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Influence of tobacco smoking and alcohol drinking on dysmenorrhoea: a cross-sectional analysis of data from the Taiwan Biobank

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ABSTRACT

Objectives Dysmenorrhoea, characterised by severe menstrual cramps, affects between 50% and 90% of women of reproductive age and considerably lowers their quality of life.

Design Cross-sectional study.

Setting This study explored the influence of tobacco smoking and alcohol consumption on dysmenorrhoea, using data from the Taiwan Biobank.

Participants Our analysis involved 8567 individuals and examined the associations between dysmenorrhoea and variables such as alcohol consumption, tobacco smoking, demographic characteristics and lifestyle behaviours through multivariable logistic regression.

Results The findings reveal a significant association between concurrent alcohol and tobacco use and increased dysmenorrhoea risk, with adjusted ORs suggesting higher risk levels for dual users compared with those who only smoke or drink (adjusted OR (95% CI) both alcohol and tobacco: 3.19 (1.51 to 6.72); only tobacco: 1.21 (0.89 to 1.63); only alcohol: 1.06 (0.53 to 2.13)). Additionally, factors such as higher education level and early menarche were associated with increased dysmenorrhoea risk, whereas regular exercise and multiparity exerted a protective effect against the condition (adjusted OR (95% CI) higher education: 1.43 (1.19 to 1.71); early menarche: 1.37 (1.14 to 1.64); regular exercise: 0.77 (0.64 to 0.93); multiparity: 0.64 (0.52 to 0.77)).

Conclusions Our study also highlights the complex interactions between lifestyle factors and dysmenorrhoea, underscoring the need for targeted interventions and lifestyle modifications to mitigate the condition's effects. Future research should employ longitudinal designs for causal inference and to explore the mechanisms underlying the associations observed in this study.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ Large sample size: Our study benefits from a substantial sample size of 8567 participants, which enhances the statistical power and reliability of the findings.
⇒ Comprehensive data source: The utilisation of the Taiwan Biobank, a well-established and comprehensive dataset, allows for the integration of detailed lifestyle, genetic and health information, providing a robust basis for analysis.
⇒ Multivariable analysis: By employing multivariable logistic regression, we could adjust for various demographic and lifestyle factors, thus minimising the potential for confounding and offering a clearer insight into the associations between substance use and dysmenorrhoea.
⇒ Cross-sectional design: Restricts ability to establish causality; only correlations can be inferred. This hinders understanding of causal pathways essential for developing effective interventions.
⇒ Self-reported data: Reliance on self-reported data for dysmenorrhoea symptoms and substance use may introduce bias due to under-reporting or over-reporting from social desirability or recall errors.

INTRODUCTION

Dysmenorrhoea, the medical term for painful menstruation, involves intense abdominal cramps and is often accompanied by headaches, nausea, vomiting and diarrhoea. This condition can be further classified into two types: primary and secondary dysmenorrhoea. Primary dysmenorrhoea (PD) involves cramping without an underlying pathological condition, whereas secondary dysmenorrhoea involves pelvic pathologies such as endometriosis.[1–3]

Dysmenorrhoea is the leading cause of menstrual distress and poses a critical public health challenge, particularly in less developed countries.[3 4] The condition, the severity and symptoms of which vary widely, affects an estimated 50–90% of women of reproductive age worldwide.[3] Younger women are disproportionately affected by dysmenorrhoea, which can substantially disrupt their education, work and social engagements. However, data in the Taiwan Biobank (TWB) only provide self-reports and are unable to differentiate between primary and secondary dysmenorrhoea. Therefore, the dysmenorrhoea discussed in our manuscript included

both primary and secondary dysmenorrhoea without the separation of primary and secondary ones.

