IMC Journal of Medical Science
July 2026; Vol. 20(2):001
DOI: https://doi.org/10.55010/imcjms.20.010

Open Access
Original Article

Association of shisha smoking with reproductive endocrine biomarkers: focus on prostate-specific antigen and anti-müllerian hormone

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Abstract

Background and objective: Shisha smoking is associated with multiple health risks, including reproductive disorders. This study investigated the impact of shisha smoking on prostate function and ovarian reserve by measuring serum prostate-specific antigen (PSA) and anti-müllerian hormone (AMH) levels.

Materials and methods: A cross-sectional study design was employed, involving 180 young adults aged 18–35 years. The participants comprised 90 males and 90 females who were randomly selected. They were categorized into three groups: 60 active shisha smokers, 60 secondhand smokers, and 60 non-smokers (30 males and 30 females in each category). Venous blood samples were withdrawn from all participants for the analysis of prostate-specific antigen (PSA) and anti-müllerian hormone (AMH) using the enzyme-linked immunosorbent assay technique. The obtained data were analyzed using the SPSS, with statistical significance $p < 0.05$.

Results: 17.5% of the 120 shisha smokers (active and secondhand smokers) reported reproductive disorders. The observed conditions included dysmenorrhea (8.3%), abnormal menstrual bleeding (3.3%), amenorrhea (2.5%), and erectile dysfunction (1.7%). Comparison of the AMH levels between shisha smokers and non-smokers showed that the mean anti-mullerian hormone levels were significantly lower (p=0.001) in shisha smokers (2.49 ± 0.52 ng/mL) and secondhand smokers (2.19 ± 0.25ng/mL) compared to non-smokers (2.71 ± 0.29 ng/mL). The mean prostate specific antigen levels were significantly higher in shisha smokers (0.62 ± 0.78 ng/mL) and secondhand smokers (0.69 ± 0.48 ng/mL) compared to non-smokers (0.36 ± 0.45 ng/mL).

Conclusion: Shisha smoking is associated with alterations in serum prostate specific antigen (PSA) and anti-müllerian hormone (AMH) levels in the study population.

Introduction

Tobacco use is increasing globally and has become a major public health concern. It is one of the principal causes of preventable morbidity and mortality, accounting for about eight million annual deaths worldwide [1]. Tobacco is consumed in

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many forms, including cigars, cigarettes, roll-your-own, pipes, chewable tobacco, and shisha (waterpipe). Shisha, also known as waterpipe or hookah, is a traditional tobacco-smoking device consisting of a pipe with a long, flexible tube through which the user draws smoke from a flavoured or unflavoured tobacco preparation, burned by charcoal. The smoke is cooled through water before it is inhaled [2].

The World Health Organization reported that over 100 million people worldwide engage in daily shisha smoking, cutting across both sexes and various age groups, with the highest prevalence observed among individuals aged 15–30 years [3,4]. This increasing trend is driven by the availability of attractive flavors and the misconception that waterpipe smoking poses less or no harm to human health compared to other forms of tobacco smoking [5,6]. However, accumulating evidence indicates that waterpipe contains several potentially toxic and carcinogenic chemicals like nicotine, heavy metals, volatile organic chemicals, and polycyclic aromatic hydrocarbons, that have detrimental effects on human health including cardiovascular and respiratory diseases, cancer, metabolic syndrome, infectious diseases, as well as genotoxic and epigenetic changes[7,8].

While studies on the adverse effects of tobacco use have traditionally been centered on respiratory and cardiovascular problems, there is an increasing interest on the endocrine-disrupting potential of shisha tobacco smoke [9]. Endocrine disruption chemicals (EDC) are a heterogenous group of exogenous compounds found in pesticides, pharmaceuticals, and synthetic food products, that interfere with hormone synthesis, secretion, transport, and action, leading to the alterations in physiological processes[10]. Shisha contains nicotine, heavy metals and other toxicants that potentially impacts the synthesis, secretion, and concentrations of hormones in both males and females, especially from the hypothalamus, thyroid, adrenal gland, ovaries, and testis [11,12]. In males, such disruptions may affect androgen-regulated processes, while in females, it may interfere with ovarian function and folliculogenesis. Despite these concerns, the biochemical impact of shisha smoking on specific hormonal biomarkers remains insufficiently studied.

