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Socioeconomic Status and Polycystic Ovary Syndrome

Sharon Stein Merkin, Ph.D.,[1] Ricardo Azziz, M.D., M.P.H., M.B.A.,[1,2] Teresa Seeman, Ph.D.,[1]
Ronit Calderon-Margalit, M.D., M.P.H.,[3] Martha Daviglus, M.D., Ph.D.,[4] Catarina Kiefe, Ph.D., M.D.,[5]
Karen Matthews, Ph.D.,[6] Barbara Sternfeld, Ph.D.,[7] and David Siscovick, M.D., M.P.H.[8]

Abstract

Background: Polycystic ovary syndrome (PCOS) is a common metabolic-endocrine disorder in women and is associated with a number of metabolic morbidities. We examined the association of PCOS and its components with socioeconomic status (SES) over the life course to explore the role of the environment on the development of PCOS.
Methods: Participants included 1163 women, aged 34–39, from the Coronary Artery Risk Development in Young Adults (CARDIA) Women's Study, examined at year 16 of the CARDIA study (2001). PCOS was defined according to the 1990 National Institutes of Health (NIH) criteria.
Results: Logistic regression models, adjusted for age, body mass index (BMI), waist circumference, and oral contraceptive (OC) use, demonstrated a statistically significant association between those women with low parental education/high personal education and PCOS (odds ratio [OR] 2.5, 95% confidence interval [CI] 1.4-4.4).
Conclusions: Our results indicate that women who experienced low childhood SES are at increased risk of PCOS, but this risk is limited to those who have personally attained a high level of education. More research is needed to determine the childhood socioeconomic factors that might influence this risk and whether conditions associated with upward life mobility play a role or if this group of at-risk women is simply more likely to recall the symptoms that define PCOS.

Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders in women, with an estimated prevalence of nearly 7%–10%.[1–3] This syndrome is characterized by ovulatory dysfunction, hyperandrogenism, and polycystic ovaries.[4] Despite a relatively high prevalence, the etiology and natural history of PCOS are not well understood; current theories suggest that increased risk of PCOS may involve a combination of genetic susceptibility and a myriad of environmental factors, including diet, lifestyle, and social factors.[5,6] The importance of gaining a greater understanding of this relatively common syndrome is further underscored by growing evidence that PCOS is strongly associated with several metabolic conditions, including insulin resistance,[7] type 2 diabetes mellitus, hypertension and dyslipidemia,[8] the metabolic syndrome,[9] and cardiovascular outcomes.[10]

The association of PCOS and its components with socioeconomic status (SES) might shed light on the role of the environment in the development of this condition. Research has shown that individuals with lower SES are more at risk for engaging in adverse health behaviors, including smoking, lack of physical activity, and poor nutritional diet[11,12]; among women, obesity is associated with low SES.[13,14] Studies have also shown that smoking and obesity can exacerbate insulin resistance,[15] which is a condition highly correlated with and part of the pathogenesis of PCOS.[16] Moreover, there is clear evidence of an association between low SES and cardiovascular disease (CVD),[17–20] as well as the metabolic syndrome and its various

[1]The David Geffen School of Medicine at UCLA, Los Angeles, California.
[2]Cedars-Sinai Medical Center, Los Angeles, California.
[3]Hebrew University-Hadassah Braun School of Public Health and Social Medicine, Jerusalem, Israel.
[4]Northwestern University, Feinberg School of Medicine, Chicago, Illinois.
[5]University of Massachusetts Medical School, Worcester, Massachusetts.
[6]University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania.
[7]Kaiser Permanente, Oakland, California.
[8]Cardiovascular Health Research Unit, Departments of Medicine and Epidemiology, University of Washington, Seattle, Washington.

components.[21] Considering that PCOS symptoms often begin in adolescence, we chose to examine the association between SES over the life course, from childhood to adulthood.

The objectives of this study are to (1) examine the association between SES and the prevalence of PCOS to determine if exposure to low SES over the life course is associated with an increased prevalence of PCOS; as part of this objective, we propose to examine how the different components of PCOS relate to SES, and (2) examine a possible interaction of this SES-PCOS association with race.

