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Relationship between smoking, excessive androgen and negative emotions in women with polycystic ovary syndrome (PCOS)

Yang Yang[1,2†], Hua Zhang[1,2†], Bo-Yun Huang[1], Yong-Hao Lu[1], Ii Fukuzawa[1,2], Shuhan Yang[1,2], Lifei Zhou[1,2], Liyan Luo[2], Chunyong Wang[1,2], Ning Ding[1,3], Sai Li[1], Li Shi[2] and Hao-Lin Zhang[1,2,4*]

Abstract

Background Lifestyle intervention is the first-line treatment for PCOS. Numerous studies have investigated the effect of various lifestyle factors, including dietary habit, smoking, and alcohol consumption on PCOS women. These studies have found that such factors may be associated with physiological parameters such as androgen, and emotional states like anxiety or depression. Smoking, a harmful lifestyle habit widely recognized to contribute to various diseases, has also been found to be related to PCOS. Current research has not adequately compared the effects of smoking with other lifestyle habits on PCOS, and there is little mention of its relationship with the emotional states of patients with PCOS. To further elucidate the association between smoking and other lifestyle factors with clinical symptoms in patients with PCOS, we conducted a cross-sectional evaluation using data from Peking University Third Hospital, with a special focus on analyzing smoking habits and comparing it with a variety of lifestyle factors.

Methods This cross-sectional study included 601 PCOS women and 184 healthy controls who underwent physical examinations, hormone profiles and psychological measures. We assessed the association between smoking and the clinical symptoms in PCOS women.

Results We found a significant correlation between smoking and the degree of depression in PCOS women among the three emotional states: anxiety, depression, and stress. Smoking was also significantly associated with testosterone level in PCOS participants, suggesting that PCOS women who smoke exhibited more severe depressive symptoms and higher testosterone level. In addition, compared to the control group, PCOS women had notably higher testosterone (T) and luteinizing hormone (LH) levels. Smoke and alcohol were statistically significantly more common in women with PCOS than the Control.

Conclusion Women with PCOS who smoke were found to have elevated testosterone levels and more severe depression. These findings suggest that clinicians should monitor smoking women with PCOS for symptoms of depression and assess their hyperandrogenic status.

Keywords polycystic ovary syndrome, smoking, testosterone, negative emotions

†Yang Yang and Hua Zhang contributed equally to this work.

*Correspondence:
Hao-Lin Zhang
zoe@bjmu.edu.cn

Full list of author information is available at the end of the article

Introduction

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders in women. Its prevalence among the general female population ranges from 6–13% [1], depending on the diagnostic criteria and studied cohorts. This syndrome presents a range of complex clinical features, including irregular ovulation and menstruation, hyperandrogenism, polycystic ovaries detected via ultrasound, metabolic dysfunction, and insulin resistance. Due to the metabolic disturbances and insulin resistance caused by PCOS, Type 2 Diabetes Mellitus (DM) is a common complication observed in PCOS women [2].

In the treatment of PCOS, although surgery and medication are conventional methods, the aim of medication is to attempt to control the symptoms; so far, there is no clinical method capable of curing PCOS. However, some studies indicate that dietary adjustments are an important therapeutic approach for improving PCOS. For women with PCOS, appropriate adjustments in diet and exercise are crucial treatment strategies that help enhance their overall health. In this context, gaining a better understanding of the disease risk factors, especially the impact of unhealthy lifestyle habits on PCOS, is of significant importance for the treatment and management of Polycystic Ovary Syndrome.

Smoking, as a significant factor harmful to human health, has been widely studied and discussed in academic circles. In recent years, its impact on Polycystic Ovary Syndrome (PCOS) has attracted the attention of scholars. Smoking constitutes a crucial modifiable risk factor, with studies indicating its potential role in reproductive disorders [3]. Additionally, research suggests that smoking may further increase the risk of complications during pregnancy in PCOS women [4]. A study utilizing Mendelian randomization with two sample sets concluded that the genetic predisposition to smoking initiation is associated with an increased risk of Polycystic Ovary Syndrome [5].

Various chemical substances generated from smoking can influence the human body's metabolism, endocrine system, and other processes, leading to various diseases. Studies have shown that smoking during pregnancy increases the risk of polycystic ovary syndrome in offspring. Current research indicates a close relationship between exposure to polycyclic aromatic hydrocarbons (PAHs) and adverse reproductive outcomes [6]. Smoking is one of the main factors causing exposure to PAHs. Furthermore, research has found that smoking during pregnancy increases the risk of offspring developing polycystic ovary syndrome [7]. Therefore, there is a certain relationship between smoking and decreased fertility.