Several genetic and environmental factors, including lifestyle choices such as smoking, alcohol use, diet and physical activity levels, in addition to genetic predispositions and gynaecological conditions, influence the pathophysiology of dysmenorrhoea.[4–6] Tobacco smoking exacerbates menstrual pain through mechanisms such as nicotine-induced vasoconstriction, reducing uterine blood flow and increasing the production of prostaglandins that cause uterine contractions and pain.[7–9] Evidence suggests that quitting smoking may alleviate these symptoms, although the precise mechanisms for this mitigation of symptoms remain poorly understood.[10–12] In addition to tobacco smoking, alcohol consumption may worsen menstrual cramps by affecting hormone levels, increasing inflammation and influencing sleep and hydration.[10–16] Beyond smoking and drinking, factors such as diet, exercise, stress and contraception use also influence the risk and severity of dysmenorrhoea. Although genetic predispositions to dysmenorrhoea cannot be altered, lifestyle choices can be modified to mitigate symptom severity.

In Taiwan, dysmenorrhoea poses considerable challenges to the health and everyday functioning of women. Data required on the associations between dysmenorrhoea and lifestyle choices such as smoking and drinking are provided in the Taiwan National Health and Nutrition Examination Survey (NAHSIT), a yearly survey of the nutrition and health status of the Taiwanese population.[17] The NAHSIT reveals that smoking and drinking rates for women have increased since Taiwan joined the World Trade Organisation in 2000, underscoring the importance of examining the relationship between lifestyle behaviours and menstrual pain. The present study examined the influence of tobacco smoking and alcohol consumption on the development and severity of dysmenorrhoea using data from the TWB. By identifying interactions between smoking and drinking and dysmenorrhoea, we explored the odds of dysmenorrhoea to suggest strategies to enhance management and prevention (figure 1).

METHODS

Taiwan Biobank

The TWB is a research initiative established by the government of Taiwan to create a comprehensive dataset of lifestyle and genetic information for the Taiwanese population. Participants provided written informed consent for the use of their data by the TWB. The TWB, which has 29 recruitment centres across Taiwan, provides a population-based dataset on individuals aged between 30 and 79 years with no previous cancer diagnosis. In 2023, the TWB achieved its recruitment goal and enrolled 200 000 healthy participants. The TWB includes data from epidemiological tests, blood samples and physical examinations. Each participant was also required to complete an in-depth structured questionnaire on an extensive range of demographic, epidemiological and lifestyle characteristics. The survey was supplemented by a face-to-face interview with researchers.

Dysmenorrhoea measurement

Dysmenorrhoea can be further classified into two types: primary and secondary dysmenorrhoea. PD involves cramping without an underlying pathological condition, whereas secondary dysmenorrhoea involves pelvic pathologies such as endometriosis.

Data on dysmenorrhoea of TWB were collected through a questionnaire distributed to premenopausal women. Participants were asked to document the specifics of their

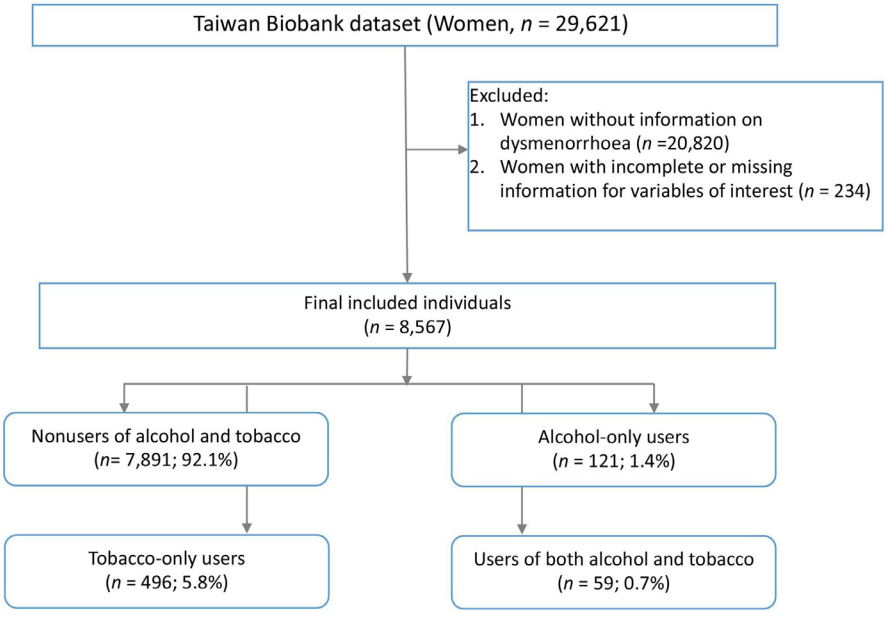

Figure 1 Flowchart of data gathering process among women participants in the Taiwan Biobank.

