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Original Research Paper

The Effect of Finasteride on Serum Cortisol and testosterone Levels in Rats

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Abstract: Finasteride is a medication used in the treatment of androgenic alopecia in both males and females, as well as in the management of benign prostatic hyperplasia (BPH) in males. The study aimed to investigate the effects of finasteride on serum cortisol and testosterone levels. Female Wistar rats were used as the experimental model. Forty-five female rats were divided into control and treatment groups. Hormone levels were measured using ELISA kits for cortisol and testosterone. Finasteride significantly reduced cortisol levels in treated rats compared to control. At the same time, testosterone levels increased in a dose-dependent manner. Finasteride alters adrenal and gonadal hormones by lowering cortisol and elevating testosterone. These findings highlight its broader impact on endocrine regulation.

Keywords: finasteride, cortisol, testosterone

1.Introduction

Finasteride is a synthetic inhibitor of 5α-reductase, commonly employed in the management of benign prostatic hyperplasia (BPH) and androgenetic alopecia [1,2]. Finasteride modifies the hormonal equilibrium in the body by inhibiting the conversion of testosterone into its more powerful metabolite, dihydrotestosterone (DHT). This process impacts not just androgen-related pathways but may possibly alter other endocrine functions [3-6].

Testosterone The steroid hormone testosterone is present in both humans and other animals. The testes are the principal source of testosterone in males. Women's ovaries generate testosterone, but at far lower quantities. In females, testosterone is produced by the ovaries and adrenal glands. Typically, blood testosterone levels in females are 10–20 times lower than in males. Women are more susceptible to variations in testosterone levels, even though testosterone is often recognized as a male sex hormone due to the disparity in concentration between the genders. Cortisol, the principal glucocorticoid hormone synthesized by the adrenal glands, is generated in the zona fasciculata layer of the adrenal cortex. The anterior pituitary secretes adrenocorticotropic hormone (ACTH), which is pivotal in stress response, metabolism, and immunological control. Enhances the action of low-density lipoprotein (LDL) receptors and stimulates cholesterol desmolase, the rate-limiting enzyme in cortisol production that transforms cholesterol to pregnenolone. The majority of glucocorticoids are inactive and associated with either albumin or corticosteroid-binding globulin (CBG) [10,11]. Given both testosterone and cortisol are steroid hormones derived from cholesterol and possess analogous biosynthetic routes, alterations in one system may possibly influence the other [12-14].

Animal studies, particularly in rats, provide valuable insights into the biochemical and physiological effects of finasteride. Investigating how finasteride administration

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modifies serum testosterone and cortisol levels in rats can help clarify its broader endocrine effects. Such findings are important not only for understanding the drug's safety profile but also for anticipating possible metabolic or stress-related consequences in clinical use [15-19].

2.Methodology

In this experiment, forty-five female Wistar rats aged 90 days and weighing 160–180 g were used. The animals were divided into three groups: control, low-dose finasteride (1 mg), and high-dose finasteride (5 mg). Blood samples were collected at different intervals. After collection, the blood was allowed to coagulate for one hour at room temperature or overnight at 2–8℃. Then, the samples were centrifuged at 1000×g for 20 minutes at 2–8℃, and the serum supernatant was separated for hormonal assays [20-22].

Principle of Assay for Cortisol

Cortisol levels were determined using a Rat Cortisol ELISA Kit (A303698), which is based on a competitive enzyme immunoassay. The microtiter plate was pre-coated with cortisol antigen. In this method, cortisol in the serum samples competes with immobilized cortisol for binding to biotinylated anti-cortisol antibodies. After incubation, unbound antibodies were washed away, and an HRP-streptavidin conjugate was added. Following another incubation, TMB substrate solution was introduced, producing a blue color that turned yellow after adding the stop solution. The optical density was measured at 450 nm using a microplate reader, and the cortisol concentration was calculated from a standard curve [23,24].

Test principle for Testosterone

Testosterone levels were measured using a Rat Testosterone ELISA Kit, which also employs a competitive ELISA principle. The ELISA plate was pre-coated with testosterone antigen, and testosterone in the samples competed with the immobilized antigen for binding to an HRP-conjugated detection antibody [25-28]. After washing away the excess, a substrate solution was added, leading to a yellow color formation. The absorbance was read at 450 nm, and the testosterone concentration was determined by comparing sample values to a standard calibration curve [29-32].

