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ORIGINAL RESEARCH

Effect of high soda beverage consumption on the fertility of naive male mice

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Abstract

Background: The consumption of soda drinks has risen in recent years. Excessive intake of soda beverages has been linked to numerous detrimental effects. This study sought to examine the impact of soda consumption on the fertility of male mice. **Methods**: Twenty male mice were allocated to a control group and a soda group. After four months, blood samples from mice were used to estimate male sex hormones, prolactin, adropin, endothelin-1, and nitric oxide (NO). Additionally, the testis was obtained for semen analysis and assessment of testicular lipid peroxidation (LPO), reduced glutathione (GSH), catalase, glutathione peroxidase (GSH-Px), and superoxide dismutase (SOD) levels. Furthermore, testicular tissue NO synthase immunohistochemistry was estimated for the mice in the two groups, while the semen was obtained from caudae epididymides. **Results**: Soda drinking significantly raised body weight and lowered the testicular weight/body weight ratio. Compared with the control group, the soda group had increased endothelin-1 and NO and decreased testosterone and adropin levels. Soda consumption lowered testicular catalase, GSH-Px, and SOD and elevated LPO. Regarding semen analysis, soda significantly decreases sperm count and motility, whereas it increases abnormal sperm morphology. Soda drinking significantly increased testicular nitric oxide synthase (NOS) immunohistochemistry compared with the control group. **Conclusions**: The study concludes that the high consumption of soda beverages reduces the fertility of male mice via the induction of oxidative stress. This stress diminishes sperm counts and motility while augmenting the prevalence of defective sperm. In addition, soda drinking decreases testosterone and adropin levels.

Keywords

Soda beverage; Fertility; Semen analysis; Oxidative stress; Antioxidants

Efecto del alto consumo de bebidas gaseosas sobre la fertilidad de ratones machos naive

Resumen

Antecedentes: El consumo de bebidas gaseosas ha aumentado en los últimos años. La ingesta excesiva de bebidas gaseosas se ha relacionado con numerosos efectos perjudiciales. Este estudio buscó examinar el impacto del consumo de refrescos en la fertilidad de ratones macho. **Métodos**: Se dividieron veinte ratones macho en un grupo de control y un grupo de refrescos. Después de cuatro meses, se utilizaron muestras de sangre de ratones para estimar las hormonas sexuales masculinas, prolactina, adropina, endotelina-1 y óxido nítrico (NO). Además, se obtuvieron los testículos para el análisis de semen y la evaluación de los niveles de peroxidación lipídica testicular (LPO), glutatión reducido (GSH), catalasa, glutatión peroxidasa (GSH-Px) y superóxido dismutasa (SOD). Además, se estimó la inmunohistoquímica de la NO sintasa del tejido testicular para los ratones de los dos grupos, mientras que el semen se obtuvo de los epidídimos de la cola de caballo. **Resultados**: El consumo de refrescos aumentó significativamente el peso corporal y redujo la relación peso testicular/peso corporal. En comparación con el grupo de control, el grupo de refrescos tuvo un aumento de endotelina-1 y NO y una disminución de los niveles de testosterona y adropina. El consumo de refrescos redujo la catalasa testicular, GSH-Px y SOD y elevó la LPO. En cuanto al análisis de semen, los refrescos disminuyen significativamente el recuento y la motilidad de los espermatozoides, mientras que la morfología anormal de los espermatozoides aumenta. El consumo de refrescos aumentó significativamente la inmunohistoquímica testicular nitric oxide synthase (NOS) en comparación con el grupo de control. **Conclusiones**: El estudio concluye que el elevado consumo de bebidas gaseosas reduce la fertilidad de ratones macho mediante la inducción de estrés oxidativo. Este estrés disminuye el recuento y la motilidad de los espermatozoides al tiempo que aumenta la prevalencia de espermatozoides defectuosos. Además, el consumo de refrescos disminuye los niveles de testosterona y adropina.

Palabras Clave

Bebida gaseosa; Fertilidad; Análisis de semen; Estrés oxidativo; Antioxidantes

1. Introduction

One in six couples attempting conception have infertility. Infertility is defined as the inability to conceive after 12 months of unprotected intercourse [1]. Male factors are responsible for 58% of these occurrences, including one-third of instances attributable to a combination of male and female causes [2]. Genetics account for merely 10%–15% of male infertility, underscoring increasing evidence showing that testicular function, particularly spermatogenesis, is susceptible to environmental factors such as chemicals, air pollution, and dietary influences [3]. About 20% of infertility cases indicate that the male is solely responsible, while another 30% to 40% involve the male as a contributing factor. According to the World Health Organization, male infertility is defined as the incapacity of a male to impregnate a fertile female after a minimum of one year of consistent unprotected intercourse. The preliminary assessment comprises a comprehensive sexual history and physical examination, along with two distinct semen analyses. If anomalies are detected, then hormonal tests and an optional scrotal ultrasound may be conducted. This process is typically adequate for a first assessment of the type and severity of the underlying issue [4]. Male fertility is influenced by various factors, encompassing reversible and irreversible conditions. Age, environmental pollutants, hereditary diseases, medication, and systemic diseases of each partner are additional factors that should be considered. Identifying contributing factors, administering treatment for reversible conditions, determining eligibility for assisted reproductive techniques, and providing counseling for irreversible and untreatable conditions are the primary objectives of evaluating male infertility [5].

