Original

Comparative Effects of L-Carnitine and T-Bhq on Reproductive Hormone Dysregulation in Alcohol-Exposed Wistar Rats

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ABSTRACT

Background: Reproductive dysfunction has been linked to chronic alcoholism, especially due to oxidative stress. Antioxidants like L-carnitine and tert-butylhydroquinone (tBHQ) could mitigate alcohol-induced reproductive toxicity, but their comparative effects are understudied. This study compared the effects of l-carnitine and t-BHQ on reproductive hormone dysregulation in alcohol-exposed Wistar rats.
Methodology: Thirty-five male Wistar rats were divided into seven groups (n=5): control, alcohol-only, alcohol+L-carnitine, alcohol+tBHQ, alcohol+L-carnitine+tBHQ, L-carnitine-only, and tBHQ-only. Alcohol was administered orally (2 g/kg/day), L-carnitine (100 mg/kg/day), and tBHQ (50 mg/kg/day) for 60 days. At the end of the experimental period, serum testosterone, dihydrotestosterone (DHT), 5-α reductase, luteinizing hormone (LH), and follicle stimulating hormone (FSH) were assayed.
Results: Alcohol exposure significantly reduced testosterone (from 10.33 ng/ mL in the control group to 1.12 ng/mL) while significantly increasing the levels of DHT (59.67 pg/Ml to 1237.67 pg/mL), 5-α reductase (91.83 pg/mL to 698.33 pg/mL), LH (26.83 mIU/mL to 64.50 mIU/mL) and FSH (2.98 mIU/mL to 18.33 mIU/mL). L-carnitine and tBHQ independently restored testosterone to above-control levels (16.67 and 17.33 ng/mL, respectively), with their co-administration yielded much more increase. Both agents significantly reduced alcohol-induced increase in DHT and 5-α reductase, with tBHQ exerting a stronger suppressive effect. Similarly, LH and FSH concentrations were normalized toward control values following treatment.
Conclusion: Results shows that L-carnitine and tBHQ mitigated hormone dysregulation in male Wistar rats caused by excessive alcohol exposure. These findings suggest that L-carnitine preserves mitochondrial integrity to sustain steroidogenesis, while tBHQ more effectively modulates enzyme activity and oxidative stress in the testes.

Keywords: Alcohol, L-carnitine and t-BHQ, testosterone and dihydrotestosterone, 5-α reductase, luteinizing hormone, follicle-stimulating hormone.

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INTRODUCTION

Alcohol consumption is a global public health concern [1-3] and its detrimental effects on the human body are not only limited to the central nervous system, but also spread to other organ system, including the reproductive axis. There is a substantial body of literature that chronic alcohol consumption leads to impairment of male reproductive functions [4,5], which are mainly mediated through disruption of the hypothalamic-pituitary-gonadal-axis, testicular oxidative stress, and reduction in the activities of steroidogenic enzymes [6,7]. This leads to an endocrine imbalance that is characterized by lowered testosterone levels, irregular secretion of gonadotropin and impaired spermatogenesis, all of which cause male infertility [8].

The metabolic process of ethanol in the testes produces too much reactive oxygen species (ROS) [9, 10]. The accumulation of these ROS affects the mitochondrial functioning, destroy the Leydig and Sertoli cells, and change the expression of several important enzymes including 5-α reductase [11-13]. As a result, there is a decrease in the production of testosterone and an increase in its metabolism to dihydrotestosterone (DHT) resulting in an imbalance that disturbs the androgen-dependent physiological functions [12,14]. Also, low testosterone suppresses the negative feedback on the hypothalamus and the pituitary, thus increasing the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) [15,16]. All these hormonal derangements reflect the pathophysiological processes of alcohol-induced infertility.

Considering all these detrimental impacts, there is a growing interest in the use of antioxidants and cytoprotective compounds capable of mitigating oxidative stress and restoring reproductive functions. L-carnitine is naturally occurring quaternary ammonium derivative that is involved in the transport of fatty acids to mitochondria where they can undergo a beta oxidation [17, 18]. Its antioxidant qualities and its ability to preserve mitochondrial integrity make it a therapeutic option to protect Leydig cell functioning and maintain steroidogenesis during oxidative stress.