Materials and Methods

Study population

The study population included participants in the Coronary Artery Risk Development in Young Adults (CARDIA) Women's Study. CARDIA is a prospective, multicenter, population-based study of coronary artery disease (CAD) risk factors in young adults. This study population included 5115 men and women aged 18–30 at baseline, recruited in 1985–1986 from four metropolitan areas, including Birmingham, Alabama, Chicago, Illinois, Minneapolis, Minnesota, and Oakland, California, using community-based or list-based sampling frames. Follow-up examinations were conducted 2, 5, 7, 10, 15, and 20 years after baseline, with retention rates of 91%, 88%, 81%, 79%, 74%, and 72%, respectively. Details of the study design have been published.[22] At the year 15 examination, CARDIA women were recruited to participate in an ancillary study at year 16 (2002–2003), the CARDIA Women's Study (CWS), designed to examine the associations among androgens, polycystic ovaries, and cardiovascular risk. To be eligible for CWS, women had to have attended the year 15 examination have at least one ovary, and not be pregnant. A total of 1163 women (86% of the eligible sample) completed the CWS examination. Our study sample consisted of 545 white and 618 black women, aged 34–49, who participated in the CWS.

Measures

Ovulatory dysfunction was based on self-reported information obtained from the CWS questionnaire, where the participants were asked a variety of questions about the regularity of their menstrual cycles (excluding when they were pregnant, nursing, or experiencing menopause or surgery) when they were aged 20–30, as well as in the past year (number of days between menstrual cycles were classified as Less than 20, 20–35, 26–33, 34–45, More than 45). Additional information was obtained regarding oral contraceptive (OC) use and reasons for use.

Information on hirsutism included self-reported information on unwanted hair growth (not including lower leg or underarm) recalled from ages 20–30 and in the past year (yes or no). Testosterone (T) was measured using stored sera from the CARDIA year 2 follow-up examination (to correspond to the 20–30 age range) and sera collected at the CWS (year 16) examination. Total T was measured using a competitive immunoassay (Bayer Diagnostics) that employs direct chemiluminescent technology on the 180 Automated Chemiluminescent System (ACS) Free T was calculated based on total T and sex hormone-binding globulin (SHBG) measures.[23] Total and free T cutoffs of 80 and 0.65, respectively, were based on the 95th percentile T values for the nonhirsute, nonoligomenorrheic study population. T values were not considered for women using OC.

Life course SES included measures of parental education to indicate childhood SES and respondents' personal education to indicate adult SES. Although education is the only indicator used as a proxy for SES in this study, it is a common measure that has been well established in its inverse relationship with health and considered by some to be the best measure of SES with regard to health.[24] Parental education was assessed at baseline, based on participants' reports of the highest level of education obtained by their parents or primary caregiver. Personal education levels were measured in terms of the highest education degree earned as of year 15 (i.e., when participants were aged 32–47 years and not likely to change education levels). Both parental and personal education levels were classified as $\leq$high school and $>$high school education. Income was not considered in this analysis, as it was only available starting at year 5 and no parental income information was available.

To consider a life course trajectory of SES, we created four categories reflecting the possible combinations of parental and personal education, including low parental/low personal education, low parental/high personal education, high parental/low personal education, and high parental/high personal education.

Age and race were reported at baseline. Body mass index (BMI) and waist circumference were obtained from the year 2 examination (second follow-up visit) to correspond as much as possible to when participants were aged 20–30. BMI levels were classified as underweight ($<$18.5), normal (18.5–24.0), overweight (25.0–29.0), and obese ($\geq$30). High waist circumference was based on National Cholesterol Education Program (NCEP) criteria and classified as $>$88 cm.[25]

Defining PCOS

The definition of PCOS was based on the National Institutes of Health (NIH) criteria suggested at an expert conference in 1990, considering the presence of ovulatory dysfunction plus clinical or biochemical signs of hyperandrogenism after exclusion of related disorders.[26] We defined ovulatory dysfunction as $\geq$34 or $<$20 days between menstrual cycles or using OC to regulate periods as recalled from when participants were aged 20–30. Hyperandrogenism was determined based on hirsutism (self-reported unwanted hair growth at ages 20–30) or high T, defined by either high free T ($>$0.65 ng/dL, $>$0.02 nmol/L) or high total T ($>$80 ng/dL, $>$2.78 nmol/L) measured at year 2 for those not taking OC. Participants were also asked about experiences during the CWS year. We expanded the PCOS definition to include information on the PCOS components as recalled from the past year, but only for women aged $<$40 who had no PCOS based on recall information from ages 20–30; for these same women, current OC use to regulate periods/vaginal bleeding was considered a marker of ovulatory dysfunction. When considering PCOS status, we included only participants who had at least some information on both ovulatory dysfunction and hyperandrogenism ($n = 938$).

