearly rounded in shape. The cytoplasm of the islets' cells appeared pale acidophilic, and most of their nuclei were vesicular with prominent nucleoli. However, few cells showed small darkly stained nuclei (Figure 2 A).

LM examined sections of the exocrine and endocrine pancreatic tissues of subgroup IIIB showed partial regression of some of the altered microscopic architecture as compared to subgroup IIB. However, there were persistence of some structural alterations as compared to the control group.

The examined H&E-stained sections obtained from the exocrine part of the pancreas of subgroup IIIB showed apparent restoration of the usual pancreatic lobulation, however other areas showed apparently wide separation between the acini, markedly dilated congested blood vessels, mononuclear cellular infiltration, and homogenous acidophilic material. Many serous pancreatic acinar cells resembled the usual pancreatic structure revealing vesicular nuclei with prominent nucleoli, well apparent basal basophilia and apical acidophilia nearly similar to the control. However, small darkly stained nuclei were seen in some acinar cells. The pancreatic ducts were dilated and lined by squamous epithelial cells (Figures 2 B-C).

The endocrine part of pancreas of subgroup IIIB showed partial restoration of the usual pancreatic structure, showing that the islet of Langerhans was regular with well-defined outline and surrounded by deeply stained exocrine pancreatic acini. However, some islets were apparently widely separated from the exocrine pancreatic acini. The endocrine cells of the islets of Langerhans appeared nearly rounded in shape. The islets' cells cytoplasm appeared pale acidophilic, and many of their nuclei were vesicular with prominent nucleoli. However, some cells showed small darkly stained nuclei with vacuolated cytoplasm. In addition, there were dilated, congested blood capillaries between the cells of the islets (Figures 2 B-C).

The TEM results

TEM sections of control group showed that the exocrine acinar cell had apical spherical electron dense zymogen granules, well-developed numerous arrays of rER, and rounded, euchromatic nucleus with prominent nucleolus. The intercellular spaces in between the adjacent cells were minimal and appeared as electron lucent spaces. The adjacent exocrine acinar cells were joined together by desmosomes along their lateral surfaces. The endocrine part of the pancreas showed that the islets of Langerhans appeared with well-defined nearly regular outline and in close association to the exocrine part (Figures 3 A-B). Beta cells showed rounded or slightly oval euchromatic nuclei with almost regular outline and their cytoplasm contained secretory granules, each with an electron dense core surrounded by an electron lucent halo. Alpha cells had euchromatic nuclei and showed nearly homogenous electron dense granules (Figure 3 C).

TEM sections of exocrine pancreatic part of subgroup IIA showed distorted serous acinar cells with widening of intercellular spaces as compared to the control group. Some acinar cells showed swollen mitochondria with inapparent cristae and dilated rER cisternae, and apparently few electron dense zymogen granules of different sizes. The nucleus appeared irregular, shrunken, and exhibited characteristic pyknotic distortion with more condensed peripheral chromatin that appeared marginated along the inner nuclear membrane. Moreover, a heterogeneous electron-dense structure was noticed (Figures 4 A-B).

TEM sections of the endocrine part of pancreatic tissue of subgroup IIA showed loss of regular boundary with wide space between it and the exocrine part, together with the appearance of collagen fibrils in between the endocrine cells, with nearby active fibroblasts as compared to the control. Many Beta cells of islets of Langerhans showed euchromatic nucleus and had cytoplasmic granules nearly similar to the control (Figures 4 C-D). However, few endocrine cells were affected containing shrunken irregular more electron dense nucleus and granules with small electron dense cores that were surrounded by wider electron lucent halos as compared to the control. Swollen Golgi apparatus, cytoplasmic vacuoles, and autophagosome were also noticed (Figure 4 E).

The exocrine part of pancreatic tissue of subgroup IIB by TEM showed more alteration of the usual pancreatic architecture of the pancreatic acinar cells as compared to subgroup IIA. Some pancreatic acinar cells had irregular shrunken nuclei with more condensed chromatin. Other cells showed markedly degenerated nuclei. The majority of the cells of the acini showed more vacuolation of the cytoplasm, however the zymogen granules were fewer or inapparent as compared to subgroup IIA (Figure 5 A). The endocrine part of the pancreas showed more distortion of the usual architecture of islets of Langerhans as compared to subgroup IIA. The distorted endocrine cells had cytoplasmic vacuoles and shrunken irregular nuclei with condensed chromatin. Apoptotic blebs were noticed. Active fibroblasts and collagen fibrils were seen in between the endocrine islets' cells (Figures 5 B-C). The Beta cells of islets of Langerhans showed irregular shrunken heterochromatic nucleus. The cytoplasm contained markedly dilated Golgi apparatus, granules with wider halo spaces around their dense cores, and autophagosome (Figure 5 D).

Examination of ultrathin sections of pancreatic tissue of subgroup IIIA illustrated marked restoration of the regular histology of the pancreatic tissue as compared to subgroup IIA. The exocrine part of the pancreas of subgroup IIIA showed apparently normal intercellular spaces between the serous acinar cells. The serous acinar cells had a basal rounded euchromatic nuclei surrounded by numerous arrays of rER with well apparent mitochondria in between the cisternae of rER. The apical part of the cell contained electron dense zymogen granules of variable sizes. A heterogeneous electron-dense structure was also noticed (Figure 6 A).

The endocrine part of the pancreas nearly resembled the usual pancreatic ultrastructure of islet of Langerhans. Beta cells revealed euchromatic nuclei with inapparent nucleolus. Its cytoplasm contained an autophagosome and numerous granules, each with an electron dense core surrounded by an electron lucent halo. However, few granules show wider electron lucent halo around their dense core. The Alpha cells contained numerous homogenous electron dense granules (Figure 6 B).

Examination of ultrathin sections of the exocrine part of the pancreas of subgroup IIIB showed apparently normal intercellular spaces between the serous acinar cells. Some serous acinar cells had basal rounded euchromatic nuclei with prominent nucleoli surrounded by arrays of rER with mitochondria in between the cisternae of rER. Other cells showed euchromatic nucleus with inapparent nucleolus, cytoplasmic vacuoles, and a heterogeneous electron-dense structure. Regarding the electron dense zymogen granules that appeared of different sizes, they showed varied content in the serous acinar cells. Some serous cells contained moderate, or few zymogen granules as compared to the control group, yet, other acinar cells had inapparent zymogen granules (Figures 6 C-D). The endocrine part of the pancreas of subgroup IIIB showed partial regression of the microscopic changes as compared to subgroup IIB. The Beta cells revealed euchromatic nuclei and cytoplasmic granules with an electron dense core surrounded by an electron lucent halo. However, few granules showed wider electron lucent halo around their dense core. Cytoplasmic vacuoles were also noticed (Figure 6 E).

