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Sleep disturbances may influence lifestyle behaviours in women with self-reported polycystic ovary syndrome

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Abstract

Polycystic ovary syndrome (PCOS) is associated with a higher prevalence of sleep disturbances and obesity. Treatment of PCOS includes modifying lifestyle behaviours associated with weight management. However, poor sleep in the non-PCOS population has been associated with poorer lifestyle behaviours. The aim was to investigate whether sleep disturbance confounds or modifies the association between lifestyle factors and PCOS. This was a cross-sectional analysis from the Australian Longitudinal Study on Women's Health cohort aged 31–36 years in 2009 were analysed (*n* 6067, 464 PCOS, 5603 non-PCOS). Self-reported data were collected on PCOS, anthropometry, validated modified version of the Active Australia Physical Activity survey, validated FFQ and sleep disturbances through latent class analysis. Women with PCOS had greater adverse sleep symptoms including severe tiredness ($P = 0{\cdot}001$), difficulty sleeping ($P < 0{\cdot}001$) and restless sleep ($P < 0{\cdot}001$), compared with women without PCOS. Women with PCOS also had higher energy consumption (6911 (SD 2453) *v.* 6654 (SD 2215) kJ, $P = 0{\cdot}017$), fibre intake (19·8 (SD 7·8) *v.* 18·9 (SD 6·9) g, $P = 0{\cdot}012$) and diet quality (dietary guidelines index (DGI)) (88·1 (SD 11·6) *v.* 86·7 (SD 11·1), $P = 0{\cdot}008$), lower glycaemic index (50·2 (SD 4·0) *v.* 50·7 (SD 3·9), $P = 0{\cdot}021$) and increased sedentary behaviour (6·3 (SD 2·8) *v.* 5·9 (SD 2·8) h, $P = 0{\cdot}009$). There was a significant interaction between PCOS and sleep disturbances for DGI ($P = 0{\cdot}035$), therefore only for women who had adequate sleep was PCOS associated with a higher DGI. For women with poorer sleep, there was no association between PCOS and DGI. The association between PCOS and improved diet quality may only be maintained if women can obtain enough good quality sleep.

Key words: **Sleep: Polycystic ovary syndrome: Diet: Nutrition and physical activity**

Polycystic ovary syndrome (PCOS) is a common condition affecting between 5 and 18 % of reproductive-aged women[1,2]. It is characterised by clinical or biochemical hyperandrogenism, oligo-ovulation, anovulation and polycystic ovaries[3]. PCOS is associated with higher rates of infertility[4], pregnancy complications, type 2 diabetes[5], CVD[6] and psychological disorders such as depression and anxiety[7]. Insulin resistance is a key pathophysiological feature of PCOS. It is present in a form that is mechanistically distinct from obesity-associated insulin resistance[8] in the majority of women with PCOS, regardless of BMI[9], and worsens the clinical presentation of the syndrome. Excess weight will also worsen both insulin resistance and the adverse features associated with the syndrome[10]. In keeping with this, lifestyle (diet, exercise and/or behavioural) interventions for weight management (prevention of weight gain, modest weight loss or weight loss maintenance) are recommended as first-line treatment in PCOS[11].

Abbreviations: ALSWH, Australian Longitudinal Study on Women's Health; DGI, dietary guidelines index; LC, latent class; MET, metabolic equivalent values; PCOS, polycystic ovary syndrome.

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Women with PCOS have greater longitudinal weight gain, higher BMI and obesity prevalence than women without PCOS[12]. The reason for this is unclear. It is possible that the predisposition to weight gain in PCOS may be related to adverse lifestyle habits. The limited literature to date has reported both poorer lifestyle habits such as increased sedentary time[13], reduced physical activity[14] and higher energy intake[13] and positive lifestyle habits such as higher diet quality[15] and lower glycaemic index[13] in women with PCOS, compared with those without PCOS. Positive changes in lifestyle habits in women with PCOS may be a response to advice on improving lifestyle following diagnosis, consistent with International Evidence-Based Guidelines[11]. Conversely, prior to diagnosis adverse lifestyle habits in PCOS may reflect the contribution of poor diet, limited physical activity or sedentary behaviour to worsening weight and insulin resistance and making the features of PCOS more severe[10] and a diagnosis of PCOS more likely[12].

Women with PCOS also have a higher prevalence of sleep disturbances[16], including difficulty initiating sleep[16], restless sleep[17], severe daytime tiredness[17] and clinical sleep disorders such as obstructive sleep apnoea[18] and insomnia[16]. Epidemiological evidence in men and women suggests that reduced sleep duration and quality are associated with metabolic conditions such as obesity[19], diabetes[20] and CVD[21]. The mechanisms supporting these associations are not fully elucidated. However, previous research suggests short or disturbed sleep may increase hunger[22], decrease satiety[22], increase propensity to select foods high in carbohydrate[23] and fat[24] and reduce diet quality[25]. Furthermore, sleep disturbance is also associated with sedentary behaviour[26] and lower levels of physical activity[27]. Sleep disturbances in women with PCOS may therefore further exacerbate insulin resistance, worsen energy balance and worsen both the presentation of PCOS and metabolic consequences[28]. In the general population, interventions aimed at improving sleep have been shown to improve the success of weight loss in women[29] and improve the body's ability to reduce adipose stores[30]. Optimising sleep may, therefore, be important for women with PCOS to support and sustain healthy lifestyle changes. We have previously reported both positive (better diet quality) and negative (poorer sleep behaviours, higher energy intake and sedentary time) associations with lifestyle behaviours in women with PCOS, compared with those without PCOS[13,17]. However, whether sleep disturbances modify the association between PCOS and lifestyle is not currently known.