Soda is a beverage with the highest sales globally [6, 7]. Soda comprises numerous chemical constituents, including sugar (sucrose or high-fructose corn syrup), phosphoric acid, and caffeine, which have been linked to infertility and negative birth outcomes [8]. In recent years, significant concerns have emerged with regard to the impact of soda on human health. Chronic cola drinking may have adverse effects such as weakening teeth [9], osteoporosis [10], hypokalemic myopathy [11], metabolic syndrome, type 2 diabetes mellitus [12], and chronic kidney disease [13]. Various studies indicate that excessive soda intake may elevate the risk of reproductive disorders, such as reduced fetal growth, preterm delivery, and abortion [14]. To our knowledge, limited research has examined the relationship between soda consumption and testicular function.

Although negative relationships with one or more semen parameters have been reported, the correlations differed among studies. One study identified an inverse correlation with advancing sperm motility [15], but another investigation indicated diminished semen volume associated with increased soda consumption. Moreover, these studies indicated that sperm counts are diminished in men who consume one liter of soda or more daily [16]. The present study aimed to investigate the correlation between high soda consumption and infertility in naive mice.

2. Materials and methods

2.1 Trial design

Twenty-two-month-old male Bagg Albino (BALB/c) mice that weigh between twenty and twenty-five grams were purchased from the animal facility of Umm Al-Qura University. The mice were accommodated in a standard rodent enclosure with woodchip bedding in a spacious, well-ventilated room maintained at

25 °C and subjected to a 12-hour light/dark cycle. During the trial, all mice received a regular mouse meal and tap water. These mice were randomly assigned to two groups (10 mice each) after two weeks of acclimation.

1. The first group was assigned as a control group. The mice in this group received no treatment.

2. The second group was assigned as a soda group. In this group, mice were given 14.3 mL/kg body weight per day of soda orally via intragastric gavage [16].

Both groups consumed the same quantity of ordinary rodent diets daily during the four months of the trial.

2.2 Body weight and testicular weight measurement

The body weight of each mouse in the two groups was recorded after four months of the experiment by utilizing a digital scale (OHAUS, Scout Pro SPU601, China). Moreover, the right testis of each mouse was used for the measurement of testicular weight.

2.3 Blood sample collection

Four months later, all mice were anesthetized using chloroform, and then the blood samples were drawn in a plain tube from the mouse's retro-orbital venous plexus.

2.4 Testicular collection

After four months, all the mice were euthanized, and their testes were swiftly removed for the assessment of testicular weight, preparation of testicular homogenate, and immunohistochemical examination. Each mouse's testes were weighed to calculate its testicular weight/body weight ratio.

2.5 Preparation of testicular homogenate

A testicular homogenate was made using an (OMNI International Inc. 2 mL bead kit, Cat. Number 10032-364, Seoul, Korea). Tiny left testis fragments, radio-immunoprecipitation assay buffer, and a protease inhibitor were added to a 2 mL microtube filled with beads. A Bead Ruptor 12 (Homogenizer, OMNI International Inc., Seoul, South Korea) was used to homogenize the microtube for two minutes. It was then centrifuged for 30 min at 8 °C and 15,000 rpm (SIGMA 1-14 k). Finally, the supernatant was kept at −20 °C until analysis [17].

2.6 Semen analysis

The vas deferens and caudae epididymides were dissected, and then the sperm was collected by cutting the vas deferens using a forceps or a cannula. In a sterile Petri dish, the vas deferens was incubated for 15 min with 0.9 mL of Roswell Park Memorial Institute (RPMI)-1640 solution.

2.6.1 Sperm count

Ten microliters of diluted sperm suspension were introduced into the counting chamber of a hemocytometer. Counting commenced at 200× magnification using a light microscope following a 5-minute incubation time [18].

2.6.2 Sperm morphology

In a test tube, 10 mL of 1% eosin and nigrosine stain was applied to a 40 mL sperm solution for 1 hour at room temperature. One drop of suspension was spotted at 400× magnification on a slide. Each mouse had 200 spermatozoa examined [19].

2.6.3 Sperm motility

In order to evaluate motility, the sperm suspension was examined under a light microscope at a magnification of 1000×. Every spermatozoon is classified according to its motility into:

1. Progressive motility (rapid motility): spermatozoa that are actively moving, either in a linear or circular motion, regardless of their speed.

2. Non-progressive motility (slow motility): all other patterns of motility that lack progression, such as swimming in small circles, the flagellar force barely displacing the head, or when only a flagellar pulse is present.

3. Immotile: no movement.

The motile sperm % was determined using the following formula:

Percentage of dead sperm = (number of dead sperm times 100)/total number of sperm studied [20].

2.7 Testicular immunohistochemistry for the detection of nitric oxide synthase activity

The testicular tissue of the left testis was fixed for 24 hours in 10% formaldehyde, before being transferred to 70% ethanol. After that, the tissues were embedded in paraffin blocks, and 5 μm slices were cut out of them. After being in xylene for 5 minutes, the pieces were rehydrated for 10 minutes in 100% ethanol and then 95% ethanol. They were then heated in a microwave for 10 minutes and left to cool for 30 minutes. After being washed three times in distilled water for five minutes each, the slides were labeled with NOS antibodies. Lastly, a wash buffer was used three times for five minutes each time to clean the slides. Under a light microscope, the Sulzbacher scoring system was used to qualitatively evaluate the slides.