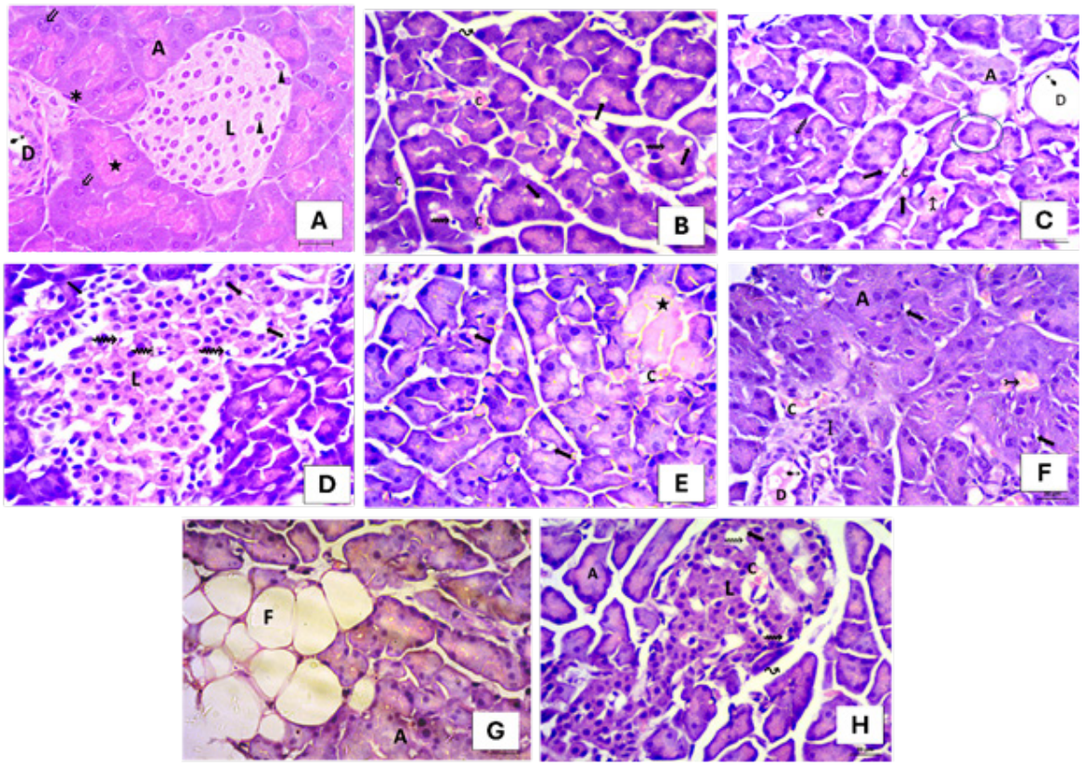

Fig. 1: H&E-stained sections of the pancreas of groups I and II X400. [A] control group showing closely packed serous acini (A). The acinar cells are pyramidal in shape, showing intense basal basophilia (□), apical acidophilia (□), basal rounded vesicular nuclei (□). The pancreatic duct (D) is lined with cubical epithelium with rounded nuclei (□). A well-defined pale-stained islet of Langerhans (L) is seen, the islets' cells have pale acidophilic cytoplasm, and vesicular nuclei (▲). [B -D] subgroup IIA showing apparent decrease in the basal basophilia and apical acidophilia (○). Few acini show loss of basal basophilia and apical acidophilia (A), others show vacuolated cytoplasm (□) and small darkly stained nuclei (□). The pancreatic duct (D) appears dilated and is lined by flattened epithelial cells (□). Notice the dilated congested blood vessels (c) and the homogenous acidophilic material (□) in-between the pancreatic acini. An islet of Langerhans (L) is seen with irregular outlines and altered architecture. Islets' cells are widely separated and show vacuolation of their cytoplasm (□). Some of the affected endocrine cells have small darkly stained nuclei (□). [E-H] subgroup IIB showing loss of lobulation of pancreatic tissue. Most acini lost their basal basophilia and apical acidophilia (A) with some acinar cells appearing as homogenous acidophilic substance (□), others show vacuolation of the cytoplasm and small darkly stained nuclei (□). Notice dilated congested blood vessel (C) with nearby mononuclear cellular infiltration (I) and dilated pancreatic duct (D) that is lined by flattened epithelial cells (□) with retained acidophilic secretion. Notice the homogenous acidophilic material (□) in between the pancreatic acini and the accumulation of different sizes of unilocular fat cells (F) in between the acini. Serous acini (A) are widely separated from the endocrine part (□). Notice an islet of Langerhans (L) with irregular outline and disturbed cellular architecture. Some islets' cells show vacuolation of the cytoplasm (□) and small darkly stained nuclei (□). Notice the dilated congested blood capillaries (C) in between the islets' cells.

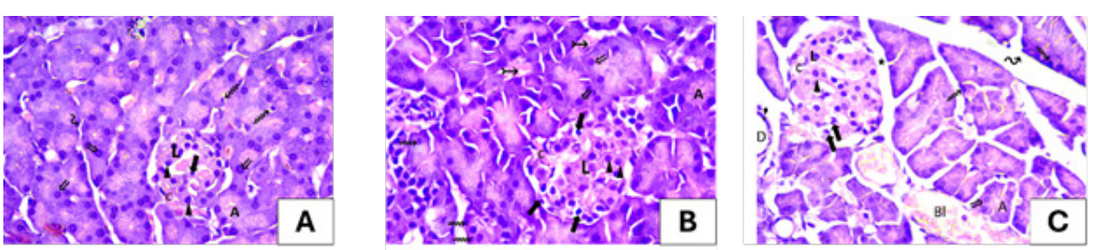

Fig. 2: H&E-stained sections of the pancreas of group III X400. [A] subgroup IIIA: both exocrine and endocrine parts appear nearly similar to the usual pancreatic structure. The exocrine part shows restoration of pancreatic lobulation with almost normal septa (□). Most serous acinar cells (A) contain vesicular nuclei (□) with cytoplasmic basal basophilia and apical acidophilia. Few cells appear with dark small nuclei (□) with perinuclear halo. The islet of Langerhans (L) appears well-defined surrounded by delicate vascular (C) connective tissue. Most of cells in the islet of Langerhans have vesicular nuclei (▲), however few islet cells have small dark nuclei (□). [B, C] subgroup IIIB showing pancreatic acinar cells (A) which appear almost normal with basal basophilia and apical acidophilia, in addition to vesicular nuclei (□). Some pancreatic acinar cells contain dark small nuclei (□). Homogenous acidophilic material (□) is seen in between the pancreatic acini. wide septa (□) and markedly dilated congested blood vessel (Bl) are seen. A dilated pancreatic duct (D) is lined with flattened epithelial cells (□). A regular islet of Langerhans (L) with well-defined outline is seen. Some islets' cells have vesicular nuclei (▲), while other cells have small dark nuclei (□) with vacuolated cytoplasm. Dilated congested capillaries (C) can be seen in between islet cells. Notice an islet of Langerhans (L) is widely separated from the pancreatic acini (□).