dysmenorrhoea experiences. The questionnaire in TWB is included in the online supplemental appendix.

Alcohol consumption and tobacco smoking

In this study, individuals were classified as regular alcohol drinkers if they consumed more than 150 cubic centimetres (cc) of alcohol per week for a minimum of 6 months.[18] Similarly, individuals were classified as regular smokers if they had been smoking consistently for at least 6 months.[19]

Covariates

Data on several covariates were collected from the TWB. These covariates included demographic and health characteristics, such as sex, age, body mass index (BMI) and educational level. Additionally, this study examined lifestyle and behavioural characteristics, such as regular exercise, coffee and tea consumption and dietary habits—including hormone consumption, vegetarianism and healthy eating statuses. Gynaecological health factors included dysmenorrhoea, ovarian and uterine problems and reproductive history (nulliparous, primiparous or multiparous). The age of menarche was recorded, and individuals were segmented into subgroups depending on whether the age of menarche occurred before or after 12 years. BMI was calculated using the standard formula: weight (in kilograms)/height^2 (in metres).

Statistical analysis

Individuals were segmented into four groups depending on alcohol consumption and tobacco use patterns: non-users, tobacco-only users, alcohol-only users or tobacco and alcohol users. Continuous variables were analysed using means and SD, whereas categorical variables were presented as percentages. A one-way analysis of variance was employed for continuous variables, and χ^2 or Fisher's exact tests were employed for categorical variables to compare variables across groups. Multivariable logistic regression was conducted to examine the adjusted ORs (AOR) for dysmenorrhoea risk associated with tobacco smoking and alcohol consumption.

Baseline demographic characteristics and lifestyle behaviours were controlled for in the regression analysis. Variables included for adjustment in the multivariable logistic model if p<0.05. All data transformations and statistical computations were performed using SAS for Windows V.9.4 (SAS Institute, Cary, North Carolina, USA). Statistical significance was determined at an alpha level of 0.05.

RESULTS

Baseline characteristics of TWB participants

We evaluated the baseline characteristics of 8567 participants stratified into the aforementioned four groups according to alcohol consumption and smoking statuses (table 1). The mean age differed substantially between groups, being highest in non-users of both alcohol and tobacco (51.3 years) and lowest in users of tobacco only (46.7 years; p<0.001). A significant difference was also observed in the percentage of participants younger than 50 years between the four groups, with those younger than 50 years significantly more likely to smoke but less likely to drink or to drink and smoke than those older than 50 years (p<0.001). Furthermore, educational attainment differed between the groups, with non-users of alcohol and tobacco more likely to have college or graduate education (43.6%; p=0.002). Moreover, behavioural factors such as regular exercise and coffee and tea consumption also differed significantly between the four groups, with concurrent users of tobacco and alcohol significantly less likely to consume tea and coffee or exercise regularly (p<0.001). Lifestyle choices such as vegetarianism also differed significantly between the groups, with non-users of tobacco and alcohol significantly more likely to be vegetarian than users of these substances (p=0.029). The prevalence of dysmenorrhoea also differed significantly between groups, with those who used tobacco significantly more likely to experience the condition (18.6% for users of both tobacco and alcohol, followed by 12.6% for users of tobacco only, 8.3% for users of alcohol only and 7.5% for users of neither; p<0.001). Furthermore, no significant difference was observed in the prevalence of ovarian problems between groups (p=0.125). By contrast, the mean age of menarche differed significantly between groups, with those who regularly used alcohol only more likely to have an older age of menarche (13.8 years), followed by users of both alcohol and tobacco (13.7 years), users of neither (13.6 years) and users of tobacco only (13.4 years; p=0.017). Additionally, the women surveyed differed significantly with respect to parity, with a higher percentage of nulliparous individuals in the non-user group (42.0%; p=0.015).