2.8 Testicular homogenate antioxidant and oxidative stress parameter estimation

In testicular homogenate, the reduced glutathione (GSH), lipid peroxidation products (LPO), superoxide dismutase (SOD), glutathione peroxidase (GSH-Px), and catalase (Elabscience kits, E-EL-H6156, Houston, TX, USA) were estimated by a Varioskan LUX instrument (ELISA machine, Thermo Fisher Scientific, Waltham, MA, USA) using colorimetric assay.

2.9 Sex hormones and prolactin measurement

Serum luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin, and testosterone were measured using competitive Enzyme Linked Immunosorbent Assay (ELISA) with the Varioskan LUX instrument (Thermo Fisher Scientific, USA) [21].

2.10 Serum adropin, endothelin-1, and NO estimation

Serum adropin, endothelin-1, and NO levels were measured using the ELISA technique with the Varioskan LUX instrument (Thermo Fisher Scientific, USA) [22].

2.11 Statistical analysis

Prism software version 8 (Dotmatic, Boston, MA, USA) was used for statistical analysis. All data were expressed as mean ± Standard Deviation (SD), and all comparisons of body weight, semen analysis parameters, sex hormones, oxidative and antioxidant parameters, and other chemical parameters between the two groups were performed using the *t*-test. The correlation between parameters in soda group was done by Spearman test. The significance level was set at $p < 0.05$.

3. Results

3.1 Body and testicular weights

Table 1 presents the body weight, testicular weight, and testicular weight/body weight ratio of the two groups after 4 months. Over 16 weeks, the body weight of mice in the soda group markedly exceeded that of the control group ($p < 0.001$). Moreover, the mice in the soda group had significantly lower testicular weight/body weight ratio compared to the mice in the control group ($p < 0.05$). The testicular weight showed a statistically non-significant decrease in the mice in the soda group.

TABLE 1. Bodyweight, testicular weight, and testicular weight/body weight ratio of mice in the two groups. The comparison was done using *t*-test.

Parameters	Control group n = 10	Soda group n = 10
Body weight (g)	30.61 ± 2.48	41.14 ± 5.30***
Testis weight (g)	1.31 ± 0.07	1.29 ± 0.05
Testicular weight/ body weight ratio	0.043 ± 0.003	0.033 ± 0.002*

*$p < 0.05$; ***$p < 0.001$.

3.2 Oxidative stress and antioxidant parameters in testicular homogenate

Table 2 displays LPO, GSH, catalase, GSH-Px, and SOD levels in the testicular tissue of mice in the two groups. The LPO level was markedly increased in the soda group compared with the control group ($p < 0.001$). Soda consumption significantly decreased antioxidant enzymes (catalase, GSH-Px, and SOD) compared with the control group ($p < 0.001$). Furthermore, GSH was markedly reduced in the soda group relative to the control group ($p < 0.001$).

3.3 Male sex hormones and prolactin

The sex hormones and prolactin levels are displayed in Table 3. In comparison to the control group, testosterone levels were markedly reduced in the soda group ($p < 0.001$). FSH, LH, and prolactin exhibited no statistically significant variations between the two groups.

TABLE 2. Lipid peroxidation (LPO), reduced glutathione (GSH), catalase, glutathione peroxidase (GSH-Px), and superoxide dismutase (SOD) in the testis of mice in the two groups. The comparison was done using *t*-test.

Parameters	Control group n = 10	Soda group n = 10
LPO (nmol/g)	22.31 ± 4.62	45.29 ± 6.13***
GSH (nmol/g)	4.37 ± 0.84	0.97 ± 0.01***
Catalase (U/g)	43.25 ± 4.06	10.65 ± 2.36***
GSH-Px (U/g protein)	0.48 ± 0.02	0.09 ± 0.03***
SOD (µg/g protein)	21.20 ± 4.38	11.07 ± 2.44***

***$p < 0.001$.

TABLE 3. Male sex hormones and prolactin of mice in the two groups. The comparison was done using *t*-test.

Parameters	Control group n = 10	Soda group n = 10
FSH (ng/mL)	0.023 ± 0.0001	0.022 ± 0.0001
LH (ng/mL)	0.027 ± 0.0005	0.026 ± 0.0002
Testosterone (ng/mL)	2.59 ± 0.03	0.18 ± 0.01***
Prolactin (ng/mL)	1.79 ± 0.25	1.93 ± 0.23

***$p < 0.001$. *FSH: follicle-stimulating hormone; LH: luteinizing hormone.*

3.4 Semen analysis

The semen analysis of the two groups is displayed in Table 4. Sperm count was significantly decreased in the soda group compared with the control group ($p < 0.001$). Moreover, the soda group exhibited a substantial decrease in sperm motility when compared with the control group ($p < 0.001$). In addition, sperm distortion is significantly increased with soda consumption ($p < 0.001$).

TABLE 4. Semen analysis of mice in the two groups. The comparison was done using *t*-test.