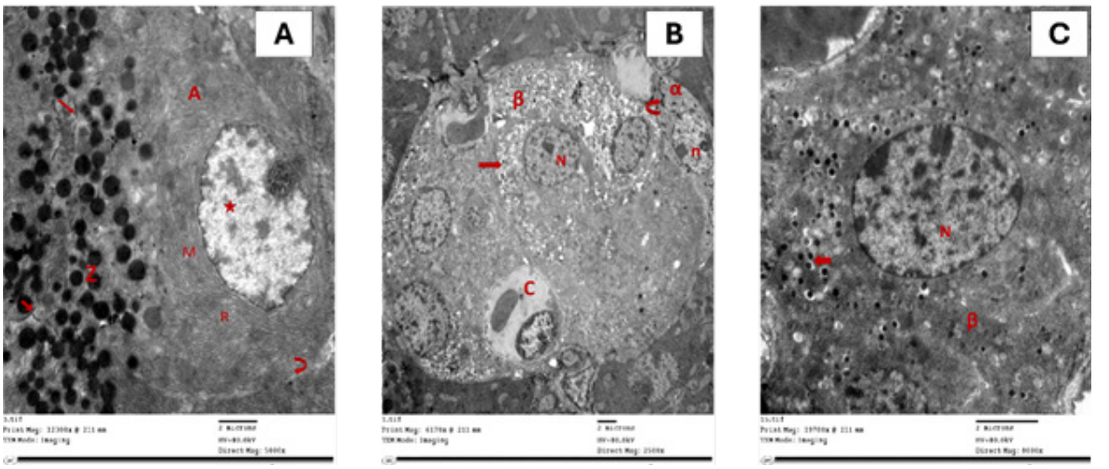

Fig. 3: Electron micrographs of pancreas from control group [A] showing acinar cell (A) with basal euchromatic nucleus (□) surrounded by numerous parallel cisternae of rough endoplasmic reticulum (R), mitochondria (M), and apical numerous electron dense zymogen granules (Z). A narrow intercellular space (□) and desmosomes (□) are seen in between the adjacent acinar cells (TEM x 5000). [B] showing an islet of Langerhans with distinct outline containing closely packed endocrine cells with blood capillaries (C) in between. The Beta cell (β) has rounded euchromatic nucleus (N) with regular contour, its cytoplasm contains numerous granules, each having an electron dense core and surrounded by an electron lucent halo (□). The alpha cell (α) contains nearly homogenous electron dense granules (□) and shows euchromatic nucleus (n) (TEM x 2500). [C] showing Beta cell (β) with euchromatic nucleus (N). The cytoplasm contains numerous granules with electron dense cores and surrounded by electron lucent haloes (□) (TEM x 8000).

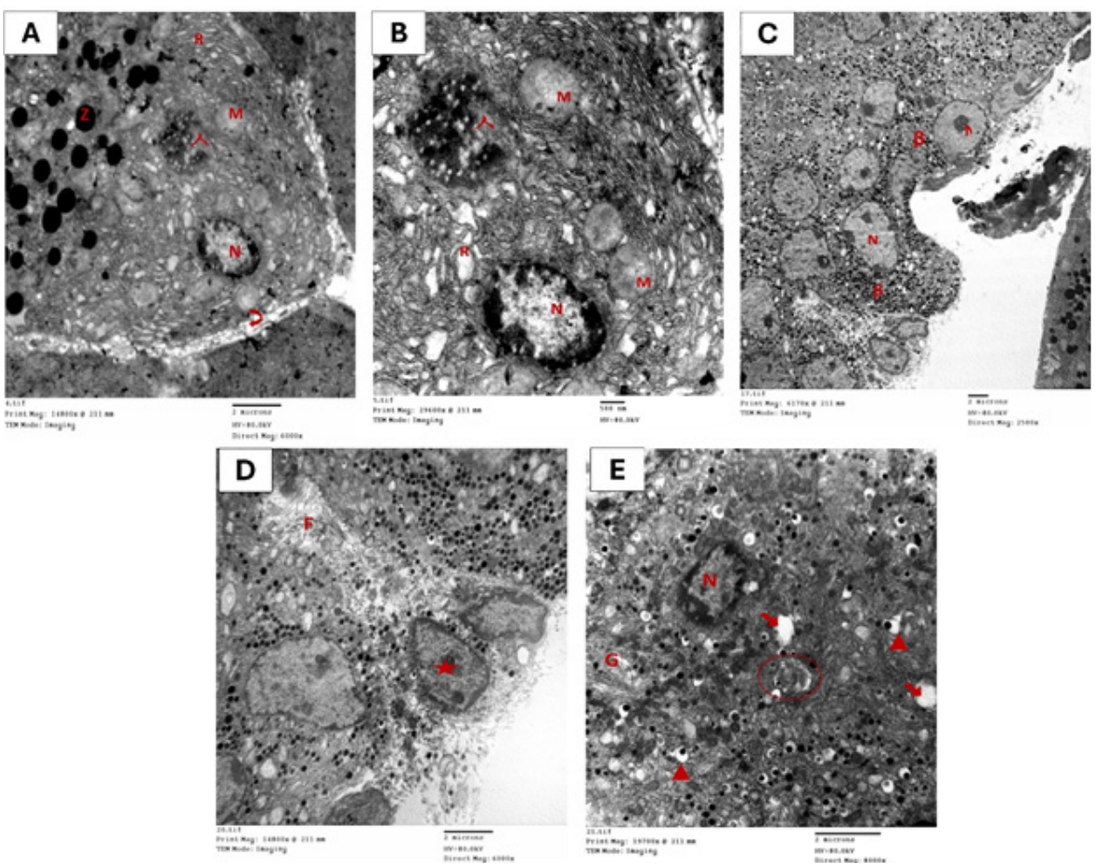

Fig. 4: Electron micrographs of pancreas from subgroup IIA [A] showing altered structure of the pancreatic acinar cells with apparent widening of their intercellular space (□). The acinar cell has shrunken nucleus (N) with condensed peripheral chromatin, swollen mitochondria (M), dilated rER cisternae (R), and few zymogen granules (Z). Notice the heterogeneous electron dense structure (□) (TEM x6000). [B] showing swollen ballooned mitochondria with inapparent cristae (M) and dilated rER cisternae (R) (TEM x 12000). [C] showing an islet of Langerhans with loss of regular well-defined outline and wide separation from the exocrine part. Many Beta cells (β) have euchromatic nuclei (N) and contain cytoplasmic granules with electron dense cores that are surrounded by electron lucent halos (TEM x 2500). [D] showing collagen fibrils (F) in between the endocrine cells, with nearby active fibroblasts (□) (TEM x 6000). [E] showing Beta cell with shrunken irregular nucleus (N). It contains cytoplasmic granules with wider halo spaces around their electron dense cores (▲), swollen Golgi apparatus (G), and cytoplasmic vacuoles (□). Notice the presence of an autophagosome (○) (TEM x 8000)