Associations between tobacco and alcohol use and dysmenorrhoea

Our analysis focused on the associations of alcohol and tobacco usage with dysmenorrhoea risk (table 2). After adjusting for potential confounders, the findings revealed a statistically significant association between dysmenorrhoea risk and the consumption of both alcohol and tobacco, with an AOR of 3.19 (95% CI 1.51 to 6.72) and a p value of 0.002, suggesting high dysmenorrhoea risk. No significant association was found for tobacco-only or alcohol-only users. The present study also revealed that education level was correlated with dysmenorrhoea risk, with those with college or graduate school education having an AOR of 1.43 (95% CI 1.19 to 1.71), indicating high risk. By contrast, regular exercise appeared to protect against dysmenorrhoea, indicated by a low AOR of 0.77 (95% CI 0.64 to 0.93). With respect to gynaecological problems, a significant association with dysmenorrhoea was observed in participants with uterine problems (AOR 1.50; 95% CI 1.23 to 1.82). By contrast, multiparity was associated with reduced dysmenorrhoea risk (AOR=0.64, 95% CI: 0.52 to 0.77).

Table 1 Baseline characteristics of participants of the Taiwan Biobank stratified by drinking and smoking behaviour (n=8567)

	Non-users of alcohol and tobacco (n=7891; 92.1%)	Tobacco-only users (n=496; 5.8%)	Alcohol-only users (n=121; 1.4%)	Users of alcohol and tobacco (n=59; 0.7%)	P value
Independent variable					
Age, mean (SD), years	51.3 (9.9)	46.7 (9.6)	50.9 (9.1)	48.8 (10.6)	<0.001***
< 50, n (%)	3271 (41.5)	306 (61.7)	52 (43.0)	27 (45.8)	<0.001***
≥50	4620 (58.5)	190 (38.3)	69 (57.0)	32 (54.2)	
Residential urbanicity, n (%)					
Rural	568 (7.2)	35 (7.1)	13 (10.7)	3 (5.1)	0.446
Non-rural	7323 (92.8)	461 (92.9)	108 (89.3)	56 (94.9)	
Education level, n (%)					
College or graduate school	3443 (43.6)	186 (37.5)	42 (34.7)	18 (30.5)	0.002**
High school, elementary school or less	4448 (56.4)	310 (62.5)	79 (65.3)	41 (69.5)	
BMI, mean (SD)	23.5 (3.4)	24.0 (4.0)	23.9 (3.2)	24.4 (4.8)	0.002**
< 25, n (%)	5650 (71.6)	330 (66.5)	82 (67.8)	39 (66.1)	0.062
≥25	2241 (28.4)	166 (33.5)	39 (32.2)	20 (33.9)	
Behavioural factor, n (%)					
Regular exercise	3789 (48.0)	159 (32.1)	62 (51.2)	22 (37.3)	<0.001***
Coffee consumption	3082 (39.1)	284 (57.3)	67 (55.4)	33 (55.9)	<0.001***
Tea consumption	1475 (18.7)	146 (29.4)	39 (32.2)	19 (32.2)	<0.001***
Gynaecological problem, n (%)					
Dysmenorrhoea	589 (7.5)	61 (12.3)	10 (8.3)	11 (18.6)	<0.001***
Ovary problem	396 (5.0)	31 (6.3)	11 (9.1)	2 (3.4)	0.125
Uterus problem	2089 (26.5)	142 (28.6)	36 (29.8)	13 (22.0)	0.502
Age of menarche, mean (SD), years	13.6 (1.5)	13.4 (1.4)	13.8 (1.6)	13.7 (1.7)	0.017*
< 12, n (%)	1604 (20.3)	118 (23.8)	23 (19.0)	13 (22.0)	0.298
≥12	6287 (79.7)	378 (76.2)	98 (81.0)	46 (78.0)	
Healthy product, n (%)					
Hormones included	1317 (16.7)	75 (15.1)	19 (15.7)	12 (20.3)	0.684
Hormones not included	6574 (83.3)	421 (84.9)	102 (84.3)	47 (79.7)	
Vegetarian, n (%)	463 (5.9)	16 (3.2)	3 (2.5)	2 (3.4)	0.029*
Parity, n (%)					
Nullipara	3311 (42.0)	234 (47.2)	52 (43.0)	32 (54.2)	0.015*
Primipara	1297 (16.4)	94 (19.0)	23 (19.0)	8 (13.6)	
Multipara	3283 (41.6)	168 (33.9)	46 (38.0)	19 (32.2)	