Likewise, tert-butylhydroquinone (tBHQ), which is a synthetic phenolic antioxidant commonly used as a food preservative, has been found to induce the nuclear factor erythroid 2-related factor 2 (Nrf2) signaling pathway, which reduces oxidative stress [19, 20]. In addition to its general cytoprotective ability, tBHQ has also shown activity in preserving testicular architecture, suppressing lipid peroxidation, and restoring hormonal levels during toxic attacks [21, 22]. Its ability to control steroidogenic enzymes implies that it is directly involved in controlling homeostasis of reproductive hormones.

Although there are evidences on the individual protective effects of L-carnitine and tBHQ, there is little or no literatures comparing their efficacy or studying the possible synergism in the context of alcohol-induced reproductive toxicity [18, 20, 22]. This paper thus aims to determine and compare the influences of L-carnitine and tBHQ on the reproductive hormone imbalance in alcohol-treated male Wistar rats. This study particularly examined their effects on the concentration of serum testosterone, dihydrotestosterone (DHT), 5-α reductase, luteinizing hormone (LH), and follicle-stimulating hormone (FSH).

The outcomes of this study will contribute to a better understanding on the use of antioxidants in managing and mitigating alcohol-related male reproductive dysfunctions. This study will also provide experimental evidence for future translational studies.

RESULTS

Background characteristics of the experimental groups

The thirty-five Wistar rats that were used in this study were all similar in their baseline features prior to the commencement of alcohol, L-carnitine, or tBHQ. No significant differences ($p > 0.05$) were observed between all groups in respect of:

i. Initial body weight
ii. General physical activity and feeding behavior.
iii. Hormone concentrations (testosterone, DHT, LH, FSH, and 5-a reductase) of the baselines.
iv. Health condition and no clinical disease appearance.

Random allocation was done to maintain similar physiological conditions in each group as they commenced the experiment. Therefore, the changes observed at the end of the experiment can be attributed to the effects of the alcohol exposure or the antioxidants (L-Carnitine and tBHQ), administered, rather than to pre-treatment differences among the animals.

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Testosterone and Dihydrotestosterone Concentration

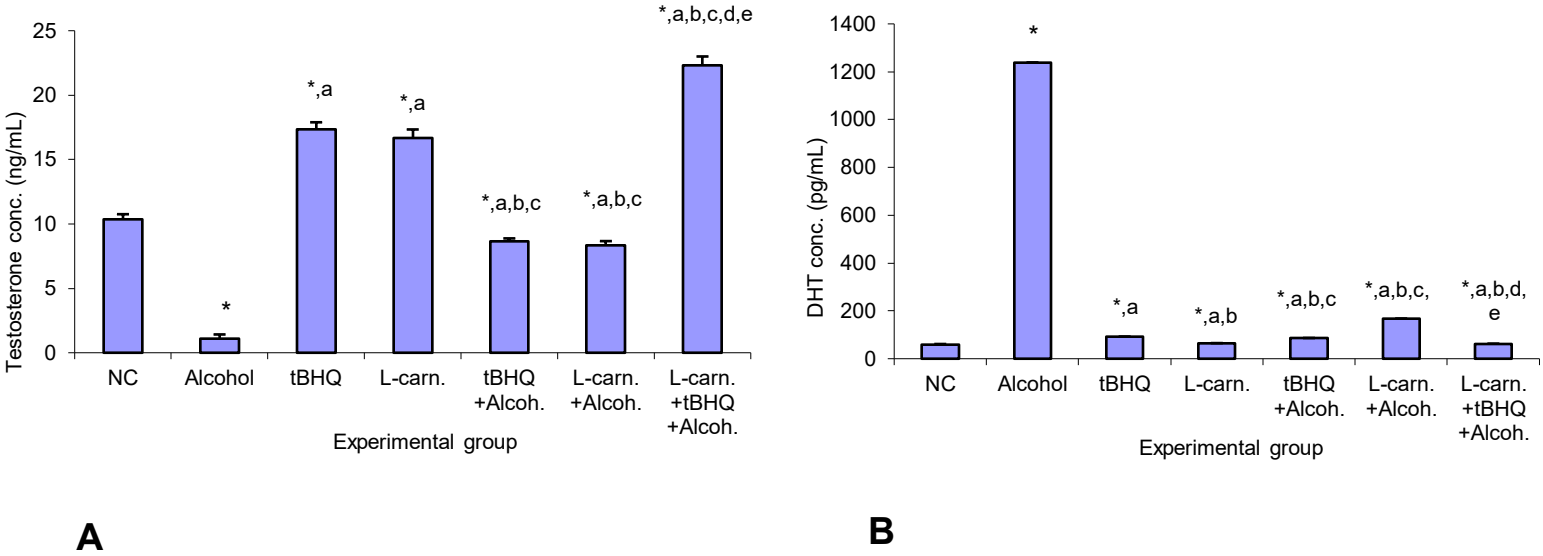

Figure 1: Testosterone and Dihydrotestosterone concentrations in the different experimental groups. **A.** Testosterone concentration**. B.** Dihydrotestosterone concentration**.**