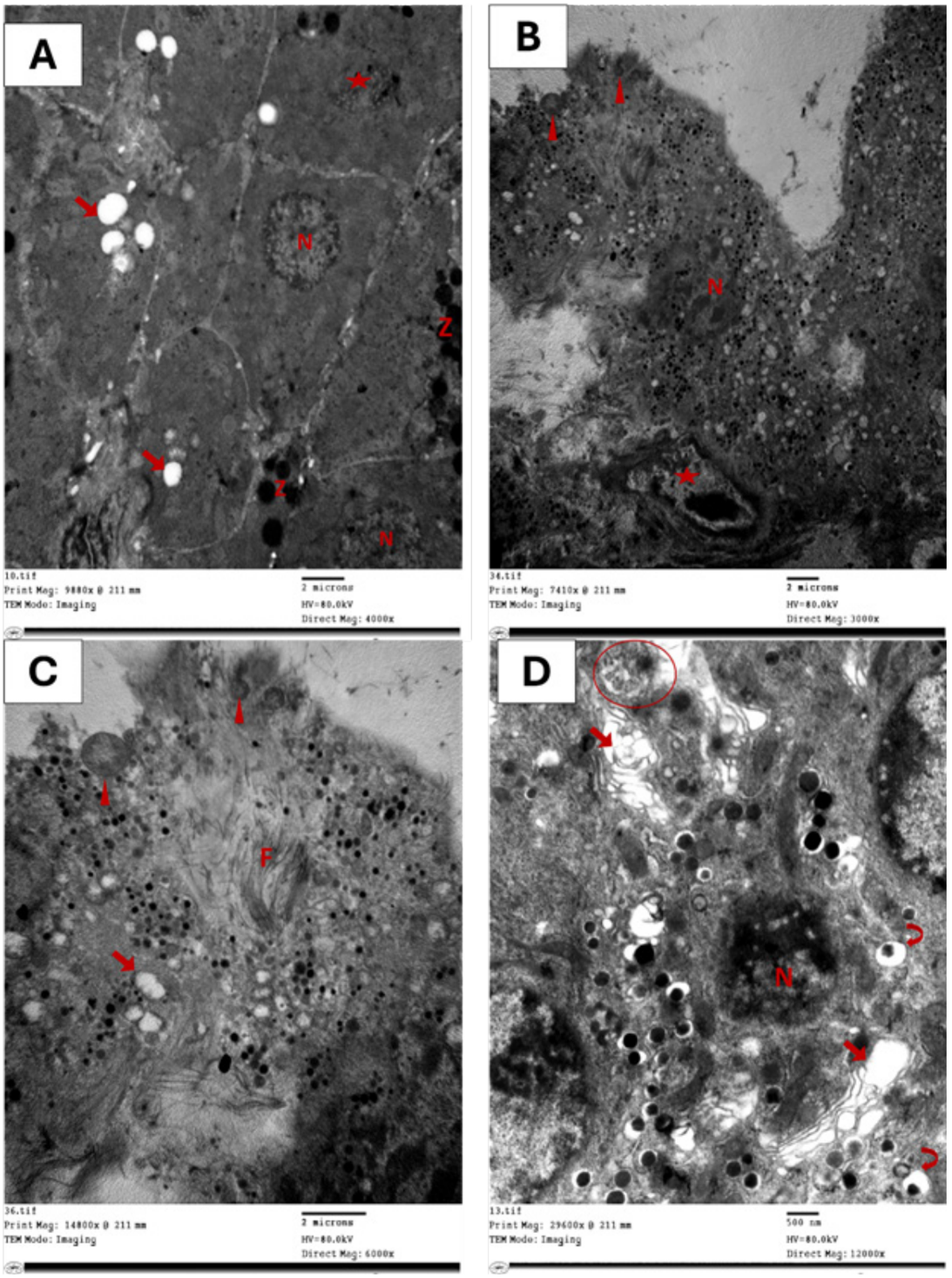

Fig. 5: Electron micrographs of pancreas from subgroup IIB [A] showing distortion of the usual architecture of the pancreatic acinar cells. Some acinar cells have irregular shrunken nuclei with condensed chromatin (N), and others show markedly degenerated nuclei (□). The acinar cells contain cytoplasmic vacuoles (□). The zymogen granules are unobserved in most of the cells and appear few (Z) in some cells (TEM x4000). [B] showing an islet of Langerhans with alteration of the usual endocrine pancreatic architecture. The endocrine cell contains shrunken irregular nucleus (N). Notice the apoptotic blebs (▲). Active fibroblast (□) is present in between endocrine cells (TEM x 3000). [C] showing endocrine cells with cytoplasmic vacuoles (□), and apoptotic blebs (▲). Collagen fibrils (F) are noticed in between the endocrine islets' cells (TEM x 6000). [D] showing Beta cell with irregular shrunken heterochromatic nucleus (N). The cytoplasm contains markedly dilated Golgi apparatus (□), granules with wider halo spaces around their dense cores (□), and an autophagosome (○) (TEM x 12000).

