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The effect of Balinese arak exposure on Leydig cell count and corpus cavernosum area in Wistar (*Rattus norvegicus*) rats

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ABSTRACT

Backgrounds: Sexual dysfunction affects men of all ages, ethnicities, and cultural backgrounds. Approximately 52% of men aged 40–70 years experience sexual dysfunction of varying severity. One factor contributing to sexual dysfunction in men is the consumption of alcoholic beverages, including arak, a traditional alcoholic drink originating from Bali. This study aims to determine the effect of Balinese arak consumption on Leydig cell count and erectile function, as assessed by corpus cavernosum area, in Wistar rats (*Rattus norvegicus*).

Methods: This experimental study employed a post-test-only control group design. The animals were divided into two groups: a control group receiving a placebo (P1) and a treatment group receiving Balinese arak at a dose of 0.5 mL/day/rat (P2) for 45 days. At the end of the treatment period, the animals were anesthetized using a ketamine–xylazine combination and euthanized by cervical dislocation. The testes and penile tissue were subsequently harvested for histological examination. Data were analyzed statistically using SPSS version 25 for Windows.

Results: The mean Leydig cell count in the control group was 64.9 cells per field of view, whereas in the Balinese arak group it was 25.86 cells per field of view. The mean corpus cavernosum area in the control group was 81.79 µm2, compared with 62.33 µm2 in the treatment group. Statistical analysis demonstrated a significant difference in Leydig cell count between groups ($p < 0.05$; Welch's t-test). In contrast, no significant difference was observed in corpus cavernosum area ($p > 0.05$), although a decreasing trend was noted in the treatment group.

Conclusion: Balinese arak consumption significantly reduced Leydig cell count in Wistar rats (*Rattus norvegicus*). Although no statistically significant difference was observed in the corpus cavernosum area, a decreasing trend was evident.

Keywords: *Balinese Arak, Leydig Cells, Corpus Cavernosum, Wistar Rats, Infertility.*

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INTRODUCTION

Male sexuality is a complex physiological process that plays an important role in maintaining quality of life. Normal sexual function can only be sustained through harmonious coordination among various body systems, including the nervous, cardiovascular, endocrine, and reproductive systems. Male sexual dysfunction (SD) is a multifactorial condition that cannot be categorized as a single disease entity. It encompasses disturbances in several domains of male sexual function, including arousal, erection, penetration, and ejaculation. Impairment at one or more of these stages may be classified as male sexual dysfunction. Erectile dysfunction (ED) is one of the most common yet under-treated forms of male sexual dysfunction.[1,2]

The global prevalence of male sexual dysfunction remains high. The Massachusetts Male Aging Study reported that 52% of men aged 40–70 years experience impotence of varying severity. At the age of 40, approximately 40% of men are affected, increasing to 70% by the age of 70. Furthermore, epidemiological data indicate that approximately 10 million men in the United States have been affected by impotence.[3,4] Despite extensive basic research, the risk factors and pathogenesis of this condition remain incompletely understood.

Sexual dysfunction is influenced by multiple risk factors, including: (1) disease-related factors (diabetes mellitus, cardiovascular disease, chronic kidney disease, and cancer); (2) lifestyle factors (obesity, smoking, and alcohol consumption); (3) environmental exposures (bisphenol A, polychlorinated biphenyls, phytoestrogens, and heavy metals); and (4) medication-related factors (antidepressants, cardiovascular agents, antibiotics, and anticholinergics).[5] From the perspective of erectile dysfunction, organic ED is generally associated

with abnormalities of the hormonal, neurological, or vascular systems. Several factors are known to increase the risk of ED, including harmful environmental exposures (e.g., ionizing radiation, heavy metals, or elevated estrogen levels), the use of certain medications (such as antitumor agents, antihypertensive drugs, and antibiotics), and chronic conditions, including diabetes mellitus, obesity, hypertension, hormonal disorders, stress, and anxiety disorders.[6]