The aim of this study is to investigate whether sleep disturbance confounds or modifies the association between lifestyle factors and PCOS. The hypothesis was that in women with PCOS sleep would modify the association between PCOS and lifestyle behaviours.

Subjects and methods

Study population

This analysis used data from the Australian Longitudinal Study on Women's Health (ALSWH). The ALSWH is a national Australian study of women's social, mental and physical health. Initially, three age-based cohorts 18–23 years (born 1973–1978), 45–50 years (born 1946–1951) and 70–75 years (born 1921–1926) were recruited from the Medicare database in 1996. Women from rural areas were oversampled to allow comparison. The ALSWH cohorts have been followed up every 3–4 years since this time. This study is a cross-sectional analysis of the 1973–1978 cohort (survey 5) conducted in 2009. This survey was chosen because it contained both sleep and dietary information for women of reproductive age. Ethics approval was obtained from relevant Human Research Ethics Committees at the University of Newcastle and the University of Queensland. Details of the ALSWH have been published elsewhere[31,32].

Initially, the 1973–1978 cohort contained 14 247 women. In 2009, when survey 5 was conducted 13 223 women were still considered eligible for the survey with a response rate of 62·0 % (*n* 8200). Details of this sample are published elsewhere[33]. Participants were excluded from the analysis if (i) self-reported sleep data were biologically implausible (<3 h or >12 h), (ii) they had missing sleep data, (iii) dietary data indicated energy intake >14 700 kJ/d or <2100 kJ/d or there was incomplete FFQ data (>10 % of items with missing responses), leaving 6067 women included in the analysis. PCOS diagnosis was identified by the question 'In the last 3 years have you been diagnosed with or treated for PCOS?'. PCOS status was thereby identified by self-report of physician-diagnosed PCOS and has been validated against menstrual cycle irregularity, a key feature and diagnostic criterion for PCOS in previous published ALSWH studies[12].

Demographic variables

A range of demographic data were self-reported. Level of education was obtained through the question 'what is the highest qualification you have completed' with answers grouped into the following categories for analysis: no formal qualification/high school certificate, trade/diploma or degree. Income was obtained through the question 'What is the average gross (before tax) income that you receive each week, including pensions, allowances and financial support from your parents?' response categories were categorised into the following for analysis: 'no income', 'low income' ($AU1–36 399), 'medium income' ($AU36,400–77 999) and 'high income' (≥$AU78,000). Occupation was obtained by asking participants to mark a box which listed ten career categories including 'no paid work'. Responses were grouped into the following categories for analysis: professional (scientist, doctor, nurse, teacher, etc.), associated professional (technician, youth worker, police officer, etc.), clerical/trade (hairdresser, secretary, personal assistant, cleaner, sales assistant, etc.) and no paid work. Marital status was obtained through the question 'what is your current marital status', with the following responses available: married, *de facto*, separated/divorced, widowed and never married. Depression status was obtained through the Centre for Epidemiological Studies – Depression (CES-D 10) questionnaire[34]. A score ≥10 was used as the cut-off for depression[35]. Smoking status was determined with the question: 'How often do you currently smoke cigarettes or any tobacco products?', with possible answers being 'Daily', 'At least weekly', 'Less often than weekly' and 'Not at all'. Current non-smoking participants

were asked if they had smoked more than 100 cigarettes in their lifetime, with those who did being considered as ex-smokers. Participants were asked if they had previously given birth or were currently pregnant. Country of birth data was utilised from survey 1 in 1996; this question asked participants to choose one of the following options: Australia, UK, Italy, Greece, New Zealand, Vietnam or other (please specify).

Height, weight and waist circumference were self-reported. Height and weight were used to calculate BMI. The WHO BMI guidelines[36] were used to categorise BMI as underweight (BMI < 18·5 kg/m^2), healthy weight (BMI 18·5–24·9 kg/m^2), overweight (BMI 25–29·9 kg/m^2) or obese (≥30·0 kg/m^2). Waist circumference was also self-reported with instructions on how to measure including 'Find your navel (belly button) and measure at that level. Be careful not to have the tape too tight. You should be able to slip your little finger under it comfortably'. Self-reported measures of height, weight and waist circumference have been shown to be a valid and reliable method of measurement[37].

Lifestyle variables

Four areas of lifestyle were included in this analysis: diet, physical activity, sedentary time and sleep. Dietary intake was assessed using a 74-item validated FFQ, the Dietary Questionnaire for Epidemiological studies. This FFQ uses serving size information and frequency of consumption to capture the previous 12 months intake. Nutrient intakes were calculated using Australian Food Composition data[38]. This FFQ has been validated against a weighed food record in Australian young women[39]. Diet quality was measured using the dietary guidelines index (DGI)[40]. The DGI reflects compliance with the Dietary Guidelines for Australian adults and the Australian Guide to Healthy Eating and comprises dietary indicators for the consumption of five core food groups (vegetables, fruit, cereal, meat and alternatives and dairy). An additional category is calculated that is not part of the core food groups, entitled 'extras', which includes discretionary foods high in energy, added sugars, added salt, saturated fat or alcohol. Each component was scored on a scale of 0–10, with 10 indicating an optimal intake. The total score was the sum of thirteen3 indicators, with the DGI having a possible range of 0–130 and a higher score indicating greater compliance with the dietary guidelines. Details on the development of the DGI have been previously described[41].

Physical activity (planned and incidental) and sedentary time were self-reported via a validated modified version of the Active Australia Physical Activity survey[42]. Participants were asked to report the duration and frequency of the following leisure-time and transport activities in the last 7 d including walking briskly, moderate-intensity leisure activity and vigorous-intensity leisure activity lasting 10 min or longer. Metabolic equivalent values (MET) were assigned to each category (3·0, 4·0 and 7·5 MET, respectively). Total MET were calculated as minutes × respective MET scores (i.e. minutes walking × 3·0 + minutes vigorous activity × 7·5). Outliers were truncated at 28 h/week for total physical activity. Sedentary activity was reported on a weekday and usual weekend 'how many hours and minutes in total do you typically spend sitting down while doing things like visiting friends, driving, reading, watching television, or working at a desk or computer?'. Participants with data for total sitting time of greater than 16 h/d were not included in the analyses.