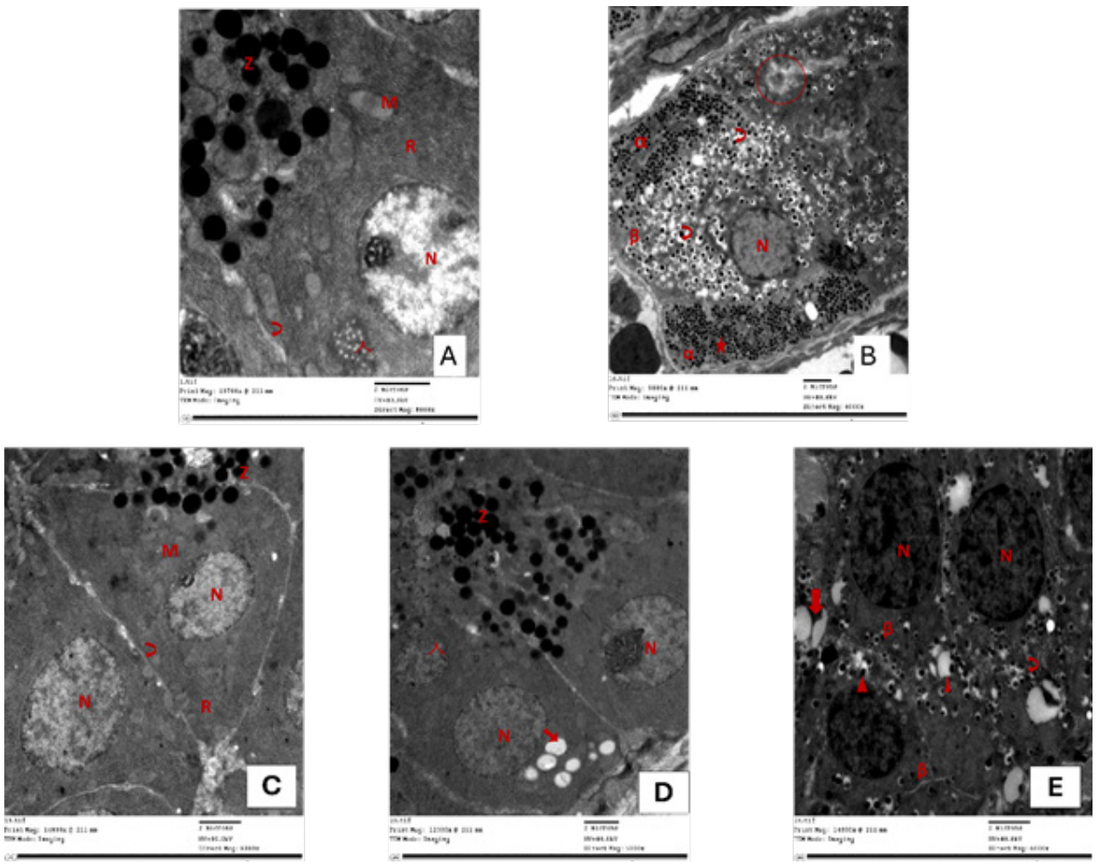

Fig. 6: [A-B] Electron micrographs of pancreas of subgroup IIIA [A] showing acinar cell with a basal rounded euchromatic nucleus (N) surrounded by numerous arrays of rER (R), and well apparent mitochondria (M). Apical electron dense zymogen granules (Z) of different sizes are well evident. Apparently normal intercellular space (□) is seen between the acinar cells. Notice the presence of a heterogenous electron dense structure (□) (TEM x 8000). [B] Beta cell (β) reveals euchromatic nucleus (N) with inapparent nucleolus. Its cytoplasm contains numerous granules, each with an electron dense core surrounded by an electron lucent halo. However, few granules show wider electron lucent halo around their dense core (□). Notice the presence of an autophagosome (○). Alpha cells (α) contain numerous homogenous electron dense granules (□) (TEM x 4000). [C-E] Electron micrograph of pancreas from subgroup IIIB [C] showing partial restoration of the usual architecture of pancreatic acinar cells with almost normal intercellular spaces (□). The acinar cells have basal euchromatic nuclei (N) surrounded by arrays of rER (R) and mitochondria (M) in between cisternae of rER. Zymogen granules of different sizes (Z) appear apparently fewer however, other acinar cells have inapparent zymogen granules (TEM x 6000). [D] showing acinar cell with basal euchromatic nucleus (N) with prominent nucleolus and electron dense zymogen granules (Z) of different sizes. Other cell shows euchromatic nucleus with inapparent nucleolus, cytoplasmic vacuoles (□), and a heterogeneous electron-dense structure (□) (TEM x 6000). [E] showing part of an islet of Langerhans. Beta cells (β) appear with euchromatic nuclei (N) and cytoplasmic granules with an electron dense core surrounded by an electron lucent halo (□). Some cytoplasmic granules show wide halo spaces around their dense cores (▲). Cytoplasmic vacuoles (□) are also noticed. (TEM x 6000)

DISCUSSION

Scince the popularity of energy drinks increasing more and more, it is important to think about any potential negative effects, therefore, this work was done to study the influence of different doses of Red Bull as an example of energy beverages that contain caffein on the histological structure of the pancreas of adult male albino rats and the possible outcome of its cessation.

In the present experiment, focal altered pancreatic structural changes were noticed in both exocrine and endocrine pancreatic tissues in both Red Bull administered subgroups, IIA and IIB compared to the control group. Regarding the lobulation of the pancreatic tissue, both subgroups that administered Red Bull in this study (IIA and IIB), showed wide separation in-between the pancreatic acini, this was in accordance with[11]. This study concluded that the higher dose of Red bull administration caused more profound and noticeable structural changes that extended to include wider areas of pancreatic tissue in subgroup IIB. Accordingly, this denotes that the damaging effect of Red Bull on pancreatic tissue is dose dependent. This was in accordance with Rehman *et al*.[7], Qassim *et al*.[12], and Memon *et al*.[13] who studied the effect of low dose of energy drink consumption on the pancreas histological of rats for 4 weeks in comparison to the effect of double the dose of the same energy drink in the same circumstances and the same duration. In agreement, the fact that the harmful effect of energy drink consumption is dose dependent was also proved in many studies in different body organs as per Salokhiddinovna[14], and Mihaiescu *et al*.[15].

The altered histological findings on the pancreas in the present study were nearly similar to that observed

by Qassim *et al*.[12], Abdel-Kareem *et al*.[16], and Memon *et al*.[13]. Prior research ascribed these alterations to the inflammatory reaction triggered by energy drinks, thst caused proinflammatory cytokines to be released that results in several degenerative alterations, oxidative stress, and cell death by apoptosis. Added to that, several studies have indicated that most adverse effects of energy drinks can be primarily attributed to the active ingredients, particularly caffeine. Caffeine causes an imbalance in the oxidant-antioxidant environment, leading to increased oxidant stress in the tissues. This is due to the increased production of iNOS and tumor necrosis factor-alpha (TNF-α), which ultimately results in cellular damage[17-18]. Moreover, the hyperglycemic state resulted from energy drink consumption causes the glycation of membrane phospholipids in the cell membrane or cytoplasmic organelles, resulting in lipid peroxidation (oxidative stress) and DNA damage in various organs[17-19]. These data were in accordance with the altered pancreatic cytoplasmic and nuclear cellular structure including the endocrine as well as the exocrine part of the pancreas in both subgroups of group II in the present study.

Fatty degeneration and the buildup of degenerative materials within the cytoplasm may be the cause of the acinar and islet cells' observed cytoplasmic vacuolation in this study as reported by Hałas *et al*.[20] and khayyat *et al*.[21]. Furthermore, it is possible that these vacuolations are a consequence of the oxidative stress caused by energy drink consumption. This oxidative stress can lead to damage of cell membranes and organelle membranes, resulting in increased permeability and failure of the energy dependent sodium (Na+) potassium (K+) pump ion pumps. Nuclear pyknosis seen in this study in the pancreatic acinar and islet cells was regarded as a sign of apoptosis as mentioned by Jiang *et al*.[22]. Apoptosis is characterized by DNA damage, shrinking, and nuclear chromatin clumping[23].

In the present study, some acinar cells were substituted with homogenous acidophilic substance giving the cell a glassy look in subgroup IIB. Kumar *et al*.[24], declared that this is a feature of necrosis, the necrosis of the cells was developed secondary to damage to the cell membrane and organelles membrane resulting from oxidative stress, which causes enzymes to seep out, causing cellular death, cytoplasmic content degradation, and cell membrane rupture, causing digestion and degradation of all cellular contents. One possible explanation for the retained secretion in the dilated pancreatic ducts found in this trial is the buildup of secretion brought on by pancreatic tissue damage and malfunction. These results were aligned with the results of Abonar *et al*.[25] who accredited these changes to the oxidative stress that damages the mitochondria, lowering ATP synthesis and the energy required by the duct cells to move secretions, which causes the ducts to dilate.

Congested dilated blood vessels and capillaries were seen in the current study in the exocrine and the endocrine pancreatic tissues. This agreed with the results of Abonar *et al*.[25], Qassim *et al*.[12], Abdel-Kareem *et al*.[16], and Memon *et al*.[13]. They ascribed these alterations to the microcirculatory disruptions brought on by the oxidative stress condition's excessive nitric oxide generation. However, Zahoor *et al*.[26] contributed the impaired endothelial function to be linked to the distinctive way, in which taurine interacts with caffeine and both are found in energy drinks.

The homogenous acidophilic material observed between the pancreatic acini in the present study might be explained according to Yassien and El-ghazouly[27] who mentioned that the endothelial cell membranes of the capillary endothelial cells are destroyed by pancreatic oxidative stress and lipid peroxidation products, which leads to increased permeability and edema.

The presence of different sizes of unilocular fat cells that appeared accumulated in subgroup IIB in the current study can be explained according to Navina and Singh[28] who mentioned that pancreatic inflammation leads to basolateral leakage of pancreatic enzymes. The basolateral leakage of lipases into fat during pancreatitis leading in subsequent lipolysis of fat and increased concentration of free fatty acids locally may be explained by this mechanism, which eventually accumulate in fat cells causing diffuse excess of intra-pancreatic fat deposition, a condition known as peri-fat acinar necrosis pancreas.