Similar to other alcoholic beverages, Balinese arak undergoes comparable metabolic processes following consumption and therefore has the potential to exert similar biological effects on spermatozoa, particularly when consumed excessively.[7] The impact of alcohol on spermatogenesis is closely associated with the generation of reactive oxygen species (ROS). Ethanol, the primary component of alcoholic beverages, is a known inducer of ROS formation in the body. ROS production begins during ethanol metabolism, primarily via the cytochrome P450 (CYP450) enzyme system. This metabolic process generates ROS, including peroxide derivatives, as well as acetaldehyde as a by-product. The accumulation of ROS may lead to oxidative stress, lipid peroxidation, protein inactivation, increased cytokine production, mitochondrial dysfunction, and DNA damage.[8]

The dose of Balinese arak administered in this study (0.5 mL/day/rat) was determined based on a previous study by Alfiah Hayati et al. (2018),[9] which demonstrated significant effects following the administration of Balinese arak containing 40% v/v alcohol (equivalent to 6.85 M) for 45 days at that dose. In contrast, a lower dose of 0.1 mL/day/rat did not produce significant effects. Therefore, the 0.5 mL/day/rat dose was selected for the present study, with the same treatment duration of 45 days. In rodent models, chronic ethanol exposure typically ranges from 0.5–3 g/kg body weight/day and has been shown to induce endocrine alterations, oxidative stress, and histopathological changes in reproductive tissues without causing severe acute toxicity or high mortality. This dosage range has been widely used to evaluate dose-dependent effects on Leydig cell function, steroidogenesis, and testicular structure. For instance, Rengarajan et al. administered ethanol at doses of 0.5, 1, and 3 g/kg body weight to examine its effects on luteinizing hormone (LH) receptors and glucose utilization in Leydig cells.[10] The 45-day exposure period was selected as it approximates 90% of a complete spermatogenic cycle in adult rats (approximately 50–52 days), thereby allowing chronic exposure to influence all stages of sperm development.[11] This duration is also consistent with other chronic alcohol exposure protocols reported in the literature, which commonly employ 6–8 weeks of treatment to assess reproductive outcomes.[12] Although not identical, this period represents long-term exposure relative to the reproductive biological cycle of rats. Based on those mentioned above, this study aims to determine the effect of Balinese arak consumption on Leydig cell count and erectile function, as assessed by corpus cavernosum area, in Wistar rats (Rattus norvegicus

METHODS

This study employed an experimental post-test-only control group design among 14 rats. The animals were divided into two groups: a control group receiving a placebo (P1) and a treatment group receiving Balinese arak at a dose of 0.5 mL/day/rat (P2). Allocation of animals to the control and treatment groups was performed using simple random sampling. The study and preparation of histological specimens were conducted at the Histology Laboratory, Faculty of Medicine, Udayana University, Denpasar, Bali. The research was carried out between January and October 2025.

Inclusion criteria consisted of healthy adult male Wistar rats (Rattus norvegicus) aged approximately three months, with a body weight of ±200 g. Rats that were ill, physically impaired, or died during the study were excluded and classified as dropouts. The dependent variables were Leydig cell count and corpus cavernosum area, while the independent variable was administration of Balinese arak. Controlled variables included feed type and quantity, body weight, age, sex, species, ambient temperature, lighting conditions, and cage environment.

The Balinese arak used in this study was a commercially standardized product with an alcohol content of approximately 40% v/v (ARAK BALI ABM). The arak was stored in a sealed glass bottle at room temperature (27 °C), in a dry environment protected from direct sunlight. Rats were housed according to their assigned groups (P1 and P2), with seven rats per cage, and were provided with standard feed and drinking water ad libitum. The animals underwent a two-week acclimatization period during which they received distilled water via oral gavage and had free access to water. Following acclimatization, the treatment group (P2) received Balinese arak (40% v/v) at a dose of 0.5 mL/day/rat for 45 days. Standard drinking water was provided ad libitum outside the administration schedule. The treatment was administered each morning after feeding.