Sleep data were self-reported. Sleep quantity was ascertained by asking for sleep duration in hours and minutes on a workday and non-workday. Sleep quality was assessed with the following questions: (1) How often during the last week did you feel 'My sleep was restless' on a frequency scale: rarely or none of the time (<1 d), some or little of the time (1–2 d), occasionally or moderate amount of the time (3–4 d) or most or all of the time (5–7 d); (2) In the past month, 'Have you had difficulty falling asleep?', yes or no; (3) In the last 12 months have you had any of the following, 'Severe tiredness' or (4) 'Difficulty sleeping' on a frequency scale: never, rarely, sometimes or often. These were also classified into binary variables of 'rarely or none of the time/some or little of the time' *v.* 'occasionally or moderate amount of the time/most or all of the time' for 'my sleep was restless'; 'never/rarely' *v.* 'sometimes/often' for 'have you had difficulty sleeping' and 'severe tiredness'. As previously described for our cohort[17,43], the latent class (LC) analysis identified three mutually exclusive groups: (1) average sleep duration (mean values and standard deviation) (sleep duration of 456·0 (SD 53·8) min/d and 489·6 (SD 60)·1 min/d on a workday and non-workday, respectively), with little or no sleep disturbances, (2) average sleep duration (452·5 (SD 55·92) min/d and 504·0 (SD 64·44) min/d sleep on a workday and non-workday, respectively) and sometimes/often experiencing sleep disturbances including severe tiredness, difficulty falling asleep, restless sleep, difficulty falling asleep and (3) short sleep duration (342·2 (SD 51·5) min/d and 374·1 (SD 60·2) min/d sleep on a workday and non-workday, respectively) and sometimes/often experiencing sleep disturbances including severe tiredness, difficulty falling asleep, restless sleep and difficulty falling asleep. Women were classified into these classes according to their highest probability of class membership.

Statistical analysis

PCOS status was the exposure variable. The primary outcome measures were lifestyle behaviours including daily dietary intake energy (kJ), percentage energy: protein, carbohydrate, fat, saturated fat, monounsaturated fat, polyunsaturated fat, fibre (g), DGI, glycaemic index, glycaemic load and alcohol (g), daily physical activity (MET/mins) and sitting time (h/d) and daily sleep (quantity and adverse sleep-associated symptoms). The aim of the analyses was to assess whether sleep confounds or modifies the association between lifestyle factors and PCOS. The difference in lifestyle behaviours between women with and without PCOS was assessed via unpaired two-sided *t* test for continuous measures and χ^2 for categorical measures. The relationship between lifestyle behaviours and PCOS status was assessed using univariable and multivariable regression analyses. Univariable analysis included the lifestyle behaviours as the outcome and PCOS as the predictor (model 1). To investigate the potential role of sleep disturbance in these associations,

multivariable analyses were conducted for lifestyle behaviours adjusting for sleeping behaviour LC (model 2) and sleep behaviour, plus BMI, age, education level, income, occupation, marital status, parity, pregnancy, depression, smoking and country of birth (model 3). Confounders were selected on the basis of correlation ($P < 0{\cdot}2$) with PCOS and DGI, and on hypothesis testing based on postulated associations with lifestyle factors. Models were constructed to avoid collinearity and assessed for standard assumptions. Likelihood ratio tests were used for model evaluation and selection. The interactions between PCOS status and sleep behaviour LC were assessed in all the final models. Multivariable regression analyses were performed assessing the association between lifestyle behaviours as the dependent variable and PCOS status as the independent variable stratified for each of the sleeping behaviour LC. Significance for demographics, dietary and sleep outcomes was subject to a Bonferroni correction which were <0·004, <0·004 and <0·007, respectively. All other data were subject to a significance of <0·05. Sensitivity analyses were conducted in the DGI regression which adjusted for energy intake. However, the results of these analyses did not change the directionality or significance of the relationship and therefore the original regression is reported, in line with the other diet variables. Data analysis was performed in Stata V14 (Stata Corp).

Results

Demographics

The demographic characteristics of women with and without PCOS are reported in Table 1. Women with PCOS were slightly younger and on average had higher BMI, weight, waist circumference and depression as per the Centre for Epidemiological Studies – Depression and a lower proportion of women with PCOS had children. There were no differences in education level, household income, occupation, country of birth, pregnancy status, marital status or smoking status.

Lifestyle behaviours

The descriptive statistics for lifestyle behaviours of controls and women with PCOS are reported in Table 2. Women with PCOS had higher daily intake of energy, fibre, higher diet quality, lower glycaemic index and spent more time being sedentary (all *P*-values ≤ 0·021). As previously reported using this dataset, there were significant differences in the sleep behaviour categories for women with and without PCOS, with those with PCOS having a lower proportion of women in LC 1 (~8 h sleep/no adverse sleep-associated symptoms) and a higher proportion of women in LC2 (~8 h sleep/adverse sleep-associated symptoms) and LC3 (~6 h sleep/adverse sleep-associated symptoms) ($P < 0{\cdot}001$) This is supported by the individual sleep disturbance variables[17]. There was no difference between women with and without PCOS in the duration of sleep reported. However, a significantly higher proportion of women with PCOS reported adverse sleep symptoms (restless sleep ($P < 0{\cdot}001$), difficulty sleeping ($P < 0{\cdot}001$), severe tiredness ($P = 0{\cdot}001$)) and difficulty falling asleep ($P = 0{\cdot}001$)).