Parameters	Control group n = 10	Soda group n = 10
Sperm count (10^6)	104 ± 11	62 ± 10***
Progressive motile (%)	81 ± 18	33 ± 4***
Non-progressive motile (%)	8 ± 4	41 ± 7***
Immotile (%)	11 ± 3	26 ± 3**
Normal sperm (%)	88 ± 12	41 ± 9***
Abnormal sperm (%)	12 ± 3	59 ± 12***

**$p < 0.01$; ***$p < 0.001$.

3.5 NO, adropin, and endothelin-1 levels

Fig. 1 displays NO, adropin, and endothelin-1 levels in the two groups. Adropin concentration was considerably reduced in the soda group compared with the control group ($p < 0.01$). Furthermore, the levels of endothelin-1 and nitric oxide were significantly elevated in the soda-consuming mice compared to the control group mice ($p < 0.001$).

3.6 Correlation between parameters in the soda group

In the soda group, a positive correlation exists between testosterone and sperm count (Fig. 2), as well as between testosterone and SOD ($p < 0.01$).

Moreover, a positive correlation observed between sperm count and SOD ($p < 0.05$) (Fig. 3).

Negative associations were seen between LPO and testosterone ($p < 0.001$), as well as between LPO and sperm count ($p < 0.001$) (Fig. 4).

Adropine positively correlated with testosterone levels ($p < 0.05$), whereas endothelin-1 showed a negative correlation with testosterone levels and sperm count ($p < 0.05$).

3.7 NOS immunoreactivity in testicular tissue

Figs. 2,3 present the immunohistochemical reactivity of NOS in the two groups. The NOS immunohistochemical reactivity was significantly increased in mice ingested soda with a score of 5 (Fig. 5A) compared with the control group with a score of 2 (Fig. 5B).

4. Discussion

The failure of a couple to get pregnant after having regular, unprotected sex for a year is known as infertility [2]. About 20% of infertility cases indicate that the male is solely responsible, while another 30% to 40% involve the male as a contributing factor. The preliminary assessment comprises a comprehensive sexual history and physical examination, along with two distinct semen analyses. If anomalies are detected, then hormonal tests and an optional scrotal ultrasound may be conducted. This process is typically adequate for a first assessment of the type and severity of the underlying issue [23]. Male fertility is influenced by various factors, encompassing reversible and irreversible conditions. Age, environmental pollutants, hereditary diseases, medication, and systemic diseases of each partner are additional factors that should be considered. Identifying contributing factors, administering treatment for reversible conditions, determining eligibility for assisted reproductive techniques, and providing counseling for irreversible and untreatable conditions are the primary objectives of evaluating male infertility [2]. A previous study conducted on 189 American men aged between 18–22 revealed that men who consumed more than 200 mL of sugar-sweetened beverages daily exhibited a reduced proportion of progressive sperm motility; however, no additional correlations with semen quality or reproductive hormones were identified [15]. A Chinese study similarly revealed an inverse dose—response relationship between cola consumption and semen volume

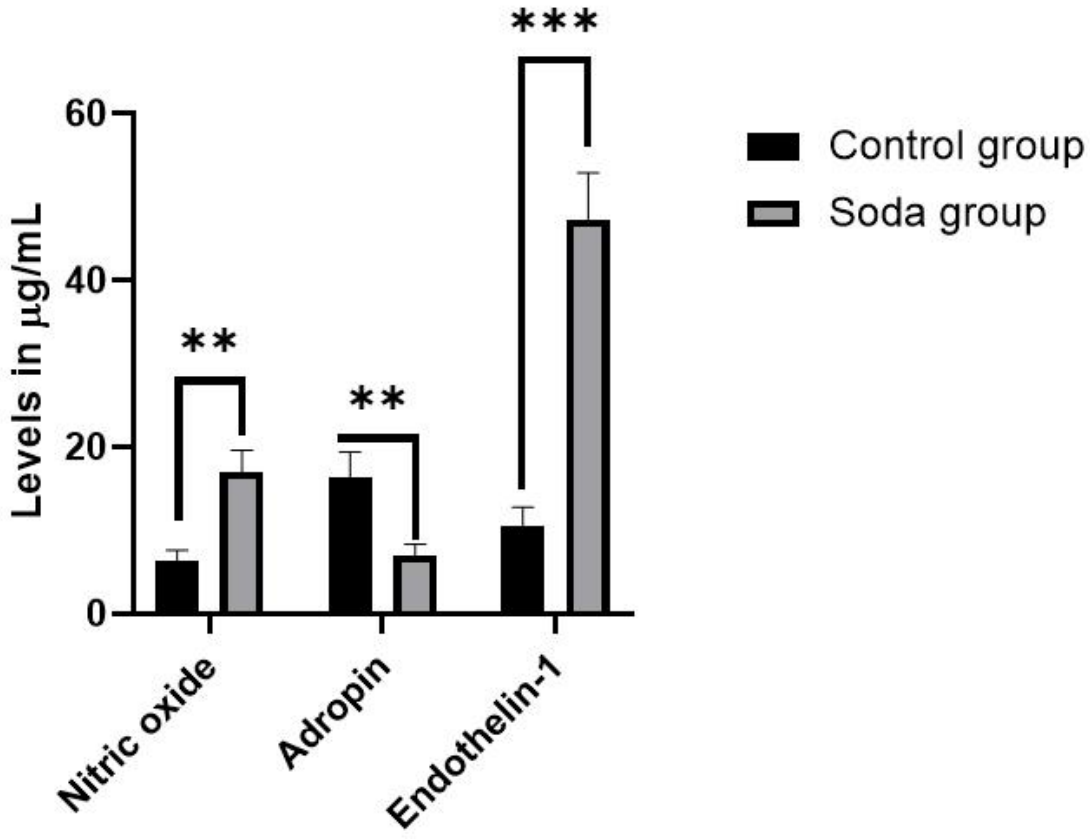

FIGURE 1. Nitric oxide, adropin, and endothelin-1 levels in the two. $^{**}p < 0.01$; $^{***}p < 0.001$.