3. Results and discussion

cortisol

Table (1) presents cortisol levels (mean ± standard error) measured across three experimental groups (Control, (1mg) and (5mg) finasteride) over two consecutive months. Statistical significance was determined using Least Significant Difference (LSD) testing at $p < 0.05$. The control group maintained the highest cortisol levels during both measurement periods (95.63±1.44 and 97.34±1.29, respectively). These values were significantly higher than both treatment groups ($p < 0.05$). The (1mg) exhibited intermediate cortisol levels (86.55±2.01 and 78.84±1.99), which were significantly lower than the control group but significantly higher than the (5mg) in both measurement periods. The (5mg) treatment group demonstrated the lowest cortisol levels (77.69±3.98 and 68.02±4.07), significantly lower than both the control and (1mg) across both time periods. The LSD value for comparing between groups was 5.012, indicating that mean differences greater than or equal to this value were considered statistically significant at $p < 0.05$. The LSD value for comparing between time periods was 3.0124, meaning differences exceeding this threshold between measurements were statistically significant.

Table 1: presents the mean cortisol levels and standard errors across the experimental groups.

Group	First Month	Second Month	LSD periods
Control	95.63±1.44 Aa	97.34±1.29 Aa	3.0124
G1 (1mg)	86.55±2.01 Bb	78.84±1.99 Bc	
G2 (5mg)	77.69±3.98 Cc	68.02±4.07 Cd	
LSD groups	5.012		

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This results in table (1) noticed decrease in cortisol level from the first month, cortisol is inactivated in liver partly via 5AR (types 1 & 2) to 5α-dihydrocortisol. Finasteride selectively inhibits type 2 (SRD5A2). In human hepatocytes, pharmacologic 5AR2 inhibition with finasteride augments cortisol action (because less is inactivated), altering lipid metabolism. This argues against a primary drop in effective cortisol signaling that accepted with [33].

Clinical endocrine studies in people on finasteride (5 mg/day) showed no significant changes in serum cortisol (and no adrenal steroidogenesis impairment beyond expected shifts in 5α-metabolites) [34,35].

In humans, finasteride does not meaningfully lower circulating cortisol. Most clinical studies and the FDA label report no significant change in serum cortisol with finasteride. Mechanistically, 5-α-reductase (5AR) helps inactivate cortisol in liver; inhibiting 5AR can reduce cortisol clearance and thereby augment local glucocorticoid action, especially in liver more consistent with unchanged or effectively higher tissue exposure, not lower cortisol [36,37].

These findings are aligned with previous studies. [38] highlighted the importance of regulating HPA axis activity to prevent chronic stress-related disorders. Furthermore, [39] provided evidence that certain pharmacological agents and finasteride extract can attenuate HPA axis responses, leading to reduced circulating cortisol under both basal and stress-induced conditions [40].

Testosterone

Table (2) presents testosterone levels (mean ± standard error) measured across three experimental groups (Control, (1mg), and (5mg)) over two consecutive months. Statistical significance was determined using Least Significant Difference (LSD) testing at $p < 0.05$. The control group maintained the lowest testosterone levels during both measurement periods (1.05±0.02 and 0.86±0.06, respectively). These values were significantly lower than both treatment groups ($p < 0.05$). The (1mg) exhibited intermediate testosterone levels (2.01±0.10 and 3.13±0.09), which were significantly higher than the control group but significantly lower than the (5mg) in both measurement periods. The (5mg) demonstrated the highest testosterone levels (3.23±0.12 and 5.97±0.14), significantly higher than both the control and 1 mg treatment groups across both time periods. The LSD value for comparing between groups was 1.214, indicating that mean differences greater than or equal to this value were considered statistically significant at $p < 0.05$. The LSD value for comparing time periods appears to be missing from the table.

Table 2: presents the mean Testosterone levels and standard errors across the experimental groups

Group	First Month	Second Month.	LSD periods
Control	1.05±0.02 Cc	0.86±0.06 Cc	1.453
G1 (1mg)	2.01±0.10 Bb	3.13±0.09 Ba	
G2 (5mg)	3.23±0.12 Ab	5.97±0.14 Aa	
LSD groups	1.214		

This table (2) results of control levels of Testosterone in hair agree with [41] that show Control levels of testosterone in hair provide an important baseline for evaluating hormonal changes. Measuring testosterone in hair reflects long-term exposure rather than short-term fluctuations seen in blood or saliva. This stable indicator helps in distinguishing normal physiological levels from abnormal elevations or deficiencies. Therefore, using hair samples offers a reliable and non-invasive method for monitoring androgen status in research and clinical practice.