Regression analysis

Results of the multivariable regression analysis of lifestyle behaviours are reported in Table 3. When controlling for sleeping behaviour (LC) in isolation, all associations between PCOS status and lifestyle variables were maintained. When controlling for other potential confounders (BMI, age, education level, income, occupation, marital status, parity, pregnancy, depression, smoking and country of birth), the significant associations between PCOS status and energy intake and sedentary behaviour were attenuated and PCOS status was associated with alcohol intake (Table 3).

In the BMI and DGI models, there were significant interactions between PCOS and sleeping behaviour (Table 4). In stratified analysis, PCOS was associated with a higher BMI in all sleeping behaviour categories ($P < 0{\cdot}05$). The DGI analyses showed that only for women in LC1 (~8 h sleep/no adverse sleep symptoms) was PCOS associated with a higher DGI. There were no differences in diet quality between women with and without PCOS for those with poorer sleep (LC2 (~8 h sleep/adverse sleep-associated symptoms) and LC3 (~6 h sleep/adverse sleep-associated symptoms)).

Discussion

In this study, PCOS was associated with healthier (higher diet quality, fibre and lower GI and alcohol consumption) and negative (higher energy intake and sitting time) behaviours. This has been previously shown with the ALSWH cohort[13]. Further as previously shown, women with PCOS also had a higher BMI and higher prevalence of both obesity and poor sleeping behaviours including adverse sleep-associated symptoms (severe tiredness, difficulty sleeping, restless sleep and difficulty falling asleep)[17,44,45]. While these results have previously been presented, this study was able to examine the role of sleeping behaviour in supporting these relationships. The associations between energy intake, sedentary behaviour and PCOS were no longer present after adjustment for sleeping behaviour and other confounding variables. However, the positive association between PCOS and higher diet quality was only present in women with little to no sleep disturbances. The association between PCOS and BMI was also generally stronger when more severe sleep disturbances were present.

Our findings here build on our prior work reporting that women with a diagnosis of PCOS self-select marginally higher quality diets[10]. This may be a result of receiving information on optimising lifestyle following a diagnosis of PCOS, consistent with international evidence-based guidelines[11]. In addition, we showed that this positive association is strengthened on consideration of sleeping behaviour. Specifically, while women with PCOS had a 1·4-unit higher DGI when sleeping behaviour and other confounders were not controlled for, in women with no sleep disturbances the difference in DGI between women with PCOS and those without doubled to 3·1-units, with no difference observed in women with sleep disturbances. Of note, previous studies have shown a 10-unit greater DGI is associated with improved cardiometabolic risk factors[40]. Studies using similar indexes to the DGI have reported that changes in as little as

Table 1. Demographic characteristics of women with and without PCOS*
(Numbers and percentages; mean values and standard deviations)

	Control (*n* 5603)				Case (PCOS) (*n* 464)				
Characteristic	Mean	SD	%	*n*	Mean	SD	%	*n*	*P*
Age (years)	33·7	1·5			33·5	1·4			0·029
Weight (kg)	70·7	16·3			78·7	21·5			<0·001
BMI (kg/m^2)	25·6	5·7			28·6	7·3			<0·001
Waist circumference (cm)	85·8	13·8			91·5	17·1			<0·001
BMI									
Underweight			2·8	152			2·0	9	<0·001
Healthy			53·9	2930			37·7	169	
Overweight			25·4	1381			21·9	98	
Obese			17·9	976			38·4	172	
Education									0·657
No formal education/high school			18·8	1038			17·3	78	
Trade/diploma			26·0	1435			25·5	115	
Degree			55·2	3049			57·2	258	
Annual household income									0·310
No income			6·5	342			7·0	30	
Low (>$0–$36 399)			38·8	2045			38·3	165	
Medium ($36 400–$77 999)			38·6	2035			38·4	2188	
High (>$78 000)			16·1	849			19·3	83	
Occupation									0·959
Professional			47·6	2635			48·9	223	
Associate professional			19·8	1097			19·1	87	
Clerical work or trade			18·4	1019			18·2	83	
No paid work			14·2	783			13·8	63	
Marital status									0·625
Married or de facto			75·9	4240			75·2	348	
Separated/divorced/widowed			5·5	306			4·8	22	
Never married			18·6	1040			20·1	93	
Has children			59·4	3330			54·7	254	0·048
Currently pregnant			9·7	538			11·8	54	0·153
Depression (CESD)			22·4	1253			29·7	138	0·006
Current smoker			14·1	789			13·2	61	0·580
Born in Oceania			94·7	5268			92·8	428	0·102

PCOS, polycystic ovary syndrome; CESD, Centre for Epidemiologic Studies Depression scale.
* Data were analysed using unpaired two-sided *t* test for continuous measures and χ^2 for categorical measures. Data are presented as mean and standard deviation for continuous and percentage and number (*n*) for categorical data.

one point are associated with a 1·4 % decrease in the risk of abdominal obesity or a decrease in 0·053–0·095 kg/m^2 BMI[46]. However, increased diet quality has been reported to be a significant predictor of weight status and BMI and therefore while it is a small difference, it may have significant clinical implications for women with PCOS[46].