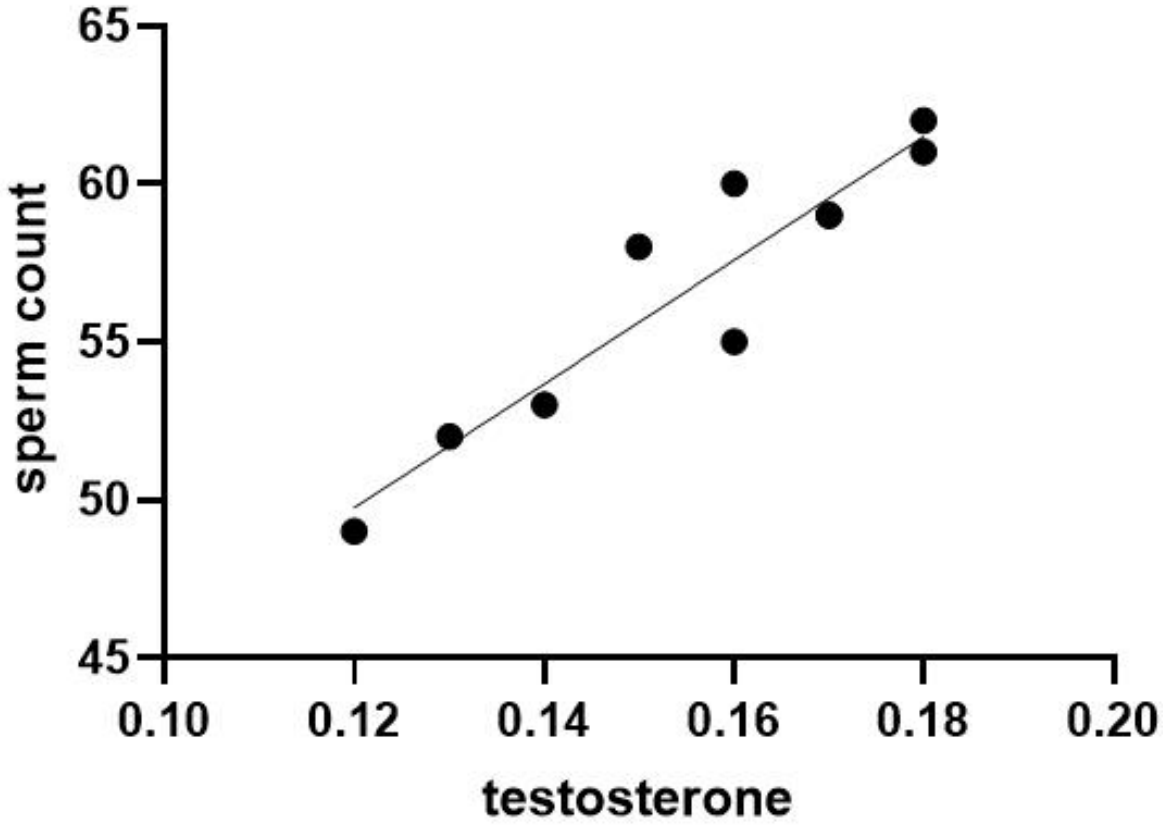

FIGURE 2. Positive correlation between testosterone levels and sperm count in the soda group.