Anti-müllerian hormone (AMH) and prostate-specific antigen (PSA) are well-documented biomarkers of reproductive physiology in females and males respectively. AMH, produced by ovarian granulosa cells, is a reliable biomarker for assessing ovarian reserve[13]. It is secreted by developing antral follicles and reflects the pool of primordial follicles. Due to its secretion pattern, AMH exhibits minimal fluctuation across the menstrual cycle, enhancing its clinical utility [14]. PSA is an androgen-regulated serine protease produced by both prostate epithelial cells and prostate cancer (PCa) and is the most commonly used serum marker for cancer [15]. Changes in the concentrations of these hormones may indicate underlying endocrine disturbances induced by environmental exposures such as shisha smoke.

Several studies have documented the detrimental effects of cigarette smoking on key reproductive biomarkers, including prostate specific antigen and anti-mullerian hormone[16,17,18]. These findings suggest that tobacco exposure may disrupt both prostate function in males and ovarian reserve in females through mechanisms such as oxidative stress and endocrine disturbance. Furthermore, many researchers have examined the effect of shisha smoking on reproductive system, with particular emphasis on alterations of luteinizing hormone (LH), follicle-stimulating hormone (FSH), estrogen, progesterone, and testosterone [19,20]. However, most of these studies have focused on general reproductive hormones, with limited attention on specific biomarkers of prostate function and ovarian reserve. The relationship between shisha smoking and PSA and AMH remains inadequately studied. Therefore, this study aims to investigate the impact of shisha smoking on prostate function and ovarian reserves by evaluating plasma PSA and AMH levels among young adults.

Materials and methods

Study area and population: This research was conducted in Yenagoa town, the capital city of Bayelsa State, Southern Nigeria. One hundred and eighty (180) young adults comprising 90 males and 90 females aged 18-35 years were recruited for the study. The participants were 60 regular exclusive

shisha users (30 males and 30 females) who smoke shisha at least thrice per week for at least 2 years, 60 secondhand shisha smokers (30 males and 30 females) who worked daily for 12 hours in enclosed environments such as bars, nightclubs, and lounges for more than 2 years, and 60 apparently healthy individuals (30 males and 30 females) without a history of any form of tobacco smoking and without known chronic or reproductive disorders at the time of the study. The exposure assessment of the secondhand smokers was based on participants' self-reported history of exposure daily for over 2 years.

Study design*:* This cross-sectional study was conducted in Yenagoa city, Bayelsa State, Southern Nigeria from March 2024 to October 2024. Participants for the study were selected using a simple random sampling technique to ensure that every eligible individual within the study population had an equal chance of being included in the study. Ethical approval was obtained from the Research and Ethics Committee of the Bayelsa State Ministry of Health (Approval No: BSHREC/Vol. 1/24/03/04), as well as written informed consent from the study participants.

Sample size determination*:* Sample size was determined using Cochran formula:

$$n = \frac{Z^2 \times p\,(1-p)}{(d)^2}$$

n= Sample size, Z= Confidence level at 95% (1.96), p= Prevalence rate of 7.1% in Lagos, Nigeria (0.071) [21], d= Error probability at 5% (0.05). The calculated minimum sample size was approximately 101 participants. After considering 10% attrition rate, the sample size increased to 111 participants. However, to improve statistical power, a total of 180 participants were recruited for the prevalence study.