p<0.05*, p<0.01**, p<0.001*** (p value, χ^2 test or one-way analysis of variance).
BMI, body mass index.;

Additionally, early menarche (younger than 12 years of age) was associated with increased dysmenorrhoea risk (AOR=1.37; 95% CI 1.14 to 1.64). Finally, our analysis revealed a positive and marginally significant association between BMI<25 and dysmenorrhoea risk (AOR=0.79; 95% CI 0.65 to 0.97). Dysmenorrhoea risk was not significantly associated with coffee or tea consumption, ovarian problems, or the consumption of health products—including hormones.

Associations between tobacco and alcohol use and dysmenorrhoea between age groups

This study assessed the influence of alcohol and smoking on dysmenorrhoea risk in women below and above 50 years of age (table 3). The AOR for concurrent alcohol and tobacco users younger than 50 years was high and significant (AOR=2.57, 95% CI 1.13 to 5.83), indicating high dysmenorrhoea risk. The risk was even greater for those aged 50 and above (AOR=7.26, 95% CI 1.58 to

Table 2 Effects of drinking and smoking behaviour on the risk of dysmenorrhoea in participants of the Taiwan Biobank

Independent variables	Adjusted OR (95% CI) Dysmenorrhoea	P value
Alcohol and tobacco use (vs absence of alcohol and tobacco use)		
Tobacco-only users	1.21 (0.89 to 1.63)	0.221
Alcohol-only users	1.06 (0.53 to 2.13)	0.869
Users of both alcohol and tobacco	3.19 (1.51 to 6.72)	0.002**
Age (≥50 vs <50 years)	0.05 (0.04 to 0.07)	<0.001***
Residential urbanicity (rura vs non-rural)	0.99 (0.73 to 1.34)	0.953
Education level (vs high school, elementary school or lower level of education)		
College or graduate school	1.43 (1.19 to 1.71)	<0.001***
Baseline comorbidity		
BMI of≥25 (vs BMI of<25)	0.79 (0.65 to 0.97)	0.027*
Behavioural factor		
Regular exercise (vs absence of regular exercise)	0.77 (0.64 to 0.93)	0.007**
Coffee consumption (vs absence of coffee consumption)	1.09 (0.92 to 1.29)	0.339
Tea consumption (vs absence of tea consumption)	1.10 (0.90 to 1.35)	0.339
Gynaecological problem		
Ovary problem (vs No)	1.07 (0.72 to 1.59)	0.743
Uterus problem (vs No)	1.50 (1.23 to 1.82)	<0.001***
Age of menarche (vs ≥12 years)		
<12 years	1.37 (1.14 to 1.64)	<0.001***
Healthy product		
Hormones included (vs not included)	1.00 (0.75 to 1.32)	0.984
Vegetarian (vs no)	0.93 (0.63 to 1.36)	0.699
Parity (vs nullipara)		
Primipara	0.81 (0.64 to 1.01)	0.060
Multipara	0.64 (0.52 to 0.77)	<0.001***

p<0.05*, p<0.01**, p<0.001***.
BMI, body mass index.;