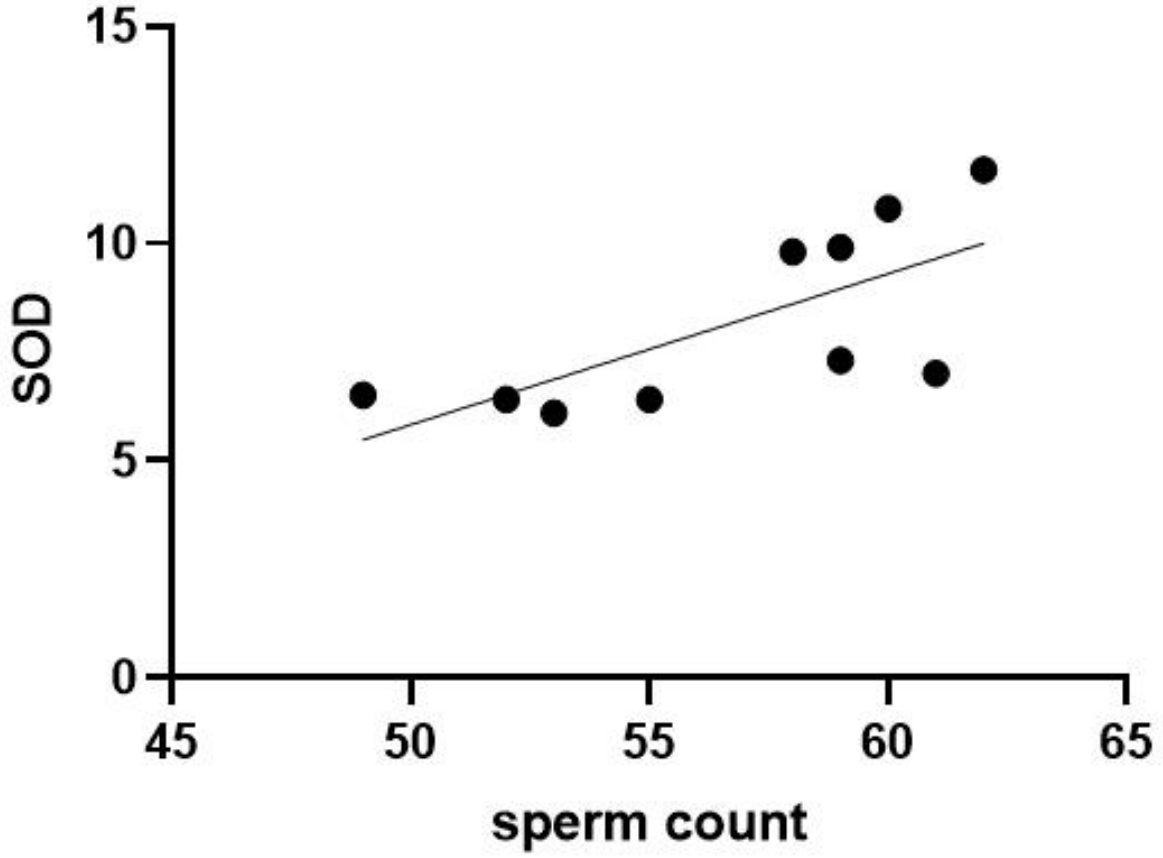

FIGURE 3. Positive correlation between SOD and sperm count in soda group. SOD: superoxide dismutase.

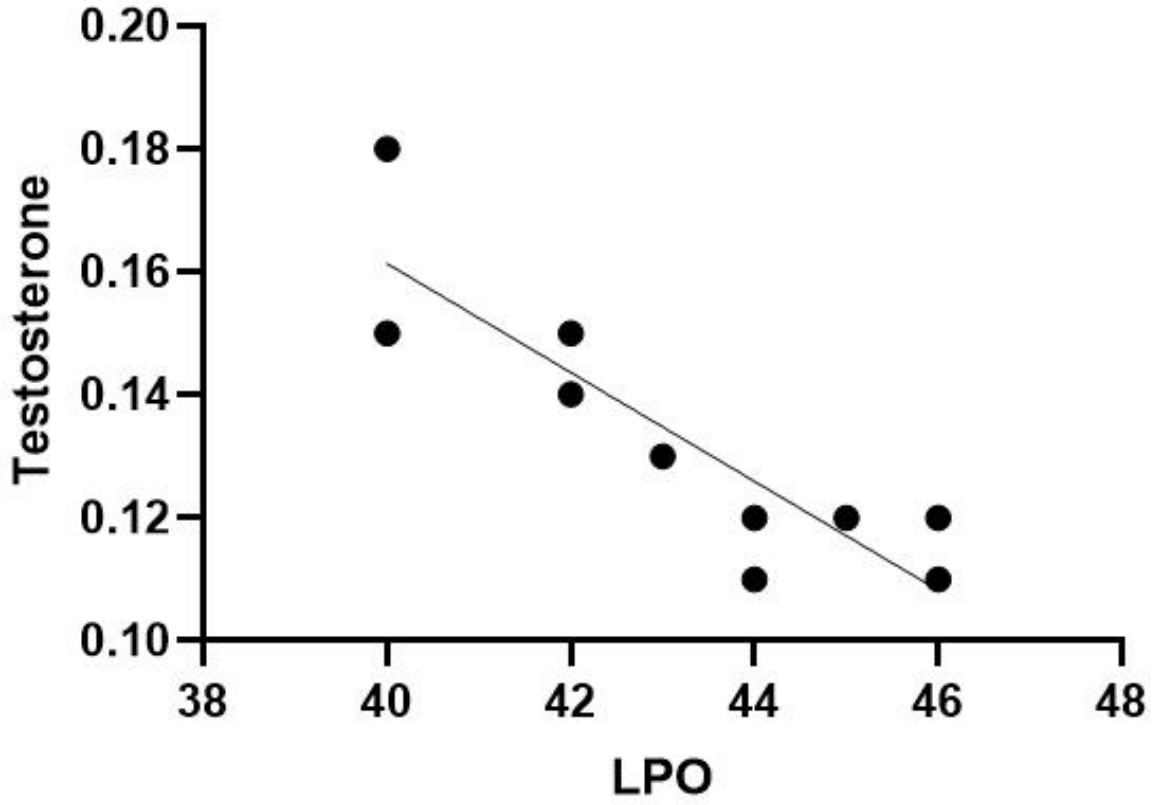

FIGURE 4. Negative correlation between testosterone levels and LPO in the soda group. LPO: lipid peroxidation.