"The sample size was estimated using G*Power version 3.1 based on one-way ANOVA for comparison of mean hormonal levels among the three study groups (non-smokers, shisha smokers, and secondhand smokers). The assumptions used included an effect size (f) of 0.25, alpha level of 0.05, statistical power of 80%, and three study groups. The minimum required sample size was calculated to be 159 participants (53 per group). To account for possible non-response, 180 participants were recruited. Equal subgroup sizes (60 participants per group) were selected to ensure balanced group comparisons and improve the statistical power and validity of the one-way ANOVA analysis. Participant allocation was therefore based on statistical considerations.

Selection criteria*: Inclusion criteria*: Individuals aged 18–35 years with no history of chronic metabolic diseases or fertility problems were included in the study. Participants comprised active shisha smokers who had smoked at least thrice weekly for a minimum of two years; secondhand shisha smokers who had worked daily for 12 hours in enclosed environments such as bars, nightclubs, and lounges for at least two years; and apparently healthy non-smokers with no history of tobacco use.

Exclusion criteria: Individuals with known chronic medical conditions, hormonal imbalances, use of other forms of tobacco, obese, irregular menstrual cycles, erectile dysfunction, low sperm volume, or a history of alcohol or substance abuse, or contraceptive users were excluded.

Data collection and preparation*:* After receiving written consent from the participants, a well-structured questionnaire was administered and retrieved after answering the relevant questions. Before the commencement of the study, the questionnaire was given to an experienced physician to validate the questionnaire. The questionnaire contained three sections namely: demographic information such as gender, age, education, occupation or marriage status, and employment status; shisha smoking status and duration; Chronic health effects of shisha smoking (e.g. cardiovascular, respiratory, reproductive and cancer diseases); and specific reproductive disorders of shisha smoking (erectile dysfunction, amonnorrhea, low libido, dysmenorrhea, and abnormal menstrual bleeding). Information on clinically diagnosed chronic diseases was self-reported by participants through the questionnaire. The data were collected one-on-one between the interviewer and the participants. Five milliliters of venous blood were collected into a plain sample container and allowed to stand undisturbed at room temperature to clot for 45 minutes. The clot was dislodged and centrifuged at 1000 rpm for 10 minutes, and the clear serum was obtained and dispensed into a well labeled tube for

measurement of anti-mullerian hormone and prostate specific antigen.

Specimen analysis: AMH: Serum sample obtained from female participants were used to measure AMH level in accordance with a double-antibody sandwich ELISA method as reported by Oke et al.[22] using human AMH enzyme linked immunosorbent assay (ELISA) kit by Span Biotech Ltd. Thekit's sensitivity was 0.023ng/mL.

PSA: Serum sample obtained from the male participants was analyzed in accordance with ELISA method reported by Hussein et al. [15] with modifications using Microplate Enzyme Immunoassay kit manufactured by ATLAS Medical, Blankenfelde-Mahlow, Germany. PSA assessment in the healthy young males was intended to explore possible early subclinical changes associated with the study exposure.

Statistical analysis*:* Data obtained were analyzed using Statistical Package for the Social Sciences (SPSS) software Version 23.0 (IBM Corp., Armonk, NY, USA). A two-tailed t-test and One-way ANOVA was used for comparing mean values of the measured biochemical parameters between the non-smokers and experimental groups. Post Hoc test (Bonferroni) was used totests significance between groups. P-values < 0.05 were considered significant.

Results

Out of the one hundred-twenty (male and female) shisha smokers, 73 (60.8%) had chronic diseases, while 47 (39.2%) were apparently healthy. Respiratory disorders 31 (25.8%) were most prevalent among the smokers, followed by reproductive disorders 21 (17.5%), cardiovascular disorders 17 (14.1%), and cancer 02 (1.7%) and diabetes mellitus 02 (1.7%)(Table-1).