It is unclear whether the 3-unit increment in DGI observed here is clinically significant. Previous research in the general population reported that reduced sleep quantity and quality are risk factors for lower quality diets (higher carbohydrate and fat diets)[22]. Experimental evidence suggests that after healthy sleep patterns have been restored, energy intake is decreased, particularly from carbohydrates and fat, which results in modest weight loss[23]. Short sleep duration and poor quality sleep may impact dietary intake in three ways (i) environmentally[22], (ii) endocrinologically and (iii) hedonically[47]. Environmentally, if a person is awake longer, there are more opportunities to eat. Endocrinologically, short sleep may increase ghrelin and decrease leptin, increasing hunger and decreasing signals for satiety[22]. Finally, hedonically, lack of sleep decreases dietary restraint and increases the drive for foods that are high in fat[24] and carbohydrates[23]. These potential mechanisms associated with reduced sleep duration and/or quality may reduce the ability of a woman with PCOS to make or sustain positive dietary change in her diet. Overall, this would increase the risk of positive energy balance and therefore weight gain which can then significantly worsen the severity of the features of PCOS[10]. In keeping with this, we also report here that the higher BMI for women with PCOS compared with those without PCOS was greater for those with poorer sleep (8 h sleep and disturbances). While we did not observe this for the most severe category of sleep disturbances (6 h sleep and sleep disturbances), this may be related to the smaller sample size and reduced power in this group.

We also confirm prior reports that women with PCOS had higher energy intake and sitting time compared with women without PCOS[13]. However, the relationship between these outcomes was removed when controlling for both sleeping behaviour and other demographic factors. This suggests that the effect of adverse sleeping behaviour in isolation is unlikely to alter these behaviours in PCOS and is not an important confounder. This is in contrast to findings in the general population, suggesting that higher energy intake[23] and lower physical activity[48] are associated with reduced sleep quality and quantity. Sedentary behaviour is proposed to have two

Table 2. Lifestyle characteristics in women with and without PCOS (Numbers and percentages; mean values and standard deviations)

	Control				PCOS				
Lifestyle characteristics	Mean	SD	%	*n*	Mean	SD	%	*n*	*P*
Dietary intake									
Energy (kJ)	6654	2215			6911	2453			0·017
Protein (%E)	21·0	3·3			21·2	3·4			0·182
Carbohydrate (%E)	40·4	5·7			40·3	6·0			0·755
Fibre (g)	18·9	6·9			19·8	7·8			0·012
Fat (%E)	36·9	4·9			36·7	5·3			0·565
Saturated fat (%E)	15·4	3·1			15·2	3·2			0·191
Monounsaturated fat (%E)	13·1	2·1			13·12	2·3			0·761
Polyunsaturated fat (%E)	5·1	1·7			5·1	1·6			0·749
DGI	86·7	11·1			88·1	11·6			0·008
Alcohol (g)	9·8	13·7			8·8	14·0			0·135
Glycaemic index	50·7	3·9			50·2	4·0			0·021
Glycaemic load	85·6	32·8			87·7	34·9			0·186
Physical activity									
Total sitting time (h)	5·9	2·8			6·3	2·8			0·009
MET/min/week	826·8	896·7			767·1	811·5			0·175
Sleeping behavior									
Latent class 1*			54·0	3022			42·5	197	<0·001
Latent class 2†			33·5	1875			40·3	187	
Latent class 3‡			12·6	705			17·2	80	
Sleep length									
Total sleep time on week day (min)	440	66			435	72			0·118
Total sleep time on weekend (min)	479	74			484	77			0·162
Sleep disturbances									
Occasionally/most of the time experiencing restless sleep			29·8	1657			37·6	174	<0·001
Sometimes/often difficulty sleeping			34·3	1893			43·9	200	<0·001
Sometime/often severe tiredness			48·6	2684			56·9	261	0·001
Difficulty falling asleep (yes)			39·8	2222			47·7	221	0·001

DGI, dietary guidelines index; PCOS, polycystic ovary syndrome.
Data were analysed using unpaired two-sided *t* test for continuous measures and χ^2 for categorical measures. Data are presented as mean and standard deviation for continuous and percentage and number (*n*) for categorical data.
Latent class analysis: *Class membership defined by: average sleep (¯8 h/d) with no adverse sleep-related symptoms, †Class membership defined by: average sleep (¯8 h/d) and sometimes/often experiencing sleep disturbances, ‡Class membership defined by: short sleep (¯6 h/d) with sometimes/often experiencing sleep disturbances.

Table 3. Multivariable linear regression of lifestyle behaviours and PCOS status (β-coefficients and 95 % confidence intervals)

	Model 1			Model 2			Model 3		
Lifestyle factors	β-coefficient	95 % CI	*P*	β-coefficient	95 % CI	*P*	β-coefficient	95 % CI	*P*
Energy intake (kJ)	257·0	45·5, 468·6	0·017	247·8	35·8, 459·8	0·022	207·2	−18·1, 432·5	0·071
Protein (%E)	0·22	−0·10, 0·53	0·182	0·21	−0·11, 0·53	0·194	−0·04	−0·37, 0·30	0·841
Carbohydrate (%E)	−0·09	−0·62, 0·45	0·755	−0·06	−0·60, 0·48	0·833	0·10	−0·47, 0·68	0·723
Fat (%E)	−0·14	−0·49, 0·10	0·565	−0·16	−0·63, 0·31	0·496	−0·89	−0·60, 0·42	0·732
Saturated fat (%E)	−0·20	−0·48, 0·10	0·191	−0·21	−0·51, 0·08	0·160	−0·16	−0·47, 0·16	0·322
Monounsaturated fat (%E)	0·03	−0·17, 0·23	0·761	0·02	−0·18, 0·22	0·844	−0·00	−0·21, 0·21	0·993
Polyunsaturated fat (%E)	0·03	−0·13, 0·18	0·749	0·03	−0·13, 0·18	0·740	0·08	−0·09, 0·25	0·374
Alcohol (g)	−0·99	−2·29, 0·31	0·135	−1·1	−2·4, 0·24	0·108	−1·5	−2·9, −0·19	0·026
Glycaemic Index	−0·44	−0·81, −0·07	0·021	−0·46	−0·83, −0·09	0·016	−0·43	−0·82, −0·05	0·029
Glycaemic Load	2·10	−1·01, 5·22	0·186	1·97	−1·15, 5·09	0·217	1·67	−1·66, 4·99	0·325
Fibre (g)	0·85	0·19, 1·5	0·012	0·86	0·20, 1·52	0·011	0·92	0·21, 1·63	0·012
DGI	1·42	0·36, 2·47	0·008	1·54	0·48, 2·60	0·004	1·48	0·36, 2·59	0·009
Physical activity (MET/min/week)	−59·7	−145·9, 26·5	0·175	−50·6	−136·9, 35·7	0·250	−49·1	−134·0, 41·7	0·289
Total sitting time (h)	0·37	0·09, 0·64	0·009	0·35	0·08, 0·62	0·012	0·17	−0·01, 0·45	0·212