The TEM cellular changes that were observed in this study such as cytoplasmic vacuolations and rER dilatation might be explained according to Strayer *et al*.[29] who attributed these changes to be hallmarks of acute cell swelling caused by a lack of control over water intake. They also reported that energy drinks disrupt the cell volume regulatory mechanisms through the disruption of the permeability of the cell membrane, damage to the cell membrane, and reduction in ATP synthesis. As a result, As a result, the cell membrane's energy-dependent sodium pump is less active, resulting in the accumulation of water in the cell. The accumulation of water in the cisternae of the endoplasmic reticulum causes the endoplasmic reticulum to expand and leads to the formation of vacuoles in the cytoplasm. Similarly, Yassien and Elghazouly[27] assumed that oxidative stress was the cause of the vacuoles seen in the cytoplasm of the islet and acinar cells in their investigation, which results in excessive endoplasmic reticulum stress and severe endoplasmic reticulum dilatation. The swollen mitochondria seen in the current study may be explained according to Kumar *et al*.[24] who attributed the mitochondrial changes to Water and other solutes entering the mitochondrial matrix after damage to the mitochondria. They also noted that oxidative stress produced ROS, which formed a channel known as the mitochondrial permeability transition pore. The opening of this channel causes loss of mitochondrial membrane potential and pH alterations, leading to oxidative phosphorylation failure and gradual ATP depletion. This was manifested by mitochondrial swelling with breaking down of their cristae. Additionally, Herrington[30], reported that ATP depletion could interfere with intracellular calcium homeostasis with increased

cytoplasmic calcium concentration. This could activate several enzymes that have harmful effects on the cells. These enzymes include phospholipases (cause damage to membranes), proteases (break down cellular membranes and cytoskeletal proteins), endonucleases (cause DNA and chromatin fragmentation), and adenosine triphosphates (ATPase) that hast ATP depletion causing mitochondrial damage.

The current TEM studies showed a seemingly reduced quantity of zymogen granules in Red Bull administered subgroups. This was in accordance with Abonar *et al*.[25], They clarified this by saying that an increase in cytosolic that travels quickly to the nucleus causes a rise in nuclear calcium, this might prevent pancreatic acinar cells from its secretory function and result in fewer zymogen granules. Cytosolic calcium also travels quickly to the nucleus. Regarding the observed nuclear alterations and the apoptotic blebs in the present study. They are most likely caused by the alteration of the arrangement of actin cytoskeleton in these cells, triggered by the administration of caffeine, finally resulting in apoptosis as explained by Hałas *et al*.[20]. The TEM results of subgroup IIA and subgroup IIB revealed heterogenous electron dense structure most probably secondary lysosomes in the exocrine serous acinar cells and autophagosomes in β cells. Similarly, Hegazy *et al*.[31] found autophagosomes and heterogeneous electron-dense bodies in their experiment on energy drinks effect on rats' renal cortex. They illustrated that the heterogeneous electron dense bodies were secondary lysosomes, moreover they explained their presence to be an indicator of intracellular degeneration of macromolecules. Likewise, they explained the presence of the autophagosomes to be a result of autophagy of damaged cytoplasmic debris or degenerated organelles. El Ghazzawy *et al*.[32] and Nagano *et al*.[33] mentioned that lysosomes and lysosomal hydrolytic enzymes activation are necessary for ROS production and development lipid peroxidation. Additionally, these results is accounted for by the fact that coffee promotes autophagy, which in turn increases the number of autophagic vacuoles in the cytoplasm[34].

TEM examination in the current study showed Intercellular spaces expansion between exocrine acinar. This was in accordance with Haschek *et al*.[23] who mentioned that cells become isolated from one another when fluid seeps into and builds up in the intercellular gaps. Cellular junctions broke down during the process of acute oedema and cell damage. This happened because the cytoskeletal filaments were disrupted, the production of junctional adhesion molecules was suppressed, and consequently, the neighbouring cells separated from one another. Furthermore, cytoskeletal alterations are brought on by the oxidative stress that coffee administration causes[35].

In the present study, TEM examination revealed the presence of active fibroblasts and collagen fibrils. Haroun *et al*.[11] and Abd-Kareem *et al*.[16] confirmed the presence of collagen fibers in the pancreatic tissue of rats after energy drink consumption for four weeks. Also, Kassab and Tawfik[36] reported increase in collagen content in the submandibular salivary gland in rats after consumption of RedBull. Moreover, Ibrahem et al.[37] found that energy drinks caused marked liver fibrosis in rats after energy drink consumption for one month.

Free radicals were thought to cause pancreatic fibrosis because of their impact on the pancreatic stellate cells, which are crucial to the disease's development. This explanation is supported by evidence from several previous studies. Nevertheless, the precise mechanism of how the free radicals can stimulate the pancreatic stellate cells had not been completely clarified[38].

Group III in the present study experimented the possible withdrawal effects on the pancreas after ceasing the rats' consumption of energy drinks and letting them recover for 15 days without any further therapy. Both LM and TEM examined sections of the pancreas' exocrine and endocrine parts of subgroup IIIA showed marked regression of many of the altered microscopic architecture as compared to subgroup IIA. Regarding the Red Bull high dose withdrawal subgroup IIIB, both LM and TEM examined sections of the exocrine and endocrine pancreas showed partial regression of some of the altered microscopic architecture as compared to subgroup IIB. Regarding the decrease in the extent of the histological alterations after stopping Red Bull consumption in both subgroups of group III in the present study compared to Red Bull administered subgroups of group II, these results might point to attempts at gland regeneration following energy drink cessation. Similarly, Hulail *et al*.[5] noticed these findings in their study on the pancreas after ceasing the rats' consumption of energy drinks and letting them recover for 15 days without any further therapy.

Regarding the regeneration potentiality of the pancreatic tissue, it was observed in specific studies following 80-90% pancreatectomy or in pancreas affected by drug induced diabetes. Still, the exact involved regenerative mechanisms are not fully explained[39-40]. However, Arutyunyan *et al*.[41] stated that pancreatic stem cells appear to dwell in the epithelium of the duct and replenish both the exocrine and endocrine sections of the pancreas. These cells constitute a rapidly proliferating pool that develops into several cell types such as exocrine, ductal, and islet cells. However, any detectable presence of embryonic multipotent pancreatic progenitor cells in the adult mammalian pancreas is extremely unlikely.

CONCLUSION

Giving the preceding findings, it is possible to assume that energy drinks caused histological alterations in the exocrine and endocrine pancreatic tissues of adult male albino rats. These effects increased by increasing the dose of energy drink consumption. On the other hand, cessation of energy drinks intake could to some extent improve the pancreas histopathological and biochemical changes. The improvement was more in the lower dose administration of

energy drink, but still complete restoration of normal status did not occur.

RECOMMENDATIONS

Consumption of energy drinks is not highly recommended, particularly on consuming these beverages in high amounts.

CONFLICT OF INTEREST

There are no conflicts of interest.