33.45). Higher educational levels were associated with increased dysmenorrhoea risk in women younger than 50 years (AOR=1.43, 95% CI 1.19 to 1.73), but this association was not significant in those older than 50 years. Regular exercise reduced dysmenorrhoea risk in those under 50 years (AOR=0.79, 95% CI 0.65 to 0.96), with no significant effect observed for those older than 50. The associations between age and gynaecological problems also differed by age. Uterine problems were significantly associated with dysmenorrhoea for those above and below 50 years old (AOR=1.40 for <50 y, 95% CI 1.14 to 1.72; AOR=2.31 for ≥50 y, 95% CI 1.27 to 4.22). Furthermore, being primiparous or multiparous reduced dysmenorrhoea risk in those under 50 years old (AOR for primiparous individuals=0.78, 95% CI 0.61 to 0.98; AOR for multiparous individuals=0.62, 95% CI 0.51 to 0.76). In terms of other lifestyle factors, tea consumption was associated with increased dysmenorrhoea risk only in those over 50 years old (OR=2.02, 95% CI 1.03 to 3.94). These findings demonstrate the strong associations between lifestyle factors, reproductive health and dysmenorrhoea risk. Additionally, associations of alcohol and tobacco use with dysmenorrhoea were more pronounced in older women. Findings on these associations are detailed in table 3.

DISCUSSION

The present study examined the associations between lifestyle factors, specifically alcohol and tobacco use, and dysmenorrhoea risk in Taiwanese women. Using data from the TWB on lifestyle, genetic and health information, this study investigated the influence of lifestyle choices on the prevalence and severity of dysmenorrhoea.

This finding is consistent with findings from other studies.[6 20] However, other studies on Australian women did not identify smoking as a significant danger factor for dysmenorrhoea, although they observed a slightly higher prevalence of dysmenorrhoea in young women who smoked.[14 20] However, although the associations

Table 3 Effects of drinking and smoking behaviour on the risk of dysmenorrhoea by age

Independent variables	Women aged<50 years adjusted OR (95% CI) Dysmenorrhoea	P value	Women aged≥50 years adjusted OR (95% CI) Dysmenorrhoea	P value
Alcohol and tobacco use (vs no alcohol and tobacco use)				
Tobacco-only users	1.24 (0.91 to 1.68)	0.173	0.55 (0.07 to 4.06)	0.556
Alcohol-only users	1.01 (0.49 to 2.12)	0.969	1.68 (0.22 to 12.70)	0.614
Users of both alcohol and tobacco	2.57 (1.13 to 5.83)	0.024*	7.26 (1.58 to 33.45)	0.011*
Residential urbanicity (rural vs non-rural)	1.01 (0.74 to 1.38)	0.945	0.88 (0.27 to 2.90)	0.831
Educational level (vs high school, elementary school or lower level of education)				
College or graduate School	1.43 (1.19 to 1.73)	<0.001***	1.49 (0.80 to 2.78)	0.209
Baseline comorbidity				
BMI (vs BMI<25)	0.81 (0.65 to 1.00)	0.055	0.62 (0.30 to 1.27)	0.191
Behavioural factors				
Regular exercise (vs no regular exercise)	0.79 (0.65 to 0.96)	0.019*	0.71 (0.39 to 1.31)	0.273
Coffee drinking habits (vs no coffee consumption)	1.12 (0.94 to 1.33)	0.216	0.72 (0.37 to 1.39)	0.326
Tea drinking habits (vs no tea consumption)	1.05 (0.85 to 1.29)	0.652	2.02 (1.03 to 3.94)	0.039*
Gynaecological problem				
Ovary problem (vs no)	1.06 (0.69 to 1.61)	0.795	1.28 (0.39 to 4.21)	0.689
Uterus problem (vs no)	1.40 (1.14 to 1.72)	0.001**	2.31 (1.27 to 4.22)	0.006**
Age of menarche (vs ≥12 years)				
<12 years	1.39 (1.15 to 1.68)	<0.001***	1.13 (0.52 to 2.47)	0.763
Healthy product				
Hormones included (vs not included)	1.21 (0.90 to 1.62)	0.208	0.15 (0.04 to 0.61)	0.009**
Vegetarian (vs no)	0.98 (0.66 to 1.46)	0.928	0.35 (0.05 to 2.60)	0.308
Parity (vs nullipara)				
Primipara	0.78 (0.61 to 0.98)	0.034*	1.44 (0.64 to 3.23)	0.380
Multipara	0.62 (0.51 to 0.76)	<0.001***	0.85 (0.43 to 1.70)	0.653