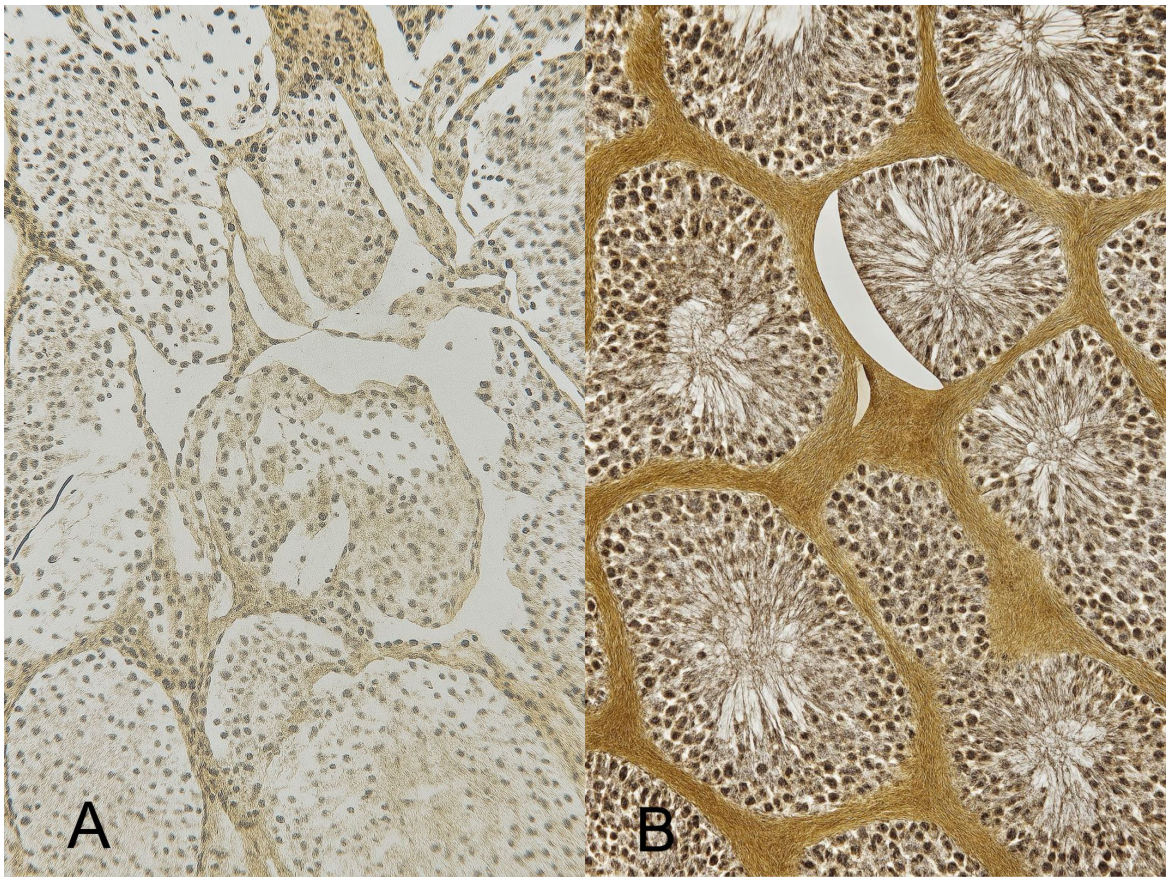

FIGURE 5. Testicular immunohistochemistry activity of NOS. (A) the testicular tissue of the control group. (B) the testicular tissue of the soda group.