REFERENCES

1. ARIFFIN, Hani', *et al*. Is the consumption of energy drink beneficial or detrimental to health: a comprehensive review?. Bulletin of the National Research Centre, 2022, 46.1: 163. https://doi.org/10.1186/s42269-022-00829-6
2. Somers KR, Svatikova A. Cardiovascular and Autonomic Responses to Energy Drinks-Clinical Implications. J Clin Med. 2020;9(2):431. Published 2020 Feb 5. doi:10.3390/jcm9020431
3. Ghozayel M, Ghaddar A, Farhat G, Nasreddine L, Kara J, Jomaa L. Energy drinks consumption and perceptions among University Students in Beirut, Lebanon: A mixed methods approach. PLoS One. 2020;15(4):e0232199. Published 2020 Apr 30. doi:10.1371/journal.pone.0232199
4. Jagim AR, Harty PS, Tinsley GM, *et al*. International society of sports nutrition position stand: energy drinks and energy shots. J Int Soc Sports Nutr. 2023;20(1):2171314. doi:10.1080/15502783.2023.2171314
5. Hulail, M., Qenawy, N., Abdel-Kareem, R., Mohamed, G. The Toxic Effect of Energy Drinks on The Structure of Pancreas of Adult Male Albino Rats. Zagazig University Medical Journal, 2020; 26(6): 1110-1117. doi: 10.21608/zumj.2019.11664.1205
6. Bano S. Histological effects of caffeinated energy drink consumption and its withdrawal on kidneys of experimental rats. Journal of Islamic International Medical College. 2020;2:128–32.
7. Rehman F, Islam ZU, Hameed U, Rehman S. The effect of energy drink on the pancreas of wistar albino rats--a microscopic study. Pakistan Armed Forces Medical Journal. 2020;70(2):524–8. https://doi.org/10.36570/jduhs.2020.3.984
8. Gaertner DJ, Hallman TM, Hankenson FC, Batchelder MA. Anesthesia and analgesia for laboratory rodents. In: Anesthesia and Analgesia in Laboratory Animals. Elsevier; 2008. p. 239–97.
9. Szabo J, Bruckner G, Koranyi L, Solymosi N, Mitchell G. Effect of macronutrient on plasma, liver and pancreatic metabolomics and their hierarchic weights in the metabolic network. Open Nutr J. 2018;12(1):40–58. Available from: http://dx.doi.org/10.2174/1874288201812010040
10. Suvarna SK, Layton C, Bancroft JD. Theory and practice of histological techniqueseighth. UK: Elsevier Health Sci; 2019.
11. Haroun H, Mohamed E, El Shahat AER, Labib H, Atef M. Adverse effects of energy drink on rat pancreas and the therapeutic role of each of bone marrow mesenchymal stem cells and Nigella Sativa oil. Folia Morphol (Warsz). 2020;79(2):272–9. http://dx.doi.org/10.5603/FM.a2019.0069
12. Qassim AH, Alsammak MA, Ayoob AA. Histopathological changes in kidney and pancreas induced by energy drinks in adult male rats. Iraqi J Vet Sci. 2022;36(1):111–6. http://dx.doi.org/10.33899/ijvs.2021.129435.1647.
13. Memon SB, Almani SA, Baloch S, Hanif S, Asad A, Qureshi R. Histomorphometric effects of energy drink consumption on pancreatic tissue of albino wistar rats. Prof Med J. 2023;30(06):764–70. http://dx.doi.org/10.29309/tpmj/2023.30.06.7399
14. Salokhiddinovna HY. Morphological Features of the Human Body in Energy Drink Abuse. European Journal Of Innovation In Nonformal Education. 2023;3(5):51–3.
15. Mihaiescu T, Turti S, Souca M, Muresan R, Achim L, Prifti E. Caffeine and Taurine from Energy Drinks-A Review. Cosmetics. 2024;11(1).
16. Abdel-Kareem R, Hulail M, Mohamed G, Qenawy N. Structural and biochemical changes induced by energy drinks in the pancreas of adult male albino rats: Ameliorative effect of green tea. Egypt J Histol. 2022;0(0):0–0. http://dx.doi.org/10.21608/ejh.2022.127251.1657
17. Ayuob N, ElBeshbeishy R. Impact of an Energy Drink on the Structure of Stomach and Pancreas of Albino Rat: Can Omega-3 Provide a Protection?. PLoS One. 2016;11(2):e0149191. Published 2016 Feb 19. doi:10.1371/journal.pone.0149191
18. Taiwo OI, Adesokan AA. Effects of High Doses of Exogenous Taurine, Caffeine, and Taurine-Caffeine Combination on Biochemical, Haematological and Histologic Parameters of Adult Rabbits. Am J Biochem. 2017;7(4):63–72.
19. Ekaluo UB, Uno UU, Edu NE, Ekpo PB, Etta SE. Effect of Trevo dietary supplement on caffeine induced oxidative stress in albino rat models. The Pharmaceutical and Chemical Journal. 2016;3(2):92–7.
20. Hałas M, Izdebska M, Klimaszewska-Wiśniewska A, Gagat M, Radciniewska D, Glińska A, *et al*. Caffeine induces cytoskeletal changes and cell death in H1299 cells. Open Life Sci. 2014;9(8):727–38. http://dx.doi.org/10.2478/s11535-014-0315-0

21. Khayyat L, Sorour JMA, Essawy A, Al Rawi M. Histological, ultrastructural and physiological studies on the effect of different kinds of energy drinks on the liver of Wistar albino rat. Int J Res Sci. 2015;1(2):15. http://dx.doi.org/10.24178/ijrs.2015.1.2.15

22. Jiang L, Zhang X, Zheng X, Ru A, Ni X, Wu Y, *et al*. Apoptosis, senescence, and autophagy in rat nucleus pulposus cells: Implications for diabetic intervertebral disc degeneration: APOPTOSIS, SENESCENCE AND AUTOPHAGY IN INTERVERTEBRAL DISC. J Orthop Res. 2013;31(5):692–702. http://dx.doi.org/10.1002/jor.22289

23. Haschek WM, Bolon B, Rousseaux CG, Wallig MA. Fundamentals of Toxicologic Pathology. Elsevier Science; 2018; 59-81.

24. Kumar V., Abbas A.K., Aster J.C. and Perkins J.A. Robbins Basic Pathology, Tenth edition, Elsevier, Canada. 2018; p: 31-56 and p: 679- 689.

25. Abonar M, Aboraya A, Elbakary N, Elwan W. Effect of energy drink on the pancreas of adult male albino rat and the possible protective role of avocado oil. Histological and immuno-histochemical study. Egyptian Journal of Histology. 2022;45(2):386–403. DOI: 10.21608/ejh.2021.59941.1425

26. Zahoor S, Rehman RS ur, Mahboob HM, Asif S, Malik U, Jehangir HMS. Association between energy drinks and cardiovascular events: A literature review. Pak Hear J. 2021;53(4). http://dx.doi.org/10.47144/phj.v53i4.1997

27. Yassien R, El-ghazouly D. TThe effect of Atorvastatin on the pancreas of adult male albino rats and the possible protective role of Resveratrol (Histological, Immunohistochemical and Biochemical study). Egypt J Histol. 2020;0(0):0–0. http://dx.doi.org/10.21608/ejh.2020.21365.1218

28. Navina S, Singh VP. Relationship between obesity and pancreatitis. Pancreapedia: The Exocrine Pancreas Knowledge Base. 2015. DOI: 10.3998/panc.2015.18

29. Strayer DS, Rubin E, Saffitz JE, Schiller AL. Rubin's Pathology: Clinicopathologic Foundations of Medicine. Wolters Kluwer Health. 2015;3–55.