In terms of ovarian reserve (OR), heavy or prolonged smoking increases the risk of ovarian reserve decline [8]. Additionally, smoking, snoring, high-calorie diet, and other factors are associated with an increased risk of ovulatory dysfunction in women with polycystic ovary syndrome (PCOS) [9]. Relevant studies suggest that smoking may reduce ovarian tissue perfusion in PCOS women by affecting androgen metabolism, leading to various reproductive disorders [10].

In the context of insulin resistance, nicotine is a potential trigger for cardiovascular metabolic disorders mediated by insulin signaling. Smoking can lead to impaired β-cell function and insulin resistance, consequently causing hyperinsulinemia and hyperglycemia [11]. Another study indicates that smoking increases the risk of gestational diabetes mellitus (GDM) in patients with polycystic ovary syndrome (PCOS), but does not decrease their risk of hypertension as it does in the general population. The research suggests that PCOS women have a higher baseline risk for various complications, surpassing the impact brought about by smoking itself [4].

In the context of hyperandrogenism in Kaohsiung, research has revealed that if a patient with Polycystic Ovary Syndrome (PCOS) has a husband who engages in daily smoking, the likelihood of exacerbation of her hyperandrogenism in Kaohsiung is significantly increased [12].

In terms of metabolic function, smoking increases reactive oxygen species (ROS) in the human body, leading to oxidative stress and metabolic disturbances [13]. Research has found that smoking may further exacerbate the metabolic disorders already present in patients with PCOS.

SHBG is also an important factor associated with PCOS, and has been reported in many previous works. Meta-analysis conducted by Y. Li et al. revealed the association between lower SHBG concentrations and higher risk of polycystic ovary syndrome (PCOS) [14]. A significant lower SHBG level was found in smoking PCOS women by J. Niepsuj et al. [15], as well as in PCOS women exposed to secondhand smoke by J. Li et al. [16]. Currently, numerous studies indicate a certain connection between smoking and PCOS. However, the comparison between the effects of smoking with other lifestyle habits and negative emotions on PCOS has rarely been reported.

To address these issues, we conducted a cross-sectional evaluation using data from Peking University Third Hospital, with a special focus on analyzing smoking habits. Despite the observational and cross-sectional nature of this study, which limits the ability to establish causal relationships, our study aimed to investigate whether smoking and various lifestyle factors could impact the emotional and clinical manifestations of PCOS women.

While this study cannot establish a causal relationship between smoking and PCOS symptoms, it provides a comprehensive analysis of various lifestyle factors, including smoking, and their potential influence on the emotional and clinical presentation of PCOS women. This research offers deeper insights, assistance, and support to clinical practitioners in the diagnosis and treatment of PCOS.

Method

Ethical approval

The clinical trial was approved by the Regional Ethical Review Board of Peking University Third Hospital (PKU3-IRB-2016-212-02) and registered on ClinicalTrials.gov (registration number NCT04264832). All participants were informed of the purpose, content and risks of the study. Written informed consent was obtained before the start of the study. All experimental procedures were approved by the Animal Care and Use Committee of Peking University Third Hospital (PKU3-IRB-2019-029-02).

Participants

A large-scale epidemiological study was conducted in women of reproductive age (19–45 years). From March 2016 to December 2021, 601 women with PCOS and 184 age-matched controls were enrolled at Peking University Third Hospital. Patients were eligible if they fulfilled the following Rotterdam diagnostic criteria 2003 [17] with at least two of the following three symptoms (1) infrequent ovulation or anovulation; (2) hyperandrogenism or clinical manifestations of high blood androgen; (3) ultrasound findings of polycystic ovaries in 1 or 2 ovaries, or ≥12 follicles measuring 2 to 9 mm in diameter and/or ovarian volume > 10 mL [18]. Individuals were excluded from the study if they had other endocrine disorders such as non-classical adrenal hyperplasia (17-hydroxyprogesterone < 3 nmol/L), thyroid dysfunction (thyroid-stimulating hormone < 0.55 or > 4. 78 mIU/mL) or hyperprolactinemia (fasting prolactin < 26 ng/mL), type I diabetes or poorly controlled type II diabetes, stage 2 hypertension (resting blood pressure ≥ 160/100 mmHg), psychiatric diagnoses or use of psychiatric medications, and none of the women had received any pharmacological or surgical treatment (except for allergy medications and occasional painkillers) within 12 weeks before entering the study. This study excluded women who had received any form of pharmacological treatment, including hormonal contraceptives, within 12 weeks prior to the study's initiation.