Table-1: *Prevalence of chronic diseases among shisha (waterpipe) smokers under study (n=120).*

Variables	Frequency (n)	Percentage (%)
Apparently Healthy Persons	47	39.2
Respiratory Diseases	31	25.8
Cardiovascular Diseases	17	14.1
Reproductive Disorders	21	17.5
Cancer	02	1.7
Diabetes Mellitus	02	1.7

The most prevalent reproductive disorder reported by the smokers was dysmenorrhea 10 (8.3%), followed by abnormal menstrual bleeding 04 (3.3%), amenorrhea 03 (2.5%), and erectile dysfunction and low libido 02 (1.7%) (Table-2). The mean AMH levels were significantly higher in non-smokers (2.71 ± 0.29 ng/mL) compared with shisha smokers (2.49 ± 0.52 ng/mL) and secondhand shisha smokers (2.19 ± 0.25 ng/mL) (p=0.001). The mean difference between non-smokers and shisha smokers was 0.22 ng/mL (95% CI: 0.07–0.37; Cohen's d = 0.52), while the difference between

Table-2: *Prevalence of reproductive disorders among shisha (waterpipe) smokers under study (n=120).*

Variables	Frequency (n)	Percentage (%)
Erectile dysfunction	02	1.7
Low libido	02	1.7
Dysmenorrhea	10	8.3
Abnormal Menstrual bleeding	04	3.3
Amenorrhea	03	2.5
Other chronic diseases	52	43.3
Apparently Healthy Person	47	39.2

non-smokers and secondhand smokers was 0.52 ng/mL (95% CI: 0.42–0.62; Cohen's d = 1.92). AMH levels were also significantly lower in secondhand smokers compared with shisha smokers, with a mean difference of 0.30 ng/mL (95% CI: 0.15–0.45; Cohen's d = 0.74) (Table-3).

Table-3: *Mean AMH and PSA levels in female shisha smokers and nonsmokers under study*

Parameter	Mean ± SD			*F-value*	*P-value*
	Non-smokers (n = 30)	**Shisha smokers (n = 30)**	**Secondhand smokers (n = 30)**		
AMH (ng/mL)	2.71 ± 0.29[a]	2.49 ± 0.52[b]	2.19 ± 0.25[c]	8.12	0.001*
PSA (ng/mL)	0.36 ± 0.45 [a]	0.62 ± 0.78[b]	0.69 ± 0.48[c]	2.39	0.036*

*AMH= Anti-mullerian Hormone; p<0.05 represents significant difference. One-way ANOVA followed by post hoc analysis was used for group comparisons. Values with different superscripts (a, b, c) are significantly different from each other at p < 0.05. *p < 0.05 indicates statistical significance.*

*PSA= Prostate Specific Antigen; One-way ANOVA followed by post hoc analysis was used for group comparisons. Values with different superscripts are significantly different at p < 0.05. Groups sharing the same superscript are not significantly different. *p < 0.05 indicates statistical significance.*

The mean PSA level was 0.36 ± 0.45 ng/mL in non-smokers, compared with 0.62 ± 0.78 ng/mL in shisha smokers and 0.69 ± 0.48 ng/mL in secondhand smokers (p <0.036). The mean difference between non-smokers and shisha smokers was −0.26 ng/mL (95% CI: −0.49 to −0.03; Cohen's d = 0.41), while the difference between non-smokers and secondhand smokers was −0.33 ng/mL (95% CI: −0.50 to −0.16; Cohen's d = 0.71). However, PSA levels between shisha smokers and secondhand smokers showed no statistically significant difference, with a mean difference of −0.07 ng/mL (95% CI: −0.30 to 0.16; Cohen's d = 0.11) (Table-3). The mean AMH values in individuals exposed to shisha smoke for greater than 5 years was significantly lower than less than 5 years. While the mean PSA levels were significantly higher inindividuals exposed to shisha smoking for more than 5 years (Table-4).