DGI, dietary guidelines index; PCOS, polycystic ovary syndrome.
The relationship between lifestyle behaviours and PCOS status (0 = control and 1 = PCOS) was assessed using univariable and multivariable regression analyses. β-coefficient = the difference in the absolute value of the parameter between those with and without PCOS.
Univariable analysis included the lifestyle behaviours as the outcome and PCOS as the predictor (crude model/model).
Multivariable analyses were conducted for lifestyle behaviours and model (1) crude unadjusted, model, (2) sleeping behaviour (latent class) and model (3) sleeping behavior (latent class), BMI, age, education level, income, occupation, marital status, parity, pregnancy, depression, smoking and country of birth.

Table 4. Interaction analysis stratified by sleeping behaviour class (β-coefficients and 95 % confidence intervals)

		DGI			BMI (kg/m^2)		
	PCOS status	β-coefficient	95 % CI	*P*	β-coefficient	95 % CI	*P*
LC1 (*n* 3220)	Non-PCOS (*n* 3023)		Ref			Ref	
	PCOS (*n* 197)	3·14	1·46, 4·82	$P < 0·001$	2·70	1·89, 3·52	$P < 0·001$
LC2 (*n* 2062)	Non-PCOS (*n* 1875)		Ref			Ref	
	PCOS (*n* 187)	0·66	−1·10, 2·41	$P = 0·463$	4·20	3·27, 5·13	$P < 0·001$
LC3 (*n* 785)	Non-PCOS (*n* 705)		Ref			Ref	
	PCOS (*n* 80)	−0·73	−3·62, 2·16	$P = 0·622$	1·87	0·19, 3·55	$P = 0·030$
Interaction *P*-value			$P = 0·035$			$P = 0·010$	

DGI, dietary guidelines index; LC, latent class; PCOS, polycystic ovary syndrome.
LC1: average sleep (˜8 h/d) with no adverse sleep-related symptoms, LC2: average sleep (˜8 h/d) and sometimes/often experiencing sleep disturbance, LC3: short sleep (˜6 h/d) with sometimes/often experiencing sleep disturbance. β-coefficient = the difference in the absolute value of the parameter between those with and without PCOS.

mechanisms that influence sleep in the non-PCOS population. Firstly, sedentary behaviour is often associated with screen time[49]. Screen time increases blue-light exposure, which influences sleep quantity and quality[50]. Secondly, poor sleep increases factors such as fatigue, low mood and poor vigilance, decreasing the likelihood of physical activity and increasing the likelihood of sedentary behaviour[51]. From our analysis, these relationships do not seem to be present in women with PCOS and future studies are needed to confirm and elucidate why these relationships may not exist in PCOS.

Multidisciplinary (diet, physical activity and behavioural therapy) lifestyle interventions for weight management (weight gain prevention, modest weight loss and weight loss maintenance) are recommended in international evidence-based guidelines for PCOS[11]. Our results support that further research is needed assessing if the screening and management of short sleep duration or poor sleep quality sleep can improve lifestyle intervention success in women with PCOS. In individuals without PCOS, adequate sleep has been shown to assist in making positive lifestyle changes. Specifically, in a cohort of women participating in a weight loss trial, adequate sleep quality and longer sleep duration (> 7 h per night) increased the likelihood of intervention success by 33 %[29]. Reduced sleep (<5·5 h) has also been reported to reduce the effectiveness of weight loss interventions through physiologically decreasing the ability of the body to reduce adipose stores[30]. The mechanisms behind this relationship are not well understood, but are believed to be related to the alteration in leptin and ghrelin secretion[30]. Ensuring women with PCOS are obtaining adequate sleep may be an important factor in supporting positive lifestyle change with important impacts on weight management and severity of symptoms experienced.

Due to the cross-sectional nature of this study, causality cannot be inferred. However, this study is strengthened by the large and representative population-based cohort. The prevalence of PCOS in this sample is similar to that reported in the wider Australian population (8 % *v.* 5–18 %)[1,2]. A limitation of this study is the self-report of physician-diagnosed PCOS, with no information on the diagnostic criteria used or PCOS phenotype. However, self-reported PCOS has been validated in this cohort against menstrual irregularity, which is a key feature and diagnostic criteria for PCOS[52,53]. Indeed, genome-wide association studies in PCOS seeking to identify candidate genes in PCOS have found that despite the diagnostic criterion used for PCOS, whether it is Rotterdam, NIH and also self-reported, women with PCOS share a similar genetic architecture[54]. Further, women with PCOS from clinical populations have a higher prevalence of obesity than those from unselected populations[52], this allows for the inclusion of varying degrees of obesity and increases the representativeness of the data. We note the self-reported nature of the data as a limitation. However, given the sample size, it would have been impractical to collect objective sleep and activity data from these participants. Furthermore, although the sleep questions were not validated, the food frequency and physical activity questionnaires have been validated in a sample of young women[39,42]. Due to the self-reported nature, true PCOS prevalence in the sample may have been under-estimated. However, previous literature reports that in this cohort self-reported PCOS correlates strongly with menstrual irregularity as a diagnostic marker of PCOS status[12]. Furthermore, the measures stated in the PCOS international recommendations would not have been feasible given this study sample a total of 14 247 women for this cohort from across Australia, including rural and remote locations[11].