p<0.05*, p<0.01**, p<0.001***.
BMI, body mass index.;

between alcohol consumption and dysmenorrhoea have been extensively studied, the exact mechanisms behind these associations remain poorly understood. Such is not the case for smoking: the association between smoking and dysmenorrhoea is well understood and involves multiple physiological and psychosocial factors that can exacerbate menstrual pain. Physiologically, smoking can lead to vasoconstriction, reducing blood flow to the uterus and potentially increasing menstrual pain through heightened uterine muscle contractions and decreased oxygen delivery.[21] Additionally, smoking is associated with increased production of prostaglandins, which are linked to inflammation and pain and may worsen menstrual cramps.[21 22] Smoking may also disrupt the metabolism of oestrogen, leading to menstrual irregularities and increased pain.[9 23] From a psychological and sociocultural perspective, smoking as a stress-coping mechanism profoundly affects menstrual health; stress itself frequently amplifies menstrual discomfort. Lifestyle factors common among smokers, such as poor diet, limited exercise and alcohol consumption, can independently influence menstrual health and exacerbate pain.[4 24 25] Moreover, smokers' health awareness and behaviours can influence their pain perception and management approaches, influencing the severity of dysmenorrhoea experienced.[6 26]

Several factors may explain these associations. The influence of alcohol on women's hormonal balance is substantial, and this equilibrium is crucial for menstrual cycle regulation. Disturbance in women's hormonal levels due to alcohol may lead to increased menstrual pain. Moreover, alcohol stimulates the release of vasopressin, a hormone that narrows blood vessels, potentially increasing menstrual pain. Additionally, alcohol is a diuretic that may cause dehydration and potentially aggravate menstrual cramps. Moreover, alcohol decreases the body's level of magnesium, a substance that can alleviate muscle cramps. Moreover, alcohol enhances the body's inflammatory response and may

thus intensify dysmenorrhoea symptoms.[27 28] Furthermore, evidence suggests that alcohol may directly affect the uterine muscles, increasing the severity of menstrual cramps.[27 28] However, individual responses to alcohol vary widely, influenced by factors such as the amount of alcohol consumed, overall health and genetic predispositions. Therefore, although smoking or drinking individually may not substantially affect dysmenorrhoea severity, using both concurrently increases the risk of experiencing dysmenorrhoea.

The association of higher educational levels with increased dysmenorrhoea risk in younger women may be attributable to increased stress or lifestyle factors associated with stress at work or in school. However, the lack of a significant association between educational attainment and dysmenorrhoea in individuals older than 50 years may reflect a traditional lifestyle or less intense occupational stressors due to lower participation in the labour force. Notably, regular exercise exhibited a protective effect against dysmenorrhoea in younger women, underscoring the role of physical activity in managing menstrual symptoms. However, this protective effect was not observed in those over 50 years old, suggesting that the benefits of exercise on dysmenorrhoea may diminish with age or be overshadowed by other age-related factors. In addition to exercise, we observed that multiparity protected against dysmenorrhoea. Several theories have been proposed to explain this phenomenon. One theory focuses on the origin of dysmenorrhoea, which is typically associated with elevated prostaglandin levels in the secretory endometrium, a crucial element in pain introduction.[29–31] Following a full-term pregnancy, the endometrium may release lower prostaglandin levels, decreasing pain.[31] Another theory suggests that childbirth may result in neuronal degradation within the uterus[31 32] due to the loss of adrenergic nerves and reduced levels of norepinephrine during the final trimester of pregnancy, leading to the reduction or elimination of menstrual pain following childbirth.[31 32] In the present study, gynaecological problems, particularly those involving the uterus, were consistently associated with increased dysmenorrhoea risk across age groups. This finding suggests the presence of underlying pathophysiological changes in individuals with gynaecological problems that increase susceptibility to menstrual pain. The association between dysmenorrhoea and uterine disorders such as endometriosis, uterine fibroids, adenomyosis, pelvic inflammatory disease, cervical stenosis and congenital uterine anomalies may contribute to the onset and severity of menstrual cramps through mechanisms such as inflammation, obstruction of menstrual flow or enhanced uterine contractions.[33 34] If a woman has any of these conditions, management of dysmenorrhoea requires a comprehensive medical evaluation to determine the appropriate treatment based on the underlying disorder and its severity.[35–37] Finally, the association between early menarche (<12 years old) and dysmenorrhoea has been explored, although the findings are inconsistent.[38]