Values are expressed as mean +SEM, n = 5.
* = p<0.05 vs control
a = p<0.05 vs alcohol
b = p<0.05 vs tBHQ
c = p<0.05 vs L-carnitine
d = p<0.05 vs tBHQ + Alcohol
e = p<0.05 vs L-carnitine + Alcohol

5-α Reductase Concentration

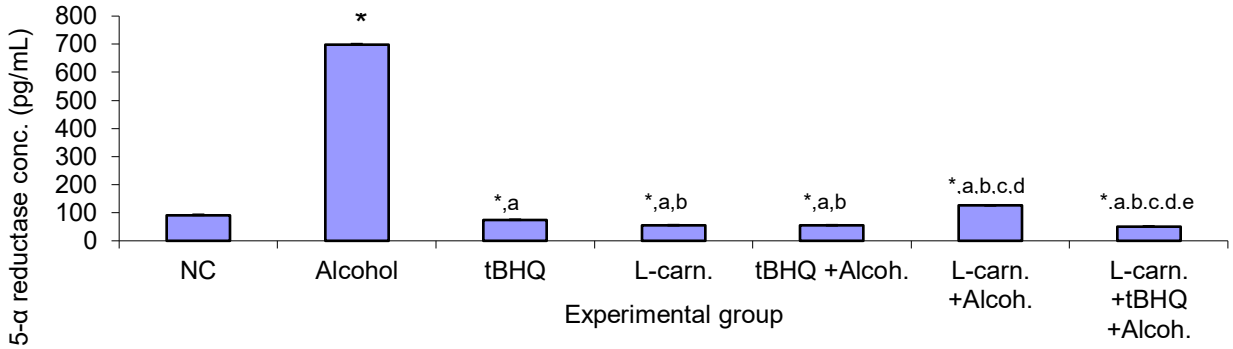

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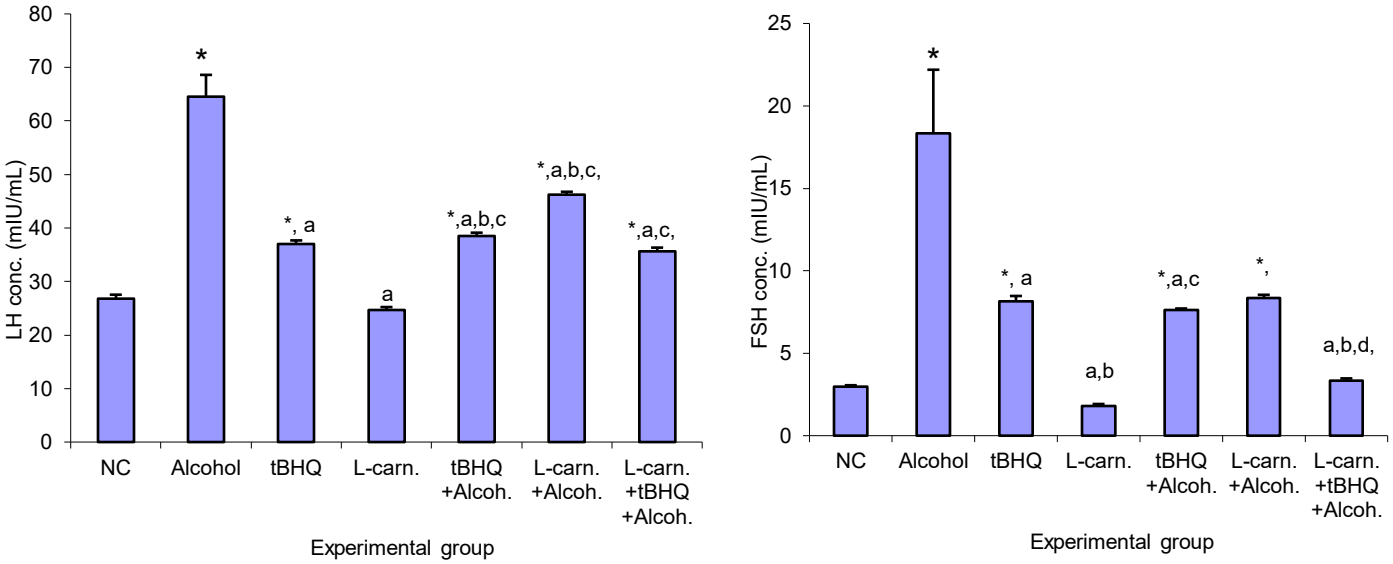
FIG. 2: 5-α reductase concentration in the different experimental groups.
Values are expressed as mean +SEM, n = 5.
* = p<0.05 vs control
a = p<0.05 vs alcohol
b = p<0.05 vs tBHQ
c = p<0.05 vs L-carnitine
d = p<0.05 vs tBHQ + Alcohol
e = p<0.05 vs L-carnitine + Alcohol