Conclusion

The results of this study suggest that the association between PCOS and improved diet quality may only be maintained if the women are getting adequate quantity and minimal adverse sleep-related symptoms. Therefore, while women with PCOS may be able to self-select a higher quality diet following diagnosis, which could potentially improve their condition consistent with evidence-based guidelines, their ability to do this may be reduced without adequate sleep quality and quantity.

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References

1. March WA, Moore VM, Willson KJ, *et al.* (2010) The prevalence of polycystic ovary syndrome in a community sample assessed under contrasting diagnostic criteria. *Hum Reprod* **25**, 544–551.
2. Azziz R, Woods KS, Reyna R, *et al.* (2004) The prevalence and features of the polycystic ovary syndrome in an unselected population. *J Clin Endocrinol Metab* **89**, 2745–2749.
3. Fauser BC, Tarlatzis BC, Rebar RW, *et al.* (2012) Consensus on women's health aspects of polycystic ovary syndrome (PCOS): the Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. *Fertil Steril* **97**, 28–38. e25.
4. Teede H, Deeks A & Moran L (2010) Polycystic ovary syndrome: a complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. *BMC Med* **8**, 41.
5. Moran LJ, Misso ML, Wild RA, *et al.* (2010) Impaired glucose tolerance, type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: a systematic review and meta-analysis. *Hum Reprod Update* **16**, 347–363.
6. Zhao L, Zhu Z, Lou H, *et al.* (2016) Polycystic ovary syndrome (PCOS) and the risk of coronary heart disease (CHD): a meta-analysis. *Oncotarget* **7**, 33715–33721.
7. Deeks AA, Gibson-Helm ME & Teede HJ (2010) Anxiety and depression in polycystic ovary syndrome: a comprehensive investigation. *Fertil Steril* **93**, 2421–2423.
8. Diamanti-Kandarakis E & Dunaif A (2012) Insulin resistance and the polycystic ovary syndrome revisited: an update on mechanisms and implications. *Endocr Rev* **33**, 981–1030.
9. Stepto NK, Cassar S, Joham AE, *et al.* (2013) Women with polycystic ovary syndrome have intrinsic insulin resistance on euglycaemic–hyperinsulaemic clamp. *Hum Reprod* **28**, 777–784.
10. Lim S, Norman RJ, Davies M, *et al.* (2013) The effect of obesity on polycystic ovary syndrome: a systematic review and meta-analysis. *Obes Rev* **14**, 95–109.
11. Teede HJ, Misso ML, Costello MF, *et al.* (2018) Recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome. *Hum Reprod* **33**, 1602–1618.
12. Teede HJ, Joham AE, Paul E, *et al.* (2013) Longitudinal weight gain in women identified with polycystic ovary syndrome: results of an observational study in young women. *Obesity* **21**, 1526–1532.
13. Moran LJ, Ranasinha S, Zoungas S, *et al.* (2013) The contribution of diet, physical activity and sedentary behaviour to body mass index in women with and without polycystic ovary syndrome. *Hum Reprod* **28**, 2276–2283.
14. Eleftheriadou M, Michala L, Stefanidis K, *et al.* (2012) Exercise and sedentary habits among adolescents with PCOS. *J Pediatr Adol Gynec* **25**, 172–174.
15. Moran L, Grieger J, Mishra G, *et al.* (2015) The association of a Mediterranean-style diet pattern with polycystic ovary syndrome status in a community cohort study. *Nutrients* **7**, 8553–8564.
16. Fernandez RC, Moore VM, Van Ryswyk EM, *et al.* (2018) Sleep disturbances in women with polycystic ovary syndrome: prevalence, pathophysiology, impact and management strategies. *Nat Sci Sleep* **10**, 45.
17. Mo L, Mansfield DR, Joham A, *et al.* (2019) Sleep disturbances in women with and without polycystic ovary syndrome in an Australian National cohort. *Clin Endocrinol* **90**, 570–578.
18. Thannickal A, Brutocao C, Alsawas M, *et al.* (2020) Eating, sleeping and sexual function disorders in women with polycystic ovary syndrome (PCOS): a systematic review and meta-analysis. *Clin Endocrinol* **92**, 338–349.
19. Cappuccio FP, Taggart FM, Kandala NB, *et al.* (2008) Meta-analysis of short sleep duration and obesity in children and adults. *Sleep* **31**, 619–626.
20. Cappuccio FP, D'elia L, Strazzullo P, *et al.* (2010) Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. *Diabetes Care* **33**, 414–420.
21. Cappuccio FP, Cooper D, D'elia L, *et al.* (2011) Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *Eur Heart J* **32**, 1484–1492.
22. Chaput JP (2014) Sleep patterns, diet quality and energy balance. *Physiol Behav* **134**, 86–91.
23. Markwald RR, Melanson EL, Smith MR, *et al.* (2013) Impact of insufficient sleep on total daily energy expenditure, food intake, and weight gain. *PNAS* **110**, 5695–5700.
24. Spaeth AM, Dinges DF & Goel N (2013) Effects of experimental sleep restriction on weight gain, caloric intake, and meal timing in healthy adults. *Sleep* **36**, 981–990.
25. Haghighatdoost F, Karimi G, Esmaillzadeh A, *et al.* (2012) Sleep deprivation is associated with lower diet quality indices and higher rate of general and central obesity among young female students in Iran. *Nutrition* **28**, 1146–1150.
26. Yang Y, Shin JC, Li D, *et al.* (2017) Sedentary behavior and sleep problems: a systematic review and meta-analysis. *Int J Behav Med* **24**, 481–492.
27. Soltani M, Haytabakhsh MR, Najman JM, *et al.* (2012) Sleepless nights: the effect of socioeconomic status, physical activity, and lifestyle factors on sleep quality in a large cohort of Australian women. *Arch Womens Ment Health* **15**, 237–247.
28. Tasali E, Van Cauter E, Hoffman L, *et al.* (2008) Impact of obstructive sleep apnea on insulin resistance and glucose tolerance in women with polycystic ovary syndrome. *J Clin Endocrinol Metab* **93**, 3878–3884.
29. Thomson CA, Morrow KL, Flatt SW, *et al.* (2012) Relationship between sleep quality and quantity and weight loss in women participating in a weight-loss intervention trial. *Obesity* **20**, 1419–1425.
30. Nedeltcheva AV, Kilkus JM, Imperial J, *et al.* (2010) Insufficient sleep undermines dietary efforts to reduce adiposity. *Ann Intern Med* **153**, 435–441.
31. Lee C, Dobson AJ, Brown WJ, *et al.* (2005) Cohort profile: the Australian longitudinal study on women's health. *Int J Epidemiol* **34**, 987–991.
32. Brown W, Bryson L, Byles J, *et al.* (1996) Women's health Australia: establishment of the Australian longitudinal study on women's health. *J Am Diet Assoc* **5**, 467–472.
33. Dobson AJ, Hockey R, Brown WJ, *et al.* (2015) Cohort profile update: Australian longitudinal study on women's health. *Int J Epidemiol* **44**, 1547f.
34. France C, Lee C & Powers J (2004) Correlates of depressive symptoms in a representative sample of young Australian women. *Aust Psychol* **39**, 228–237.
35. Andresen EM, Malmgren JA, Carter WB, *et al.* (1994) Screening for depression in well older adults: evaluation of a short form of