Control women were healthy, with no history of endocrine disorders, no clinical or biochemical evidence of hyperandrogenism (total testosterone < 60 ng/mL, free testosterone < 2 ng/mL, DHEAS < 271 μg/dL), and normal ovarian morphology on ultrasound [18]. They are excluded if they have menstrual irregularities, evidence of hyperandrogenism (Ferriman-Gallwey score > 4), or evidence of polycystic ovarian morphology on ultrasound.

Clinical data collection

All study participants received a questionnaire and underwent a physical examination. Blood samples were taken from a sub-sample of women (PCOS, $n=601$; control, $n=184$) for analysis of metabolic markers and hormones. Hyperandrogenism was assessed using the Rotterdam criteria for PCOS.

Basic characteristics

Participants were carefully characterized with regard to general health, medical history, clinical, demographic (age, race/ethnicity) and anthropomorphic measurements (body mass index, waist and hip circumference) using questionnaires, interviews and physical examination [19]. Meanwhile, participants were assessed for blood pressure, breast, thyroid, premature alopecia, and any uterine and/or ovarian problems by physical and pelvic examinations.

Hormone profiles

The hormonal profile including estrogen (E2, pmol/L), luteinizing hormone (LH, mIU/mL), serum follicle stimulating hormone (FSH, mIU/mL), prolactin (PRL, ng/mL), total testosterone (T, nmol/L) and androstenedione (A2, nmol/L) in humans were measured using the Siemens Immulite 2000 immunoassay system (Siemens Healthcare Diagnostics, Siemens, Germany) [20].

Psychological measures

Psychological states were assessed using three validated questionnaires. The Perceived Stress Scale (Chinese 14-item PSS) [21], the Self-Rating Anxiety Scale (SAS) [22] and the Self-Rating Depression Scale (SDS) [23] were used to assess mental health status.

Statistical analysis

All clinical statistical analyses were performed with R version 4.2.3 (R Foundation, Vienna, Austria. www.r-project.org). For continuous (quantitative) data, the Shapiro normality test was used to determine the normality of the sample data. Normally distributed continuous variables were expressed as mean ± standard deviation and compared using a two-tailed t-test. Abnormally distributed continuous variables were expressed as median (upper quartile, lower quartile) and compared using the Kruskal-Wallis test. Categorical (qualitative) data were described statistically using frequencies (percentages) and comparisons between groups were made using the $\chi 2$ test or Fisher's exact test. Differences were considered

statistically significant if the two-tailed *p*-value was less than 0.05.

Result

Participants and clinical characteristics

A total of 785 women fulfilled the inclusion criteria. Basic information, endocrine and metabolic parameters were available for analysis (Table 1). They included 601 women with PCOS and 184 healthy women for final analysis. The PCOS women had statistically significantly higher levels of TG, LH, LH/FSH, E2, T and A2 than the controls (all $P<0.001$). In addition, PCOS women scored statistically significantly higher on SAS, SDS and PSS ($P<0.001$, $P<0.001$ and $P<0.05$), indicating more negative emotions.

Diet and physical activity between the PCOS group and the control group was available in Table 2. Smoke and alcohol were statistically significantly more common in PCOS women than women without PCOS ($P<0.05$ and $P<0.001$). There were no statistically significant differences between the PCOS and control group for snack, coffee, milk, dessert and fried food ($P=0.233$, $P=0.257$, $P=0.116$, $P=0.109$ and $P=0.137$). It is crucial to account for sociodemographic variables, as these factors may be associated with anxiety and depression in women with PCOS, and our study indicates that there were no statistically significant differences in sociodemographic characteristics between the PCOS and control groups (Table 1).