Luteinizing Hormone and Follicle Stimulating Hormone



A **B**

Figure 3: Luteinizing hormone and follicle stimulating hormone concentration in the different experimental groups. A. Luteinizing hormone. B. Follicle stimulating hormone.
Values are expressed as mean +SEM, n = 5.
* = p<0.05 vs control
a = p<0.05 vs alcohol
b = p<0.05 vs tBHQ
c = p<0.05 vs L-carnitine
d = p<0.05 vs tBHQ + Alcohol
e = p<0.05 vs L-carnitine + Alcohol

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DISCUSSION

In this experiment, the comparative impact of L-carnitine and tert-butylhydroquinone (tBHQ) on alcohol causing reproductive hormone imbalance in male Wistar rats was studied.

Chronic alcohol intake in this research caused a severe decrease in serum testosterone from 10.33 ng/ mL in the control group to 1.12 ng/mL in the alcohol group, as seen in FIG 1A. The result is very consistent with other studies that have reported that ethanol exposure disrupts steroidogenesis, primarily by affecting mitochondrial dysfunction and enzymatic activities disrupting steroidogenic pathways, in addition to oxidative stress [28, 29]. When administered individually, both L-carnitine and tBHQ supplementation increased testosterone to levels above control (16.67 and 17.33 ng/mL, respectively), and their combined administration restored testosterone to supraphysiological levels, 22.33 ng/mL. This aligns with the literatures by Koohpeyma *et al.* [24], Mateus *et al.* [30] and Abdel-Emam & Ahmed [31] that demonstrates L-carnitine boosts Leydig cell mitochondrial activity, thus maintaining the ATP production that supports steroidogenesis, in this case, the production of testosterone, under oxidative or toxic challenges. Similarly, tBHQ has also been shown to conserve testicular architecture and thus sustain production of testosterone [21,22], and this is consistent with the finding that tBHQ alone elevated or maintained testosterone in damaged testes.

FIG 1B shows the concentration of dihydrotestosterone (DHT) across the experimental groups. Compared to the control group (59.67 pg/mL), there was a very significant increase in the concentration of DHT in alcohol only rats (1237.67 pg/mL). The rise could be attributed to the severe spike, nearly a seven-fold spike in of 5-α reductase activity (as shown in FIG 2) in the alcohol exposed rats (698.33 pg/mL) in comparison to the control group (91.83 pg/mL); which increases the rate of testosterone to DHT conversion [32]. Comparable outcomes were recorded in the study by Rubin *et al* [33] and Van Thiel [34], where prolonged alcohol consumption resulted to an increased metabolism of testosterone to dihydrotestosterone through the upregulation of 5α-reductase, thus increasing the level of dihydrotestosterone in the serum.

However, single and co-administrations of tBHQ and L-carnitine with alcohol significantly ($P<0.05$) reduced the DHT concentration when compared with the alcohol-only group as observed in FIG 1B. DHT was significantly reduced in the alcohol+tBHQ than in the alcohol+ L-carnitine groups. Also, administrations of tBHQ and L-carnitine singly to alcohol fed rats and in combination significantly ($P<0.05$) reduced the 5-α reductase concentration when compared to the groups administered only with alcohol groups ($P<0.05$). Similar to the results on DHT concentration, tBHQ demonstrated stronger suppression on 5-α reductase compared with L-carnitine. This is consistent with studies that has shown that tBHQ in particular preserve steroidogenic enzyme activity and reduce upregulated inflammatory/apoptotic signaling in the testis [22,25].