The data presented in this table (2) show that control groups appear to increase in the Testosterone levels from the first month. This may indicate that reverses the effect of natural factors such as natural hormone cycles or environmental factors without therapeutic intervention affecting hormone levels, that accept with [42] performed a meta-analysis of 11 clinical studies involving 1,784 people treated with finasteride. The pooled results did not demonstrate a consistent significant increase in serum testosterone levels. Variations across studies suggest that observed increases may result from altered sex hormone–binding globulin (SHBG) dynamics rather than increased androgen production [43]

In the PLESS randomized controlled trial, people with BPH treated with finasteride showed a modest but statistically significant increase in serum testosterone compared to placebo—especially in those with low baseline testosterone. These individuals also showed slight reductions in body mass index (BMI) [44]

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Numerous clinical and experimental studies support this hormonal pattern. For instance, [45] reported elevated serum testosterone levels in healthy people following both short- and long-term use of finasteride. However, despite the rise in total testosterone, overall androgenic activity may not increase proportionally due to the lower binding affinity and potency of testosterone at the androgen receptor compared to DHT [46].

A clinical study tracking hormonal changes over three months of finasteride use found testosterone levels rose by approximately 15% ($p < 0.05$) [47]

In the current study, finasteride administration also resulted in parallel increases in progesterone and estrogen levels, particularly at the 5 mg dose. These findings suggest that the treatment not only alters androgen metabolism but may also stimulate the gonadotropic axis, enhancing overall steroidogenesis. This hormonal shift is consistent with previous research on anabolic and hormonal therapies that modify multiple steroid pathways simultaneously [48,49].

In a 28-day randomized study (5 mg/day), finasteride significantly reduced DHT levels in scalp tissue and serum, but did not alter serum testosterone [50]

Overall, while finasteride may provoke mild increases in serum testosterone most prominently in individuals with low baseline T or during high dose/prolonged treatment, the findings are inconsistent and often physiologically minimal. The meta-analytic evidence emphasizes that increased testosterone is not a robust or expected outcome. Understanding these nuances is critical when counseling patients about hormonal effects of finasteride.

Conclusion

The present study demonstrated that finasteride administration significantly altered serum cortisol and testosterone levels in rats in a dose-dependent manner. Finasteride treatment resulted in a marked reduction in cortisol concentrations, suggesting that inhibition of 5α-reductase may affect hypothalamic-pituitary-adrenal (HPA) axis regulation and cortisol metabolism. At the same time, testosterone levels increased significantly in treated groups, with the highest elevation observed at the 5 mg dose. This indicates that suppression of dihydrotestosterone (DHT) formation by finasteride leads to a compensatory rise in circulating testosterone.

These findings highlight that finasteride exerts broader endocrine effects beyond androgen suppression, influencing adrenal and gonadal hormone balance. The observed hormonal shifts may have implications for stress regulation, metabolism, and reproductive physiology. Further studies are warranted to clarify the mechanisms underlying these changes and to evaluate their potential clinical relevance, particularly in long-term use.

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Author's Contributions

luma Qasim Ali suggested the research idea, performed the practical section, and Doaa Abd Al-hassan Abboud organized all the results, data, wrote and revised the manuscript. All authors agreed to the final version of this manuscript

Ethics

The study was approved by ethics committee of the department of biology, college of science, mustansiriyah university , baghdad , iraq , as did the iraqi ministry of health and environment . committee number dated BCSMU/4024/0054Z written informed consent was obtained from all patients

References

1. Nayana, J., Shankaranarayana Rao, B. S., & Srikumar, B. N. (2025). The interaction between finasteride and corticosterone levels: Implications for depression-, and anxiety-like behavior and hippocampal synaptic plasticity in male rats. *Psychopharmacology*, 1–21.

2. Sasibhushana, R. B., Rao, B. S., & Srikumar, B. N. (2024). Anxiety-, and depression-like behavior following short-term finasteride administration is associated with impaired synaptic plasticity and cognitive behavior in male rats. *Journal of Psychiatric Research, 174*, 304–318.