At the end of the treatment period, the animals were anaesthetized using a ketamine–xylazine combination and humanely euthanized by cervical dislocation in accordance with the institutional animal ethics protocol. The testes were harvested and fixed in Bouin's solution for 24 hours. The tissue was then longitudinally sectioned into three parts and refixed for an additional 24 hours. Dehydration was performed through graded alcohol immersion: 70% ethanol for 30 minutes, followed by two immersions in 80% ethanol (30 minutes each) and two immersions in 95% ethanol (30 minutes each). The tissue was then immersed in a xylol–alcohol solution for 60 minutes, followed by two immersions in pure xylol (60 minutes each). Subsequently, the tissue was embedded in liquid paraffin at 58 °C for two hours, re-immersed in fresh paraffin, and left overnight in an oven. The paraffin-infiltrated tissue was molded into paraffin blocks and stored under refrigeration until sectioning. Sections of 5 μm thickness were obtained using a microtome and stained with hematoxylin and eosin (H&E) for histological examination.

Microscopic evaluation was performed on 5–10 sections per animal. For Leydig cell analysis, three fields of view were

examined per section. The corpus cavernosum area was measured using ImageJ software, selecting the largest measurable area. Fields of view were selected using simple random sampling, and only clearly identifiable and evaluable fields were included in the analysis.

RESULTS

Leydig cell count and corpus cavernosum area were assessed from histological preparations processed as described above. Microscopic magnification ranging from 40× to 400× was used for evaluation. Leydig cells were operationally defined as cells located within the interstitial (extratubular) space between seminiferous tubules, exhibiting characteristic morphology and clearly distinguishable from surrounding cell types. Representative histological images of Leydig cells (400× magnification) and corpus cavernosum area (40× magnification) are presented in the **Figure 1A and 1B.**

Descriptive statistics were presented as mean, minimum value, maximum value, and standard deviation, which were visualized in tables, as shown in **Table 1.** Welch's t-test, a parametric statistical test, was employed to determine whether there were differences in the mean values of Leydig cell count and corpus cavernosum area between the control group and the Balinese arak treatment group. The analysis demonstrated a p-value < 0.05 for the Leydig cell count parameter, whereas the p-value for the corpus cavernosum area parameter was > 0.05. Accordingly, it can be concluded that a statistically significant difference was observed between the control and Balinese arak treatment groups with respect to Leydig cell count. In contrast, no statistically significant difference was identified in the corpus cavernosum area parameter between the two groups. The detailed results of the Welch's t-test analysis are presented in **Table 1**.

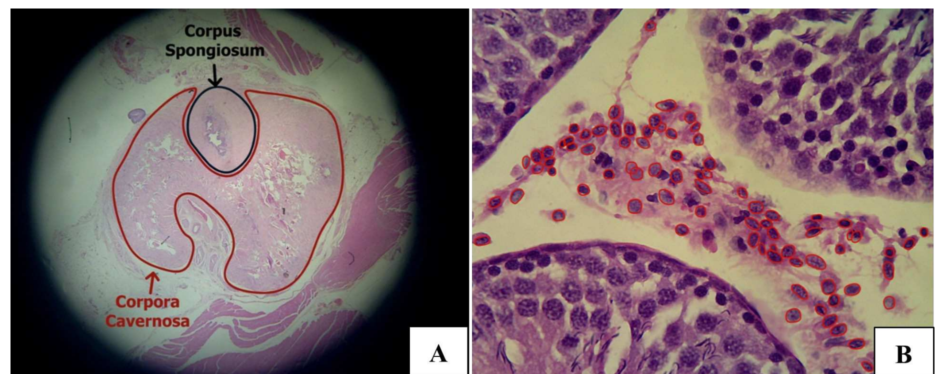

Figure 1. (A) Area of the Corpus Cavernosum of Wistar Rats (red line) and (B) Leydig cells of Wistar rats (red circles)