FIG 3A shows the concentration of luteinizing hormone (LH) in the experimental groups. Excessive alcohol intake increased the level of LH significantly from 26.83 mIU/mL, in the control group, to 64.50 mIU/mL. This aligns with the idea of primary testicular failure where low levels of testosterone remove the negative feedback inhibition on the hypothalamic-pituitary axis, leading to an increased level of LH [35]. Clinical research such as studies by Thiel *et al.* [36] and Castilla-García *et al.* [37] have also indicated increased LH in chronic alcoholics. Conversely, other studies (38,39) involving chronic exposure to ethanol, such as acute ethanol exposure, documented LH suppression which is probably due to temporary hypothalamic suppression and not long-term failure of the testicles. Therefore, our findings support the chronic alcohol approach to dysregulation of feedback.

Administrations of tBHQ and L-carnitine singly and in combination with alcohol significantly ($P<0.05$) reduced the LH concentration when compared to the alcohol group (FIG 3A); which is consistent with restored peripheral steroidogenesis [21,24], thus, re-establishing negative feedback on the hypothalamic-pituitary axis. It is also noteworthy that in our dataset, LH was higher in alcohol+L-carnitine (46.17 mIU/mL) than in alcohol+tBHQ animals (38.50 mIU/mL); which also mirriors the higher decrease in the concentration of DHT and 5-a reductase in with tBHA. So, compared to L-carnitine, tBHQ is more effective in ensuring the complete normalization of testosterone production which will, in turn, lead to a stronger negative feedback effect will result in the greater suppression of LH.

Figure 3B shows the FSH concentration in the different experimental groups. There was a significant ($P<0.05$) increase in the FSH concentration in the alcohol group 18.33 mIU/mL compared to the control group 2.98 mIU/mL. This is in line with previous literatures that have reported elevated FSH when Sertoli cell function and spermatogenesis are compromised due to heavy alcohol exposure [23,40]. Administrations of tBHQ or L-

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carnitine singly and in combination significantly ($P<0.05$) reduced the FSH concentration when compared to the alcohol groups. It was significantly lower in the alcohol+tBHQ+L-carnitine group than in the alcohol+tBHQ and the alcohol+L-carnitine groups ($P<0.05$), which means that the combined therapy provided maximal recovery.

Strength and Limitations of the Study

Strengths of the Study

Comparative and Combination Design: This research analyzed the independent and interactive effects of L-carnitine and tBHQ at the same time, which made it possible to better realize the comparative effectiveness of the two factors and the possible synergism.

Multiple Hormonal Endpoints: The experiment assessed a wide range of reproductive hormones (testosterone, DHT, LH, FSH, and 5-a reductase), which is a comprehensive measure of the functioning of the hypothalamic-pituitary-gonadal (HPG) axis.

Controlled Experimental Conditions: The animals were grouped randomly and kept in a similar environmental condition and given constant doses, which minimized bias.

Translational Relevance: The investigated mechanisms, such as oxidative stress and steroidogenesis, are closely relevant to infertility caused by alcohol in humans as well as to antioxidant therapy.

Limitations of the Study

Small Sample Size per Group (n=5): Although a small sample size is acceptable in animal studies, a bigger sample size would be more statistically powerful and generalizable.

Hormonal Analysis Only at Endpoint: Hormonal measurements were taken only after 60 days. Repeated measurements would have given information on the time and course of hormonal variation.

Single Species and Sex: This study was limited to male Wistar rats; findings might not be completely applicable to human beings and female reproduction.

Implications of the Findings of the Study

Potential Therapeutic Strategy for Alcohol-Induced Infertility: L-carnitine and tBHQ are capable of restoring testosterone, normalizing gonadotropins, and suppressing 5-a reductase, which may be useful in the treatment of alcohol-related hormonal disorders.

Public Health Education: The findings of this study highlight the need to have more intensive alcohol-use education with emphasis on reproductive health outcomes.

Recommendation in Support of the Antioxidant-Based Preventive Strategies: Public health initiatives may incorporate dietary or supplemental antioxidant strategies to mitigate alcohol-induced testicular toxicity.

CONCLUSION

In conclusion, the findings of this study indicated that chronic alcohol exposure significantly disrupted testicular steroidogenesis and pituitary-gonadal regulation, which was characterized by reduction in testosterone levels, alongside severe increase in dihydrotestosterone (DHT), 5-a reductase activity, and gonadotropins (LH and FSH). Treatment with L-carnitine and tBHQ, either singly or in combination, ameliorated these alterations, with the combined administration producing the most pronounced protective effects.

Conflict of interests: The authors declare that there is no conflicting interest in this study.

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