3. Bosse, G. D., Cadeddu, R., Floris, G., Farero, R. D., Vigato, E., Lee, S. J., ... & Peterson, R. T. (2021). The 5α-reductase inhibitor finasteride reduces opioid self-administration in animal models of opioid use disorder. *The Journal of Clinical Investigation*, *131*(10).

4. Howell, S., Song, W., Pastuszak, A., & Khera, M. (2021). Differential gene expression in post-finasteride syndrome patients. *The Journal of Sexual Medicine*, *18*(9), 1479–1490.

5. Ji, S. Y., Kim, M. Y., Hwangbo, H., Lee, H., Hong, S. H., Kim, T. H., ... & Choi, Y. H. (2021). Extract of Fructus Corni ameliorates testosterone-induced benign prostatic hypertrophy in Sprague Dawley rats. *Journal of Life Science*, *31*(6), 550–558.

6. Misztal, T., Młotkowska, P., Marciniak, E., Roszkowicz-Ostrowska, K., & Misztal, A. (2021). Involvement of neurosteroids in the control of hypothalamic-pituitary-adrenal axis activity in pregnant sheep under basal and stressful conditions. *Theriogenology*, *174*, 114–120.

7. Młotkowska, P., Marciniak, E., Misztal, A., & Misztal, T. (2023). Effect of neurosteroids on basal and stress-induced oxytocin secretion in luteal-phase and pregnant sheep. *Animals*, *13*(10), 1658.

8. Jafari, A., Panahi, N., Hesaraki, S., & Akbari, G. (2024). Protective and antioxidant effects of quercetin loaded black cumin (*Nigella sativa* L) seed oil-based nanoemulsion in testosterone-induced benign prostatic hyperplasia: An experimental study. *Archives of Razi Institute*, *79*(5), 1065.

9. Sukharev, A., Alade, T., Shah, V., & Dahmani, S. (2024). Does Propecia cause more harms than good: Assessing reproductive and non-reproductive effects of finasteride on male health. *Georgetown Medical Review*, *8*(1).

10. Rzeszotek, S., Kolasa, A., Pilutin, A., Misiakiewicz-Has, K., Sielatycka, K., & Wiszniewska, B. (2022). The interplay between finasteride-induced androgen imbalance, endoplasmic reticulum stress, oxidative stress, and liver disorders in paternal and filial generation. *Biomedicines*, *10*(11), 2725.

11. Ghomeshi, A., Yang, B., & Masterson, T. A. (2023). The adverse effects of commonly used medications on male fertility: A comprehensive review. *F&S Reviews*, *4*(3), 176–186.

12. Giatti, S., Di Domizio, A., Diviccaro, S., Falvo, E., Caruso, D., Contini, A., & Melcangi, R. C. (2021). Three-dimensional proteome-wide scale screening for the 5-alpha reductase inhibitor finasteride: Identification of a novel off-target. *Journal of Medicinal Chemistry*, *64*(8), 4553–4566.

13. Shitikova, A. M., Atroshchenko, M. M., Krokhotina, L. V., Engalycheva, M. G., & Dmitrieva, M. N. (2022). Effect of testosterone, dihydrotestosterone,

DOI: 10.36320/ajb/v18.i1.21988

estradiol and cortisol on the quality of fresh and cryopreserved stallion sperm. *[Journal Name Placeholder]*.

14. Farghaly, H. S. M., Bakr, M. H., Sayed, H. G., & Ashry, I. E. S. M. (2023). Effect of antiandrogens on valproic acid-induced neurodevelopmental changes in rats. *Assiut Veterinary Medical Journal*, *69*(178), 52–63.

15. Ntshingila, S., Oputu, O., Arowolo, A. T., & Khumalo, N. P. (2023). Androgenetic alopecia: An update. *JAAD International*, *13*, 150–158.

16. Minich, D. M., Ross, K., Frame, J., Fahoum, M., Warner, W., & Meissner, H. O. (2024). Not all maca is created equal: A review of colors, nutrition, phytochemicals, and clinical uses. *Nutrients*, *16*(4), 530.

17. Raciti, L., Formica, C., Raciti, G., Quartarone, A., & Calabrò, R. S. (2023). Gender and neurosteroids: Implications for brain function, neuroplasticity and rehabilitation. *International Journal of Molecular Sciences*, *24*(5), 4758.

18. Battipaglia, C., Spelta, E., Blanco, A. M., & Genazzani, A. D. (2025). The hormonal contraceptive choice in women with polycystic ovary syndrome and metabolic syndrome. *GREM Gynecological Endocrinology and Metabolism*, *6*(1), 1–10.