Table-4: *Mean AMH and PSA levels in shisha (waterpipe) smokers according to duration of smoking.*

Parameter	Mean ± SD			*F-value*	*P-value*
	≤2 years (n = 37)	**>2 – 5 years (n = 51)**	**> 5 years (n = 32)**		
AMH (ng/mL)	2.58 ± 0.50	2.49 ± 0.36	2.16 ± 0.23	5.74	0.005*
PSA (ng/mL)	0.34 ± 0.32	0.59 ± 0.54	0.83 ± 0.42	6.18	0.003*

AMH= Anti-mullerian Hormone; PSA= Prostate Specific Antigen; p<0.05 represents significant difference.

Discussion

The reproductive physiology is a complex interaction between neuroendocrine and endocrine signaling affecting the hypothalamus-pituitary axis and the sex organs [23] (Goldsammler et al, 2018). Exposure to environmental pollutants like tobacco smoke, has many deleterious effects on both male and female reproductive systems[24]. Shisha (waterpipe) smoke contains over 82 harmful and carcinogenic chemicals such as carbon-monoxide, heavy metals, nicotine, tar, volatile organic compounds, and polycyclic aromatic hydrocarbons, many of which possess endocrine-disruption features capable of interfering with reproductive function [25]. A typical shisha smoking session releases approximately 100 to 200 puffs, compared

to about 10 to 15 puffs from a single cigarette[2], resulting in significantly greater smoke exposure per session. Therefore, shisha smoking may exert greater adverse effect on the reproductive system than cigarette smoking[26].

In the present study, 17.5% of the 120 shisha smokers accepted that they have reproductive disorders. The observed conditions included dysmenorrhea (8.3%), abnormal menstrual bleeding (3.3%), amenorrhea (2.5%), and erectile dysfunction/low libido (1.7%). Several studies indicated hormonal imbalance, reduced sperm count, motility, and viability, menstrual irregularities, reduced ovarian reserve, and difficulties in conceiving among waterpipe smokers [27,28,29].

AMH, produced by ovarian granulosa cells, is a reliable biomarker for assessing ovarian reserve. It is secreted by developing antral follicles and reflects the pool of primordial follicles[13]. Cigarette smoking has been associated with ovarian reserve, reduced responsiveness to ovarian stimulation and impaired fertility outcomes. It accelerates follicular atresia and depletion of the primordial follicle pool, thereby contributing to premature ovarian insufficiency and earlier menopause [30,31].

In our study, comparison of the AMH values between shisha smokers and non-smokers showed that the mean AMH levels were significantly ($p<0.001$) lower in shisha smokers (2.49 ± 0.52 ng/mL) and secondhand smokers (2.19 ± 0.25ng/mL) compared to non-smokers (2.71 ± 0.29 ng/mL). Although the values remained within the reference range (1.2ng/mL- 3.2 ng/mL), the observed reduction suggests early subclinical impairment of ovarian reserve, particularly in individuals of reproductive age. Smoking shisha exposes the body to a number of harmful chemicals that increase the generation of reactive oxygen species (ROS), which causes oxidative stress. Lipid peroxidation and damage to ovarian follicular structures, particularly granulosa cells, can result from this oxidative disturbance [32,33]. As granulosa cells are the primary source of anti-müllerian hormone (AMH), their loss during follicular atresia contributes to a decline in AMH levels. The lower AMH levels observed in secondhand shisha smokers compared to active smokers may be attributed to differences in frequency and duration of exposure, as well as prolonged inhalation of toxic substances from the charcoal such as carbon monoxide, heavy metals, and particulate matter. However, findings from previous studies were inconsistent. Irteimah et al. [34] reported significant lower AMH level in non-smokers compared to hookah smokers, whereas Albeitawi et al. [35] reported a slight increase in AMH levels among shisha smokers.