PCOS smokers had higher T than PCOS non-smokers

As there were only 18 (3%) regular smokers in the PCOS group, they were combined with 77 (12.8%) occasional smokers to form a smoking group of 95 (15.8%). The comparison between non-smokers and smokers in women with and without PCOS is shown in Table 3. The S-PCOS group had statistically significantly higher T levels than the NS-PCOS group and the S-control group (both $P<0.05$, Fig. 1). The S-PCOS group also had

Table 1 Participant characteristics

Variables[a]	Control (*N* = 184)	PCOS (*N* = 601)	*P* value
Age(year)	30(27,33)	28(26,31)	< 0.001***
BMI (kg//m^2)	23.3(20.9,27.02)	25.1(21.6,28.5)	0.001**
WHR	0.81(0.78,0.86)	0.84(0.79,0.88)	0.01*
HOMA-IR	2.5(1.44,3.63)	2.4(1.61,3.67)	0.827
HOMA-B	142.19(81.2,229.82)	148.94(103.06,220.82)	0.592
PRL (ng/mL)	10.9(8.62,16)	11.15(8.44,14.88)	0.699
LH (mIU/mL)	3.84(2.43,5.32)	6.56(3.72,11.22)	< 0.001***
FSH (mIU/mL)	6.19(4.93,7.7)	5.83(4.82,7.06)	0.1
LH/FSH	0.63(0.42,0.89)	1.18(0.67,1.86)	< 0.001***
E2(pmol/L)	160(116.75,208.75)	183(136.25,240)	< 0.001***
A2(nmol/L)	7.39(5.32,10.2)	12.7(8.79,16.8)	< 0.001***
T[b](nmol/L)	0.69(0.69,0.82)	1.06(0.69,1.52)	< 0.001***
TSH (mIU/L)	2.01(1.39,2.79)	2.1(1.5,2.91)	0.339
TG (mmol/L)	0.91(0.74,1.41)	1.29(0.87,1.87)	< 0.001***
HDL (mmol/L)	1.27(1.1,1.43)	1.21(1.06,1.42)	0.202
SAS	41.25(35,46.25)	43.75(38.75,50)	< 0.001***
SDS	43.75(36.25,50)	46.25(38.75,55)	< 0.001***
PSS	24(19,28.5)	25(20,30)	0.026*
Education			0.475
Primary school and below	4 (2.35%)	23 (3.97%)	
Middle school	67 (39.41%)	242 (41.72%)	
College and above	99 (58.24%)	315 (54.31%)	
Income	40,000 (20,000, 60,000)	40,000 (30,000, 60,000)	0.150
Birthplace			0.228
North China	45 (71.43%)	275 (63.66%)	
Non-North China	18 (28.57%)	157 (36.34%)	
Work			0.751
Mental work	24 (37.5%)	50 (35.21%)	
Physical work	40 (62.5%)	92 (64.79%)	

[a]Normally distributed continuous variables were expressed as mean ± SD, and abnormally distributed continuous variables were expressed as median (upper quartile, lower quartile). Categorical variables were expressed by the number of cases (percentage). [b] The lower detection limit for total testosterone was 0.69. BMI, body mass index; WHR, Waist-to-Hip Ratio; HOMA-IR, Homeostasis Model Assessment for Insulin Resistance ; HOMA-B, Homeostasis model assessment- β; PRL, prolactin; LH, luteinizing hormone; FSH, follicle-stimulating hormone; E2, estradiol; A2, androstenedione; T, testosterone; TSH, thyroid stimulating hormone; TG, triglyceride; HDL, high density lipoprotein; SAS, self-rating anxiety scale; SDS, self-rating depressive scale; PSS, perceived stress scale

Table 2 Comparison of the diet and physical activity between the PCOS group and control group

Characteristic[a]	PCOS (*N* = 601)	Control (*N* = 184)	*P*-value
Smoke			< 0.05
never	507(84.2%)	169(91.8%)	
occasional[b]	77(12.8%)	10(5.4%)	
regular	18(3%)	5(2.7%)	
Alcohol			< 0.001
never	356(59%)	125(67.9%)	
occasional	188(31.2%)	58(31.5%)	
regular	59(9.8%)	1(0.5%)	
Snack			0.233
never	46(7.6%)	16(8.7%)	
occasional	424(70.2%)	137(74.9%)	
regular	134(22.2%)	30(16.4%)	
Coffee			0.257
never	343(56.9%)	111(60.3%)	
occasional	247(41%)	66(35.9%)	
regular	13(2.2%)	7(3.8%)	
Milk			0.116
never	95(15.8%)	23(12.6%)	
occasional	382(63.3%)	109(59.6%)	
regular	126(20.9%)	51(27.9%)	
Dessert			0.109
never	48(8.1%)	7(3.8%)	
occasional	351(59.1%)	119(64.7%)	
regular	195(32.8%)	58(31.5%)	
Fried food			0.137
never	51(8.6%)	9(4.9%)	
occasional	431(72.6%)	145(79.2%)	
regular	112(18.9%)	29(15.8%)	