Table 1. Descriptive Test Data of Leydig Cell Count

Group	Minimum (cell/fow)	Maximum (cell/fow)	Mean (cell/fow)	SD	p
Leydig Cell Count	33.00	110.00	64.90	25.12	0.006*
Control	20.67	33.33	25.86	4.30	
Arak					
Corpus Cavernosum Area					
Control	70.29	123.22	85.76	20.08	0.171
Arak	70.19	77.67	73.90	2.65	

SD: Standard Deviations; Welch's t-test: *Statistically significant if p-value less than 0.05

DISCUSSION

Alcohol consumption has been shown to exert detrimental effects on the male reproductive system, particularly through structural and functional damage to Leydig cells within testicular tissue. Numerous studies have demonstrated that increases in alcohol concentration and dosage are directly proportional to the degree of cellular damage, whereby higher levels of alcohol exposure result in more severe Leydig cell injury. This damage is not confined to Leydig cells alone but is also closely associated with pathological alterations in the seminiferous tubules, including impairment of Sertoli cells, which play a crucial role in spermatogenesis, thereby disrupting overall testicular function.[13] In addition to its effects on testicular tissue, chronic alcohol consumption also adversely affects vascular function, particularly within erectile tissue. Experimental studies in rat models have demonstrated increased nitric oxide (NO) expression in the smooth muscle of the corpus cavernosum, accompanied by elevated expression of endothelial nitric oxide synthase (eNOS) and inducible nitric oxide synthase (iNOS). Despite increased NO production, this condition paradoxically results in reduced acetylcholine-induced relaxation of corpus cavernosum smooth muscle. The attenuation of endothelium-dependent relaxation reflects endothelial dysfunction, which subsequently contributes to the pathogenesis of erectile dysfunction associated with long-term alcohol exposure.[14]

Leydig cell dysfunction is closely associated with reduced testosterone production, as Leydig cells constitute the primary source of androgens in adult males.[15] Testosterone deficiency has been shown to induce structural alterations in erectile tissue, including a reduction in smooth muscle content and increased collagen deposition (fibrosis) within the corpus cavernosum, both of which contribute to impaired cavernosal relaxation and erectile function. Furthermore, androgens play a critical role in regulating the expression of endothelial nitric oxide synthase (eNOS) in penile tissue; therefore, androgen deficiency may reduce nitric oxide bioavailability and contribute to erectile dysfunction.[16] Chronic ethanol exposure has also been reported to disrupt Leydig cell function and decrease serum testosterone levels, thereby reinforcing the mechanistic link between Leydig cell damage, androgen deficiency, and cavernosal remodelling.[17]

A study conducted by Sius et al. investigating the effects of Dayak palm wine and aren palm wine consumption on Leydig cell destruction reported that higher alcohol doses were associated with greater cellular damage. Both Dayak and aren palm wines contain ethanol, which is likewise the principal alcohol component in Balinese arak. The seminiferous tubules, as the site of spermatogenesis, are highly dependent on the functional integrity of Sertoli and Leydig cells. Damage occurring within the seminiferous tubules is therefore closely related to injury to both cell types. The study concluded that higher doses of palm wine administration resulted in a significant reduction in Leydig cell count, particularly in the aren palm wine group (3.70 ± 0.17; $p < 0.05$).[18]

Another study by Mi-Hyeon Jang et al. examined alcohol-induced apoptosis in TM3 rat Leydig cells through Bax-dependent activation of caspase-3. To evaluate the cytotoxic effects of ethanol on TM3 cells, the cells were cultured with ethanol at final concentrations of 50, 100, and 500 mM for 12 hours, followed by assessment using an MTT assay. The results demonstrated $p < 0.05$ across comparisons, indicating that ethanol-induced cytotoxicity in TM3 cells was concentration-dependent. Alcohol exposure has been shown to induce apoptotic cell death in multiple organs, including the testes. Two key regulators of apoptotic cell death are Bcl-2 and a family of cysteine proteases known as caspases. Bcl-2 mRNA expression decreased significantly to 0.76 ± 0.03 following treatment with 50 mM ethanol and to 0.51 ± 0.05 with 100 mM ethanol ($p < 0.05$). Conversely, caspase-3 mRNA expression increased markedly to 3.85 ± 0.46 at 50 mM and to 5.57 ± 0.91 at 100 mM ethanol ($p < 0.05$).[19]