Prostate cancer is one of the most common cancers and the second leading cause of cancer-related deaths affecting men globally [36]. Prostate-specific antigen (PSA), an androgen-regulated serine protease secreted by prostate epithelial cells, it is the most commonly usedserum biomarker for prostate cancer [15]. However, PSA is not a specific marker for prostate cancer, as its levels may also be elevated in benign prostatic hyperplasia and prostatitis[17].

The nexus between tobacco smoking and prostate cancer remains controversial. Some authors report no direct correlationbetween smoking and prostate cancer incidences, while others indicated a reduced risk of developing prostate cancer in current smokers, possibly due to lower prostate-specific antigen (PSA) screening rates in the population[25,37]. However, accumulating evidence suggests that smoking is associated with more aggressive, high-grade prostate cancer, as well as poorer clinical outcomes[38,39].

In our study, the mean PSA values were significantly higher in shisha smokers (0.62 ± 0.78 ng/mL) and secondhand smokers (0.69 ± 0.48 ng/mL) compared to non-smokers (0.36 ± 0.45 ng/mL) ($p=0.036$). The PSA levels were slightly higher in secondhand smokers than active shisha smokers. Despite these differences, the obtained PSA values remained below the threshold typically associated with increased risk of prostate cancer. These findings align with previous reports by Koc and colleagues [17], who reported elevated PSA levels in smokers, and Hosseini and colleagues [37], who reported waterpipe smoking as a potential risk factor for prostate cancer.

A plausible explanation for elevated PSA levels in smokers may involve hormonal mechanisms. Tobacco smoking has been linked to elevated serum

testosterone levels, which stimulate prostatic epithelial cells responsible for the synthesis and secretion of PSA. Testosterone is further converted to dihydrotestosterone (DHT) by the enzyme 5α-reductase, and DHT is a more potent androgen that plays a critical role in prostate growth and function. Since the prostate depends on androgens such as testosterone and DHT for its development and maintenance, increased androgen activity may contribute to elevated PSA levels[40].

Furthermore, this study revealed that AMH levels were significantly lower in individuals who had smoked shisha for greater than 5 years compared to those with less than 5 years of exposure; while PSA levels were significantly higher in individuals with longer duration of exposure. These findings suggest a duration-dependent effect of shisha smoking on reproductive hormones.

This study also highlighted that both active and secondhand shisha smokers exhibited altered AMH and PSA levels compared to non-smokers, indicating that even passive exposure may have measurable biological effects. To the best of our knowledge, few studies have investigated the effects of active shisha smoking and secondhand shisha smoking on AMH and PSA in 18 to 35 age group. Our findings found that shisha smoking may adversely affect ovarian reserve and prostate physiology, as seen by the serum levels of AMH and PSA. However, the exact mechanisms underlying these alterations remain unclear. Notably, the limited available studies on waterpipe smoking and AMH report conflicting findings [34,35], highlighting the need for further research in this area.

Conclusion

Our finding demonstrates that both active and secondhand shisha smoking are associated with significant alteration of serum anti-müllerian hormone (AMH) and prostate-specific antigen (PSA) levels in young adults. Furthermore, the observed alterations appear to be duration dependent.

Limitations

The sample size may limit the generalizability of our findings, particularly for reproductive disorders with relatively low prevalence. Therefore, future studies involving larger, multi-center populations be conducted to further validate and strengthen these findings.

Conflict of interest

The authors declare no conflicts of interest.

Author contributions

ENO- Conceptualization, sample/data collection, laboratory work, data entry and analysis and manuscript writing; FCE- supervision; laboratory work; review and editing; OJB- data collection, data entry, review and editing; OOS- Data collection, laboratory work, data entry and analysis.

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Cite this article as:
Onitsha EN, Ezeiruaku FC, Borobueb OJ, Sylvia OO. Association of shisha smoking with reproductive endocrine biomarkers: focus on prostate-specific antigen and anti-müllerian hormone. *IMC J Med Sci.* 2026; 20(2):001.
DOI: https://doi.org/10.55010/imcjms.20.010.