[a]Categorical variables were expressed as the number of cases (percentage)

[b]regular habits: at least one time per day, occasional habits: habits with frequency between "never" and "regular"

statistically significantly higher levels of HOMA.B and LH than the NS-PCOS group (both $P<0.05$). We also conducted an analysis of SHBG levels in a subset of our patients, reveals that a significant reduction in SHBG levels among smoking PCOS women compared to their non-smoking counterparts ($P<0.01$, Table S1)."

S-PCOS had more negative emotions than NS-PCOS

As shown in Fig. 2, significant differences between S-PCOS and NS-PCOS were observed in the SDS score ($P<0.05$). Although no significant difference was found between the groups in SAS and PSS scores, the S-PCOS group had a trend towards higher scores. We can tentatively conclude that the S-PCOS group had more negative emotions than the NS-PCOS group. Subsequent data analysis revealed that educational attainment and income level were independent factors affecting SDS scores. To further examine the impact of smoking within the PCOS cohort, we employed analysis of covariance (ANCOVA) with education and income level as covariates. The analysis demonstrated a significant correlation between smoking and negative emotional states in the PCOS group ($P<0.05$, Table S2).

Discussion

We sorted individuals based on their habits, grouping those with and without Polycystic Ovary Syndrome (PCOS). The analysis highlighted a stronger connection between PCOS and smoking, as well as alcohol intake, compared to habits like drinking milk, coffee, snacking, indulging in desserts, or eating fried foods ($P<0.5$ for smoking, $P<0.001$ for alcohol; whereas P-values for milk, coffee, snacks, desserts, and fried food were all over 0.05, showing no statistical significance). Previous studies didn't explore smoking alongside other daily habits concerning their link with PCOS. This underscores the necessity for deeper investigations into smoking's potential impact on individuals with PCOS, which holds practical significance.

Our subsequent research delved into the biochemical indicators and scale scores of PCOS women. Statistical analysis revealed significantly higher testosterone (T) levels in the S-PCOS group than in the NS-PCOS group, consistent with earlier studies [24]. Past research hinted at a possible association between nicotine and testosterone levels due to their role in metabolic pathways. Nicotine metabolism, mainly through cytochrome P450 [25], and the involvement of the cytochrome P450 family in liver testosterone biosynthesis [26] might contribute to the heightened testosterone levels observed in the N-PCOS group. Our findings indicate a correlation between smoking and a reduction in SHBG levels, consistent with the results of previous studies [15, 16].

Alongside increased testosterone levels, the S-PCOS group exhibited a notable rise in luteinizing hormone (LH) levels. Related research suggests that frequent blood sampling in PCOS women often reveals elevated average LH levels, accompanied by increased LH pulse frequency and amplitude [27]. This suggests a potential link where smoking might influence ovaries to produce more testosterone by altering LH secretion patterns. Furthermore, the progesterone-mediated negative feedback regulation of LH secretion in PCOS women could be affected, potentially contributing to heightened LH and increased testosterone. However, current research on smoking's impact on LH primarily focuses on males, calling for further investigation into how smoking affects female reproductive health through hormonal mechanisms.

Additionally, the S-PCOS group displayed more pronounced negative emotions. Statistical analysis unveiled a correlation between smoking and Self-Rating Depression Scale (SDS) scores among PCOS women ($P<0.05$). Though not statistically significant, the S-PCOS group leaned towards higher Self-Rating Anxiety Scale (SAS)

Table 3 Clinical and biochemical data from patients and controls divided according to smoking status