the CES-D (Center for Epidemiologic Studies Depression Scale). *Am J Prev Med* **10**, 77–84.
36. World Health Organisation Body mass index – BMI (2020) http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-index-bmi (accessed May 2020).
37. Tuomela J, Kaprio J, Sipilä PN, *et al.* (2019) Accuracy of self-reported anthropometric measures – Findings from the Finnish Twin Study. *Obes Res Clin Practice* **13**, 522–528.
38. Lewis J & Milligan GC (1995) *NUTTAB95: Nutrient Database for Use in Australia*. Canberra: Food Standards Australia New Zealand.
39. Hodge A, Patterson AJ, Brown WJ, *et al.* (2000) The Anti Cancer Council of Victoria FFQ: relative validity of nutrient intakes compared with weighed food records in young to middle-aged women in a study of iron supplementation. *Aust N Z J Public Health* **24**, 576–583.
40. Haines PS, Siega-Riz AM & Popkin BM (1999) The diet quality index revised: a measurement instrument for populations. *J Am Dietetic Association* **99**, 697–704.
41. McNaughton SA, Dunstan DW, Ball K, *et al.* (2009) Dietary quality is associated with diabetes and cardio-metabolic risk factors. *J Nutr* **139**, 734–742.
42. Brown WJ, Burton NW, Marshall AL, *et al.* (2008) Reliability and validity of a modified self-administered version of the Active Australia physical activity survey in a sample of mid-age women. *Aust N Z J Public Health* **32**, 535–541.
43. Bennett CJ, Truby H, Zia Z, *et al.* (2017) Investigating the relationship between sleep and macronutrient intake in women of childbearing age. *Eur J Clin Nutr* **71**, 712–717.
44. Tasali E, Van Cauter E & Ehrmann DA (2006) Relationships between sleep disordered breathing and glucose metabolism in polycystic ovary syndrome. *J Clin Endocrinol Metab* **91**, 36–42.
45. Moran LJ, March WA, Whitrow MJ, *et al.* (2015) Sleep disturbances in a community-based sample of women with polycystic ovary syndrome. *Hum Reprod* **30**, 466–472.
46. Asghari G, Mirmiran P, Yuzbashian E, *et al.* (2017) A systematic review of diet quality indices in relation to obesity. *Br J Nutr* **117**, 1055–1065.
47. Blumfield ML, Bei B, Zimberg IZ, *et al.* (2018) Dietary disinhibition mediates the relationship between poor sleep quality and body weight. *Appetite* **120**, 602–608.
48. Kredlow MA, Capozzoli MC, Hearon BA, *et al.* (2015) The effects of physical activity on sleep: a meta-analytic review. *J Behav Med* **38**, 427–449.
49. Pearson N & Biddle SJ (2011) Sedentary behavior and dietary intake in children, adolescents, and adults: a systematic review. *Am J Prev Med* **41**, 178–188.
50. Christensen MA, Bettencourt L, Kaye L, *et al.* (2016) Direct measurements of smartphone screen-time: relationships with demographics and sleep. *PLoS One* **11**, e0165331.
51. Chennaoui M, Arnal PJ, Sauvet F, *et al.* (2015) Sleep and exercise: a reciprocal issue? *Sleep Med Rev* **20**, 59–72.
52. Ezeh U, Yildiz BO & Azziz R (2013) Referral bias in defining the phenotype and prevalence of obesity in polycystic ovary syndrome. *J Clin Endocrinol Metab* **98**, E1088–E1096.
53. Joham AE, Teede HJ, Ranasinha S, *et al.* (2015) Prevalence of infertility and use of fertility treatment in women with polycystic ovary syndrome: data from a large community-based cohort study. *J Women's Health* **24**, 299–307.
54. Day F, Karaderi T, Jones MR, *et al.* (2018) Large-scale genome-wide meta-analysis of polycystic ovary syndrome suggests shared genetic architecture for different diagnosis criteria. *PLoS Genetics* **14**, e1007813.

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