19. Zeng, L. H., Rana, S., Hussain, L., Asif, M., Mehmood, M. H., Imran, I., ... & Abed, S. N. (2022). Polycystic ovary syndrome: A disorder of reproductive age, its pathogenesis, and a discussion on the emerging role of herbal remedies. *Frontiers in Pharmacology*, *13*, 874914.

20. Minnetti, M., De Alcubierre, D., Bonaventura, I., Pofi, R., Hasenmajer, V., Tarsitano, M. G., ... & Isidori, A. M. (2022). Effects of licorice on sex hormones and the reproductive system. *Nutrition*, *103*, 111727.

21. Eitah, H. E., Khaled, S. E., AbouZeid, E. M., El-Shiekh, R. A., Almagharbeh, W. T., & Gamil, N. M. (2025). Herbal remedies and traditional treatments for hirsutism and hypertrichosis. *Inflammopharmacology*, 1–25.

22. Devjani, S., Ezemma, O., Kelley, K. J., Stratton, E., & Senna, M. (2023). Androgenetic alopecia: Therapy update. *Drugs*, *83*(8), 701–715.

23. Altendorf, S., Bertolini, M., Le Riche, A., Tosti, A., & Paus, R. (2025). Frontiers in the physiology of male pattern androgenetic alopecia: Beyond the androgen horizon. *Physiological Reviews*.

24. Taha, Z. A., Chabuk, H. A. H., & Hassan, A. J. (2024). Effectiveness of the *Mentha spicata* leaves in reducing testosterone levels and aggressive behaviour in female Wistar albino rats treated with testosterone propionate. *Journal of Applied & Natural Science*, *16*(1).

25. Raafat, M., Kamel, A. A., Shehata, A. H., Ahmed, A. S. F., Bayoumi, A. M., Moussa, R. A., ... & El-Daly, M. (2022). Aescin protects against experimental benign prostatic hyperplasia and preserves prostate histomorphology in rats via suppression of inflammatory cytokines and COX-2. *Pharmaceuticals*, *15*(2), 130.

26. Nestor, M. S., Ablon, G., Gade, A., Han, H., & Fischer, D. L. (2021). Treatment options for androgenetic alopecia: Efficacy, side effects, compliance, financial considerations, and ethics. *Journal of Cosmetic Dermatology*, *20*(12), 3759–3781.

27. Cannarella, R., Curto, R., Condorelli, R. A., Grillo, A., Aversa, A., Calogero, A. E., & La Vignera, S. (2025). The influence of seminal microbiota on human testicular steroidogenesis: A prospective study. *Journal of Assisted Reproduction and Genetics*, *42*(3), 897–907.

28. Warren, A. M., & Grossmann, M. (2022). Haematological actions of androgens. *Best Practice & Research Clinical Endocrinology & Metabolism*, *36*(5), 101653.

29. Bordanaba-Florit, G., Liempd, S. V., Cabrera, D., Royo, F., & Falcon-Perez, J. M. (2022). Simultaneous quantification of steroid hormones using hrLC-MS in endocrine tissues of male rats and human samples. *Metabolites*, *12*(8), 714.

30. Yüksek, A., Yardımcı, C., Talih, G., & Göçmen, A. Y. (2022). Farklı Sugammadeks Dozlarının Plazma Serbest Hormon Düzeylerine Etkisi. *[Journal Name Placeholder]*.

31. Singh, H., Kumar, R., Mazumder, A., Salahuddin, Mazumder, R., & Abdullah, M. M. (2022). Insights into interactions of human cytochrome P450 17A1: A review. *Current Drug Metabolism*, *23*(3), 172–187.

32. Suzuki, R. (2025). Evaluation of the hair growth-promoting effects of combined treatment with hair growth stimulants. *Health*, *17*(4), 425–449.

33. Wang, K., Li, Y., & Chen, Y. (2023). Androgen excess: A hallmark of polycystic ovary syndrome. *Frontiers in Endocrinology*, *14*, 1273542.

34. Vacher, C. M., Tsompanidis, A., Firestein, M. R., & Penn, A. A. (2025). Neuroactive steroid exposure impacts neurodevelopment: Comparison of human and rodent placental contribution. *Journal of Neuroendocrinology*, e13489.