A further study evaluating morphological and molecular markers of apoptosis in the corpus cavernosum of rats subjected to chronic alcohol administration was conducted by Rogério et al. This study assessed apoptotic mechanisms through increased caspase-3 expression in the corpus cavernosum. Twenty-four Wistar rats were divided into control and alcohol-treated groups. Ethanol was administered at concentrations of 5%, 10%, and 20% during a two-week adaptation phase, followed by 20% ethanol administration for seven weeks. The findings demonstrated a significant increase in caspase-3 protein expression, as analysed by two-way ANOVA ($p < 0.0001$), indicating a significant difference between control and treatment groups.[20]

Elastic fibres, collagen fibres, and smooth muscle play essential structural roles in penile erection. A study conducted by See Min Choi et al. examining the effects of alcohol administration on the corpus cavernosum demonstrated a significant reduction in smooth muscle content and a marked increase in collagen fibre density in the 12-week alcohol group. The measured collagen fibre content was significantly higher in the 12-week alcohol group ($p < 0.05$). These findings suggest that chronic alcohol administration may lead to erectile dysfunction as a result of histological and ultrastructural alterations within the corpus cavernosum.[21]

Previous studies have clearly demonstrated that chronic alcohol administration for periods exceeding seven weeks produces significant reductions in corpus cavernosum mass or size. In the present study, alcohol administration for 45 days in Wistar rats resulted only in a decreasing trend in corpus cavernosum area. Studies investigating Leydig cell apoptosis by Sius et al. and Mi-Hyeon Jang et al. concluded that ethanol-containing alcohol exposure, particularly at higher concentrations, leads to an increased proportion of apoptotic Leydig cells. These findings are consistent with the present study, in which administration of Balinese arak containing 40% v/v ethanol (approximately equivalent to 6.85 M) produced a significant reduction in Leydig cell count.

CONCLUSION

Based on the findings of this experimental study in Wistar rats (Rattus norvegicus), it can be concluded that the administration of Balinese arak at a dose of 0.5 mL/day/rat for 45 days resulted in a significant reduction in Leydig cell count compared with the control group. These findings indicate that exposure to Balinese arak has the potential to induce structural damage to testicular tissue, particularly to Leydig cells, which play a critical role in testosterone production and the maintenance of male reproductive function. Although the mean corpus cavernosum area in the treatment group demonstrated a decreasing trend compared with the control group, the difference did not reach statistical significance. This finding suggests that the duration and dosage of Balinese arak exposure in the present study were insufficient to induce significant morphological alterations in erectile tissue. Nevertheless, the observed direction of change is consistent with the proposed mechanism underlying erectile dysfunction associated with chronic alcohol consumption.

ETHICS CONSIDERATIONS

This study has received ethical clearance from the Ethics Committee of the Faculty of Medicine, Udayana University, with approval number: 0371/UN14.2.2.VII.14/LT/2025.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest regarding this study publication.

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AUTHOR CONTRIBUTIONS

IGNP and INRS contributed to the conceptualization and design of the study, conducted the experimental procedures, performed the relevant literature search, carried out the statistical analysis, and drafted and edited the manuscript. SRHN, NPWP, and NMADS contributed to conducting the experimental study, data collection, and the relevant literature search. IGNP and INRS contributed to supervision, manuscript review, providing scientific input for revisions, and proofreading the manuscript. The authors declare that no generative artificial intelligence (AI) tools or AI-assisted technologies were used in the writing, analysis, or preparation of this manuscript.

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