The present study also observed that individuals with an earlier onset of menarche reported more dysmenorrhoea symptoms. Similarly, a Kuwaiti study of high-school students linked early menarche to a higher incidence of dysmenorrhoea, potentially due to prolonged exposure to uterine prostaglandins.[39] Our results are consistent with those of other studies demonstrating that the occurrence of dysmenorrhoea increases with a lower age at menarche.[40–42] However, another study uncovered no association between a lower age of menarche and dysmenorrhoea.[43] Furthermore, the differing patterns of vegetarianism and parity we observed render a precise determination of the contribution of these factors to menstrual health difficult. In our study, vegetarianism was not found to be associated with dysmenorrhoea. By contrast, primiparous and nulliparous younger individuals exhibited decreased dysmenorrhoea risk. For those older than 50 years, only multiparity exerted a protective effect against dysmenorrhoea. These differences may be due to differences in hormonal balance, dietary choices and reproductive history between older and younger women. In conclusion, both lifestyle modifications and clinical factors must be addressed to reduce the dysmenorrhoea risk.

Limitations

This study has several limitations. First, the study's cross-sectional design precluded causal inference. Future studies should thus adopt a longitudinal design to determine causality and the mechanisms through which lifestyle factors influence dysmenorrhoea risk. Second, the absence of a detailed classification of dysmenorrhoea by type or severity limits our ability to draw specific conclusions on the basis of these factors. Because dysmenorrhoea can be primary (without underlying pathologies) or secondary (linked to reproductive system disorders), being unable to distinguish between these conditions can obscure the effects of tobacco and alcohol use on dysmenorrhoea severity. Third, our study did not differentiate between degrees of dysmenorrhoea severity, which can range from mild to severe; this limitation may have simplified the condition's effects on quality of life and distorted its correlations with lifestyle choices. Fourth, the lack of precise measurement of alcohol and tobacco consumption levels may have substantially influenced the study's outcomes due to variability in individual usage patterns and pain severity.

Our reliance on self-reported data for both dysmenorrhoea symptoms and substance use introduces the possibility of reporting bias. Reporting bias in the case of dysmenorrhoea may be especially pronounced because pain perception and tolerance vary greatly among individuals, potentially resulting in an underestimation or overestimation of dysmenorrhoea prevalence. Additionally, the study's cross-sectional design limits our ability to infer that lifestyle factors directly cause dysmenorrhoea. A final critical limitation is the study's exclusive focus on the Taiwanese population, which may restrict the applicability

of our findings to other populations with distinct genetic, cultural and environmental contexts.

Conclusions

This study found the complicated associations between alcohol and tobacco use and dysmenorrhoea. Neither smoking alone nor drinking alone had significantly higher odds of dysmenorrhoea, but that concurrent smoking and drinking substantially increased these odds of dysmenorrhoea. Moreover, there were complicated relations between lifestyle factors and dysmenorrhoea, underscoring the need for targeted interventions and lifestyle modifications to mitigate the condition's effects. Future research should employ longitudinal designs for causal inference and explore the mechanisms underlying the associations observed in this study.

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