[16]. Furthermore, a study that was conducted on 2544 Danish young men found that those who consumed more than 14 bottles of soda per week had reduced semen volume and total sperm count compared with non-consumers, a finding that was not attributable to caffeine use [23]. Hatch *et al.* [8] observed that men's consumption of soft drinks was linked to decreased fecundability in couples attempting to conceive, irrespective of women's soft drink consumption [24]. They also found that the association of soft drink consumption and low sperm count was observed at low intake levels and expanded in magnitude with greater intake. Moreover, another recent study is consistent with Hatch's findings, which state that cola intake leads to low sperm count and increases consumption levels [25]. Furthermore, Ruff *et al.* [25] (2013) found that the consumption of a fructose/glucose solution analogous to a soft drink to male mice led to diminished male fertility, as demonstrated by a reduced number of offspring [26]. The present study revealed that drinking soda results in weight gain. The testicular weight in the soda group is reduced compared with the control group. However, the difference is not statistically significant. The drop in testicular weight is clearly reflected in the testicular weight/body weight ratio, which is considerably reduced in the soda group. Most problems that deteriorate the reproductive system, especially testicular function, are linked to oxidative stress caused by free radicals. Defects in spermatogenesis can result from life-threatening free radical attacks that obstruct blood vessels and seriously attack cells in the reproductive system. Various sperm characteristics, such as motility, count, and morphology, are extremely sensitive to free radicals, which might affect sperm quality [27]. Free radical-induced oxidative stress has a crucial role in sperm deformity, a decrease in sperm count, and the fragmentation of sperm DNA. These sperm DNA alterations cause infertility. Under high oxygen pressure, spermatozoa may produce more hydrogen peroxide (H_2O_2), which may reduce sperm motility [28]. Increased production of reactive oxygen species (ROS) reduces sperm-oocyte combination and increases spermatozoa's ability to adhere to the translucent area (zona Placida) [29]. The current study only measured LPO as a part of oxidative stress and found that soda beverages induce LPO in testicular tissue. For the maintenance of testicular cells, a balance between the production of antioxidants and the formation of free radicals must be established. Doing so is necessary because the testicular tissue must treat the harmful effects of free radicals; otherwise, the cells and tissue may suffer severe damage [30]. Antioxidants can prevent this damage by neutralizing free radicals or blocking their production in testicular cells. Notably, a part of the body's antioxidant defense system, known as the preventive antioxidant system, is related to antioxidant enzymes such as catalase, GSH-Px, and SOD. Catalase usually neutralizes the effect of H_2O_2. Incubating spermatozoa under high oxygen pressure lowers sperm rate and motility; however, this effect is reversed when catalase is added to the culture medium [31]. The current study found that mice that consume soda beverages have low catalase levels in their testicular tissue. SOD with a comparably higher amount than other antioxidant enzymes facilitate the conversion of the superoxide (NO_2) anion into O_2 and H_2O_2. Moreover, this enzyme safeguards spermatozoa from O_2 toxicity and lipid membranes from peroxidation. In the current study, soda consumption lowers testicular SOD levels. GSH serves as a cofactor for GSH-Px, protecting mammalian cells from oxidative stress by diminishing H_2O_2 and other peroxides. A study demonstrated that 5 mM of glutathione has protective benefits on sperm during freezing and is correlated with improved sperm motility post-freezing [32]. The present study found that soda consumption decreases testicular GSH and GSH-Px. Adropin is a complex peptide identified as a novel metabolic hormone that regulates lipid and glucose balance. Adropin reduces body obesity and exhibits anti-inflammatory and antioxidant properties. A previous study showed that adropin therapy markedly elevated sperm count and testicular testosterone levels. Furthermore, adropin therapy decreased oxidative and nitrosative stress. Moreover, increased mice testes treated with adropin ultimately increased SOD and catalase activity [32]. The immunohistochemistry analysis demonstrated significant positivity of adropin in the Leydig cells. Adropin, either independently or in conjunction with insulin, enhanced germ cell viability and proliferation by upregulating the expression of proliferating cell nuclear antigen (PCNA), B-cell lymphoma 2 (Bcl2), and protein of extracellular signal-regulated kinases (pERK1/2) [33]. The current study found that soda consumption decreases adropin levels in testicular tissue. Endothelin-1 is a potent vasoconstrictor peptide present at elevated levels in human seminal plasma. However, the physiological effect of endothelin-1 on spermatogenesis is still not understood. A previous study concluded that endothelin-1 increases sperm motility [34]. On the other side, a previous study found that the immunohistochemical assessment of spermatic vein specimens from 55 patients with varicocele revealed an overexpression of endothelin-1 and its receptors in varicose veins [35]. Furthermore, another study found that endothelin-1 expression rates on Sertoli cell was significantly higher in all infertile group compared to that of the control group [36]. Steroidogenesis, gametogenesis, and germ-cell death depend on NO, a reactive nitrogen species produced by NOS. Gonadal dysfunction, germ cell death, and oxidative damage result from excessive NO production [37]. Moreover, studies have shown that an increase in sperm motility can be achieved with low NO concentrations, whereas an increase in NO concentrations can restrict it. In addition, increased NOS activity may cause idiopathic asthenospermia [38]. In the current study, soda consumption increases NO levels in mice's testicular tissue. Furthermore, NOS immunohistochemical reactivity increased in the testicular tissue of mice that consumed soda beverages. Soda consumption seems to affect testosterone levels outside of the hypothalamic-pituitary-testicular axis because testosterone levels are reduced but LH levels remain unchanged. Nevertheless, this study does not prove a direct interaction with Leydig cells. Soda beverage might affect other cell populations in the testis and act indirectly on Leydig cells. Semen analysis indicated reduced sperm counts and motility in mice that consumed soda beverages. Furthermore, the incidence of defective sperm increased with soda intake. Soda drinking may collectively impact male fertility by creating oxidative stress that affects testosterone-producing Leydig cells and sperm quality. A small sample size is one of the study limitations. Furthermore, the present study didn't give a picture of the

mechanism of action of the soda beverage constituent on sperm testicular cells.

5. Conclusions

The study concludes that the consumption of soda beverages reduces the fertility of male mice via the induction of oxidative stress. This stress diminishes sperm counts and motility while augmenting the prevalence of defective sperm. Furthermore, oxidative stress diminishes testosterone levels by interacting with Leydig cells directly in the testes. Moreover, soda consumption diminishes adropin, which plays a role in stimulating sperm count and testosterone production, while simultaneously elevating nitric oxide, a reactive nitrogen species that affects sperm quality.

AVAILABILITY OF DATA AND MATERIALS

The data are contained within this article.

AUTHOR CONTRIBUTIONS

ASA—designed the research study, wrote the manuscript, performed the experiments, and analysed the data. The author read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The animal study protocol was accredited by the National Committee for Bioethics at Taif University (protocol code HPO-02-T-105) and the Committee considered that the proposal fulfills the requirements.

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CONFLICT OF INTEREST

The author declares no conflict of interest.

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