Variables	PCOS		Control	
	Smokers (*N* = 95)	Non-smokers (*N* = 506)	Smokers (*N* = 15)	Non-smokers (*N* = 169)
Age	29(26,32) ##	28(26,31)	32.07 ± 4.3	30.07 ± 4.54
BMI	25.7(22.05,27.95) #	25(21.5,28.5)	21.2(19.55,25)	23.4(21.1,27.1)
WHR	0.84(0.81,0.88)	0.84(0.79,0.88)	0.82(0.78,0.89)	0.81(0.79,0.86)
HOMA-IR	2.75(1.53,3.98)	2.4(1.61,3.65)	2.58(1.83,3.84)	2.3(1.47,3.58)
HOMA-B	187.56(127.4,255.82)*	144.63(100.67,220.63)	229.82(180.01,235.71)	136.14(75.8,222.76)
PRL	11.9(8.54,13.7)	11(8.39,14.9)	11.4(9.09,14.55)	10.8(8.63,16.23)
LH	8.85(6.57,11.8)*##	6.27(3.66,11.15)	4.5(2.15,5.5)	3.79(2.44,5.3)
FSH	6.15(5.44,7.58)	5.8(4.78,6.99)	6.5(6.01,6.8)	6.18(4.68,7.87)
LH/FSH	1.39(1.1,2.01)##	1.18(0.67,1.86)	0.71(0.36,0.88)	0.63(0.42,0.88)
E2	203.5(169.5,227.5)#	182(134,240)	137(97.7,206.25)	162(116.75,208.75)
A2	14.55(9.67,20.88)##	12.5(8.75,16.6)	7.11(4.16,9.96)	7.39(5.37,10.2)
T	1.3(0.87,1.77)*#	1.05(0.69,1.46)	0.72(0.69,1.21)	0.69(0.69,0.8)
TSH	2.03(1.39,2.44)	2.11(1.5,2.94)	1.88(1.21,2.94)	2.03(1.43,2.78)
TG	1.31(0.92,2.5)#	1.29(0.87,1.86)	0.81(0.6,0.89)	0.92(0.75,1.44)
HDL	1.19(1.05,1.4)	1.21(1.06,1.42)	1.4(1.37,1.56)	1.25(1.09,1.4)
SAS	45(40,51.88)#	43,75(38.75,50)	43.75(34.38,48.12)	41.25(35,46.25)
SDS	47.5(41.88,57.5)*	45(38.75,55)	46.58 ± 7.08	43.21 ± 9.41
PSS	26.08 ± 7.76	24.7 ± 7.51	24.07 ± 7.34	23.4 ± 7.28

Data presented as median (quartiles)

*$p < 0.05$ versus non-smokers within group. **$p < 0.001$ versus non-smokers within group

#$p = 0.05$ versus controls with same smoking status. ##$p < 0.001$ versus controls with same smoking controls

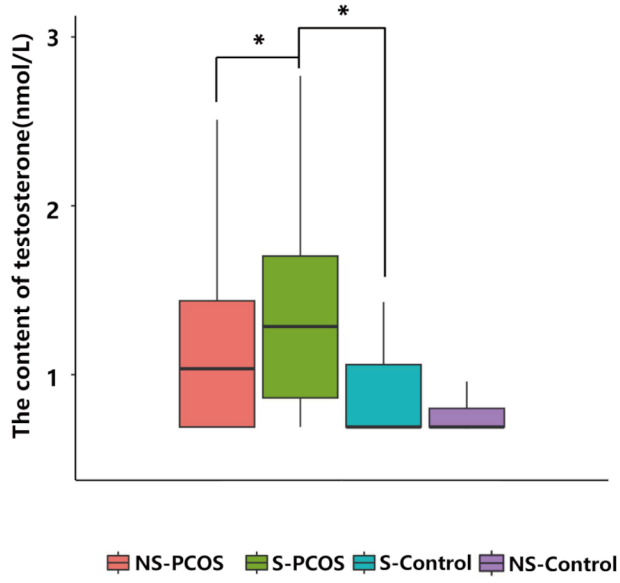

Fig. 1 Boxplots of total testosterone for S-PCOS, NS-PCOS, S-Control and NS-Control. These groups were compared by Wilcoxon rank sum test (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$)