30. Herrington CS. Muir's Textbook of Pathology. CRS press, U.S.A; 2014; 391-420.

31. Hegazy K, Elswaidy N, Kassab A, Elbakary N. The effect of energy drinks and their withdrawal on the renal cortex of adult male albino rats. Histological, ultrastructural and morphometric study. Egypt J Histol. 2022;0(0):0–0. http://dx.doi.org/10.21608/ejh.2022.138865.1686

32. El Ghazzawy IF, Meleis AE, Farghaly EF, Solaiman A. Histological study of the possible protective effect of pomegranate juice on bisphenol-A induced changes of the caput epididymal epithelium and sperms of adult albino rats. Alex J Med. 2011;47(2):125–37. http://dx.doi.org/10.1016/j.ajme.2011.06.006

33. Nagano Y, Matsui H, Shimokawa O, Hirayama A, Nakamura Y, Tamura M, *et al*. Bisphosphonate-induced gastrointestinal mucosal injury is mediated by mitochondrial superoxide production and lipid peroxidation. J Clin Biochem Nutr. 2012;51(3):196–203. http://dx.doi.org/10.3164/jcbn.12-41

34. Dirks-Naylor AJ. The benefits of coffee on skeletal muscle. Life Sci. 2015;143:182–6. http://dx.doi.org/10.1016/j.lfs.2015.11.005

35. Orabi AI, Shah AU, Ahmad MU, Choo-Wing R, Parness J, Jain D, *et al*. Dantrolene mitigates caerulein-induced pancreatitis in *vivo* in mice. Am J Physiol Gastrointest Liver Physiol. 2010;299(1):G196-204. http://dx.doi.org/10.1152/ajpgi.00498.2009

36. Kassab A, Tawfik S. Effect of a caffeinated energy drink and its withdrawal on the submandibular salivary gland of adult male albino rats: A histological and immunohistochemical study. Egypt J Histol. 2018;41(1):11–26. http://dx.doi.org/10.21608/ejh.2018.7518

37. Ibrahem N, Reda S, Mekawy N. Hepatic Changes under the Effect of Red Bull Energy Drinks and its Withdrawal in Adult Male Albino Rats (Histological and Immunohistochemical Study). Journal of Medical Histology. 2022;6(1):34–43. DOI: 10.21608/jmh.2022.170936.1106

38. Jin G, Hong W, Guo Y, Bai Y, Chen B. Molecular Mechanism of Pancreatic Stellate Cells Activation in Chronic Pancreatitis and Pancreatic Cancer. J Cancer. 2020;11(6):1505-1515. Published 2020 Jan 14. doi:10.7150/jca.38616

39. Qadir MMF, Álvarez-Cubela S, Klein D, *et al*. Single-cell resolution analysis of the human pancreatic ductal progenitor cell niche [published correction appears in Proc Natl Acad Sci U S A. 2021 Feb 9;118(6):e2025819118. doi: 10.1073/pnas.2025819118.]. Proc Natl Acad Sci U S A. 2020;117(20):10876-10887. doi:10.1073/pnas.1918314117

40. Cozzitorto C, Mueller L, Ruzittu S, *et al*. A Specialized Niche in the Pancreatic Microenvironment Promotes Endocrine Differentiation. Dev Cell. 2020;55(2):150-162.e6. doi:10.1016/j.devcel.2020.08.003

41. Arutyunyan IV, Fatkhudinov TK, Makarov AV, Elchaninov AV, Sukhikh GT. Regenerative medicine of pancreatic islets. World J Gastroenterol. 2020;26(22):2948-2966. doi:10.3748/wjg.v26.i22.2948

الملخص العربى

تأثير الجرعات المختلفة من مشروبات الطاقة على بنكرياس ذكور الجرذان البيضاء البالغة وتأثير سحبها. دراسة بالمجهر الضوئي والإلكتروني

مريم روماني، سارة عبد الجواد السباعي، عبير عبد المحسن عبد الصمد، نجوى قسطندي قليني

قسم الأنسجة كلية الطب، جامعة عين شمس، مصر

مقدمة: مشروبات الطاقة هي مشروبات غير كحولية رائجة تحتوي بشكل أساسي على الكافيين، الذي زاد استخدامه مؤخرًا بكثرة لتعزيز التركيز. تشير العديد من الدراسات الحديثة إلى أن مشروبات الطاقة التي تحتوي على الكافيين يمكن أن تؤثر سلبا على بنية ووظائف العديد من الأعضاء.

الهدف من هذه الدراسة: دراسة تأثير الجرعات المختلفة من مشروب ريد بول - كمثال على مشروبات الطاقة المحتوية على الكافيين - على بنكرياس ذكور الجرذان البيضاء البالغة وآثار سحبه، باستخدام التقنيات المجهرية.

المواد وطرق البحث: استُخدم في هذه الدراسة ثلاثة وستين من ذكور الجرذان البيضاء بمتوسط وزن حوالي ٢٠٠ جرام. قُسمت الجرذان عشوائياً إلى ٣ مجموعات: المجموعة الأولى (المجموعة الضابطة)، والمجموعة الثانية (التي تناولت مشروب الطاقة) قُسِّمت إلى مجموعتين: المجموعة الأولى (IIA) والأخرى (IIB)، حيث أعطيت ريد بول عن طريق الفم بجرعة ٧,٥ مل/كجم/يوم و١٥ مل/كجم/يوم على التوالي مرة واحدة يوميًا لمدة ٤ أسابيع. أما المجموعة الثالثة (مجموعة سحب مشروبات الطاقة) فقد قُسِّمت إلى مجموعتين: المجموعة الثالثة (IIIA) والأخرى (IIIB)، حيث أعطيت ريد بول بجرعة مماثلة للمجموعتين الفرعيتين IIA وIIB على التوالي، ثم تُركتا لمدة ١٥ يومًا دون أي علاج. فحصت العينات في نهاية التجربة بالمجهر الضوئي والمجهر الإلكتروني النافذ.

النتائج: أظهر هذه الدراسة أن تناول ريد بول سبب اضطرابًا موضعيًا في بنية كلٍّ من الغدد القنوية والغدد الصماء في البنكرياس. كانت هذه التغيرات مرتبطة بالجرعة، إذ كانت أكثر وضوحًا وامتدت لتشمل مساحات واسعة من فصيصات البنكرياس في المجموعة الفرعية التي تناولت مشروب الطاقة بجرعات عالية. أدى التوقف عن تناول ريد بول إلى تحسن جزئي في هذه التغيرات، لا سيما لدى المجموعة الفرعية التي تناولت جرعات منخفضة.

الخلاصة: تسبب استهلاك مشروبات الطاقة في حدوث تغيرات نسيجية مرتبطة بالجرعة في بنية البنكرياس، وتسبب سحبها في تحسن جزئي لهذه التغيرات، وخاصة في الفئران التي استهلكت جرعة منخفضة من مشروبات الطاقة.