35. Cao, H., Zhang, D., Wang, P., Wang, Y., Shi, C., Wu, H., ... & Wang, S. (2024). Gut microbiome: A novel preventive and therapeutic target for prostatic disease. *Frontiers in Cellular and Infection Microbiology*, *14*, 1431088.

36. Melcangi, R. C., Cioffi, L., Diviccaro, S., & Traish, A. M. (2021). Synthesis and actions of 5α-reduced metabolites of testosterone in the nervous system. *Androgens: Clinical Research and Therapeutics*, *2*(1), 173–188.

37. Wisuitiprot, V., Ingkaninan, K., Chakkavittumrong, P., Wisuitiprot, W., Neungchamnong, N., Chantakul, R., & Waranuch, N. (2022). Effects of *Acanthus ebracteatus* Vahl. extract and verbascoside on human dermal papilla and murine macrophage. *Scientific Reports*, *12*(1), 1491.

38. Demori, I., Losacco, S., Giordano, G., Mucci, V., Blanchini, F., & Burlando, B. (2024). Fibromyalgia pathogenesis explained by a neuroendocrine multistable model. *Plos One*, *19*(7), e0303573.

39. Elnady, R. E., Abdon, M. S., Shaheen, H. R., Eladawy, R. M., Azar, Y. O., & Al Raish, S. M. (2025). The future of alopecia treatment: Plant extracts, nanocarriers, and 3D bioprinting in focus. *Pharmaceutics*, *17*(5), 584.

40. Gutierrez, G., Wamboldt, R., & Baranchuk, A. (2022). The impact of testosterone on the QT interval: A systematic review. *Current Problems in Cardiology*, *47*(9), 100882.

41. Kumbhar, P. S., Chavan, R., Darekar, S., Kolekar, K., Sequeira, A., Vishwas, S., ... & Patravale, V. (2024). Bridging gap in treatment of polycystic ovarian syndrome through drug repurposing: What we achieved and where we are? *Naunyn-Schmiedeberg's Archives of Pharmacology*, 1–28.

42. Jekl, V. (2025). Adrenal disease in small mammals. *Veterinary Clinics: Exotic Animal Practice*, *28*(1), 87–106.

43. Wang, Y., & Xie, Z. (2022). Exploring the role of gut microbiome in male reproduction. *Andrology*, *10*(3), 441–450.

44. Liu, R., Sun, Z., Wang, S., Liu, X., Man, Y., Chen, M., ... & Wang, C. (2024). Wenshenqianlie capsule improves benign prostatic hyperplasia via its anti-inflammatory and antioxidant effects. *Aging (Albany NY)*, *16*(18), 12574.

45. Ziogou, A., Giannakodimos, A., Mitakidi, E., Konstantinos, T., & Giannakodimos, I. (2024). Is there an association between 5α-reductase inhibitors and metabolic syndrome? A narrative review of the literature. *Reviews on Recent Clinical Trials*, *19*(4), 229–235.

46. Banihani, S. A. (2025). Role of vitamin B6 in testosterone synthesis. *The World Journal of Men's Health*, *43*.

47. Stańczak, N. A., Grywalska, E., & Dudzińska, E. (2024). The latest reports and treatment methods on polycystic ovary syndrome. *Annals of Medicine*, *56*(1), 2357737.

48. Zucão, M. I., Grigio, V., Guerra, L. H. A., Antoniassi, J. Q., da Costa Castro, N. F., Taboga, S. R., & Vilamaior, P. S. L. (2024). Aging effects in adrenal cortex of male Mongolian gerbil: A model for endocrine studies. *Steroids*, *203*, 109366.

49. Ma, J. C., Zhang, H. L., Huang, H. P., Ma, Z. L., Chen, S. F., Qiu, Z. K., & Chen, J. S. (2021). Antidepressant-like effects of Z-ligustilide on chronic unpredictable mild stress-induced depression in rats. *Experimental and Therapeutic Medicine*, *22*(1), 677.

50. Fink, J., & Horie, S. (2022). *The multiple health benefits of testosterone*. Cambridge Scholars Publishing.

51. Wright, T., Eshar, D., Rooney, T., & Berle, E. C. P. (2022). Use of a deslorelin implant for management of hyperandrogenism associated with excessive sex hormone production in a female spayed pet rabbit (*Oryctolagus cuniculus*). *Journal of Exotic Pet Medicine*, *40*, 12–15.