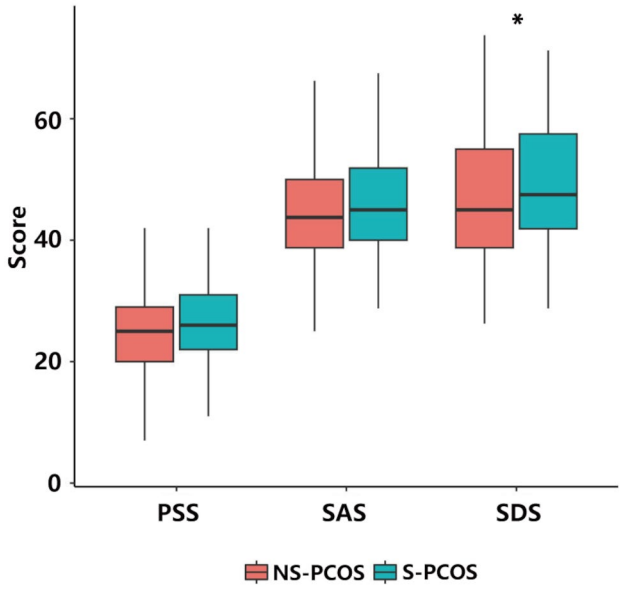

Fig. 2 Boxplots of emotional scales for S-PCOS and NS-PCOS. These groups were compared by Wilcoxon rank sum test (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$)

and Perceived Stress Scale (PSS) scores compared to the NS-PCOS group. This might be the first study suggesting that smoking exacerbates negative emotions in PCOS women. Prior research illustrated smoking's impact on emotional states across genders and various age groups, even transmitting these effects from pregnant women to their offspring [28]. Studies on female smokers revealed smoking's influence on endogenous hormone levels, including estrogen, commonly associated with female emotions [29]. These findings potentially explain smoking's exacerbation of negative emotions in PCOS women.

Smoking, a controllable unhealthy lifestyle, has garnered considerable attention in academic circles. Extensive research underscores significant negative impacts

of smoking and nicotine on female reproductive health. In an era of declining fertility rates, understanding the relationship between smoking and PCOS and its potential adverse effects becomes pivotal. Additionally, our research aims to offer guidance and aid for clinical practitioners and female patients. Highlighting smoking as a crucial detrimental lifestyle factor can significantly contribute to clinical work concerning PCOS.

We further analyze the correlation between dietary habits and emotions (Table S5). Our study results demonstrated a positive correlation between the consumption of desserts and the scores on the SAS, SDS, and PSS. Conversely, intake of coffee, milk, and fried foods was negatively correlated with these scores. However, none of these correlations reached statistical significance. Additionally, the data revealed a decrease in SAS, SDS, and PSS scores followed by an increase with escalating levels of alcohol consumption, consistent with findings from previous research [30] (Table S6). Furthermore, a significant negative correlation was observed between snack consumption and both SDS and PSS scores.

A primary limitation of our study is the lack of specific information on smoking duration and quantity among the patient group. Further research should investigate smoking's overall impact, duration, and quantity on high androgen female hormones and metabolic parameters. As a cross-sectional study, our hypothesis was that there would be a correlation between smoking, high androgen levels and negative mood. Further research is required to elucidate the mechanisms by which smoking may either precipitate or exacerbate the condition, particularly regarding its relationship with various metabolic disorders in PCOS.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13048-024-01541-x.

Supplementary Material 1

Author contributions

YY and HZ contributed equally to this work. HLZ, HZ and SL designed and organized the study, collected study objects, provided data management and wrote this manuscript. YY, BYH and YHL conducted the statistical analysis, took responsibility for the integrity and accuracy of this analysis, interpreted the data and drafted the manuscript. IF and LFZ were responsible for patient recruitment and data collection; CYW and ND provided scientific advice regarding the development of the intervention; LYL acquired the patient data and administered the treatments. HLZ revised the paper, supervised this project and contributed to the expert review and survey instrument. All authors read and approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Study protocol was approved by the Medical Science Research Ethics Committee of Peking University Third Hospital (No. 2016-212-02) and registered on ClinicalTrial.gov (NCT04264832, website: https://clinicaltrials.gov). All methods were carried out in accordance with relevant guidelines and regulations.

Patient consent statement

All participants provided written informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details

[1]Department of Traditional Chinese Medicine, Peking University Third Hospital, Beijing, China
[2]Center for Reproductive Medicine, Department of Obstetrics and Gynecology, Peking University Third Hospital, Beijing, China
[3]Department of Integration of Chinese and Western Medicine, Peking University Health Science Center, Beijing, China
[4]State Key Laboratory of Female Fertility Promotion, Center for Reproductive Medicine, Department of Obstetrics and Gynecology, Peking University Third Hospital, Beijing, China

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