In sensitivity analyses, we considered an additional measure of PCOS that defined ovulatory dysfunction more restrictively, as $>$45 days between menstrual cycles (PCOS 2). A description of this measure can be found in Table 4. In

additional analyses, we considered the set of criteria established at a consensus workshop in Rotterdam in 2003[4] that included the presence of polycystic ovaries. However, because 40% of the sample did not receive a full transvaginal ultrasound examination, we do not include those results.

Although the NIH definition of PCOS calls for the exclusion of related disorders, we should note that the prevalence of thyroid dysfunction, hyperprolactinemia, congenital adrenal hyperplasia, or androgen-secreting tumors is relatively rare in patients with suspected androgen excess or PCOS,[3] allowing us to disregard these disorders in our analysis. In our study sample, we had information available only about self-reported thyroid problems, indicated by 46 women in our study sample. In sensitivity analyses, we reran the models and excluded those participants and found the same results; we thus present the main results disregarding this condition.

Statistical analyses

Initial descriptive analyses were conducted to compare the study sample with the eligible CWS sample, based on year 15 attendance. We then assessed prevalence of PCOS and its components, by age, race, SES, obesity, and high waist circumference; p values for differences were obtained using t tests for continuous variables and chi-square tests for categorical measures. Logistic regression models were used for multivariate analyses, and models were fit for PCOS and each component. Interactions between race and each education level, as well as race and the combined life course education levels, were assessed using interaction terms. In addition, models were stratified by race groups; p values <0.05 were considered statistically significant. SAS version 9.2 was used for these analyses.

Results

Compared to nonparticipants from the CARDIA population who had attended the year 15 follow-up examination, our CWS study sample had a slightly higher percentage of black participants, women with less education, and women with higher BMI. The remaining examined characteristics were not significantly different between the groups (data not shown).

The prevalence of PCOS was 10.7%. When considering the individual components separately, 29.0% of the sample reported oligomenorrhea, 23.3% reported hirsutism, and 10.3% had high T levels (Table 1). The prevalence of PCOS and its components varied by parental and personal education levels. For all outcomes, prevalence was higher at the lower parental education level, although this association was not statistically significant for hirsutism. The association was less consistent for personal education, with a significantly higher prevalence of PCOS at the highest education level and greater prevalence of high T associated with lower education.

The only statistically significant racial difference was higher prevalence of high T among blacks compared to whites. Obese participants and those with high waist circumference had a significantly higher prevalence of high T; obese participant also had a higher prevalence of hirsutism.

Although not shown in Table 1, younger age at reporting (34–39 at the CWS examination) was associated with a higher prevalence of PCOS, oligomenorrhea, and hirsutism. This association was expected, as recall is likely better in the younger age groups for whom the request for information about their 20s reflects recall over a shorter period of time. We did not include these results in Table 1 because age is also built into the definition, where information about symptoms at year 16 was only incorporated into the definition for those <40 years.

When considering both parental educational and personal education in the same model and adjusting for age at the CWS examination, race, BMI at year 2, waist circumference at year 2, and OC use at years 2 and 16, the results were different by type of education indicator (results not shown). Low parental education was associated with higher risk of PCOS and most of its components, whereas low personal education was associated with lower risk of these conditions (for PCOS, odds ratio [OR] 2.2, 95% confidence interval [CI] 1.2-3.9 for low parental education; OR 0.4, 95% CI 0.2-0.7 for low personal education). On further examination of the joint effects of parental and personal education, the results indicated that the greatest risk of PCOS and its components was associated with those with low parental high personal education compared to high parental/high personal education. As shown in Table 2, the models yielded statistically significant ORs for this group for PCOS (OR 2.5, 95% CI 1.4-4.4) and for hirsutism (OR 1.6, 95% CI 1.1-2.4). ORs indicated risk for oligomenorrhea and high T (borderline significance). For those who were downwardly mobile (i.e., with high parental education coupled with low personal education), there was an apparent protective effect associated with PCOS, although it was not statistically significant. For those with low parental/low personal education, the only statistically significant association found was a strong risk associated with high T levels (OR 2.3, 95% CI 1.2-4.6). Interaction terms for race and education levels were not statistically significant.

Considering the evidence that obese women with PCOS are more insulin resistant than lean women with PCOS,[27,28] possibly indicating that this phenotype may be more susceptible to environmental factors, we assessed how these SES associations might interact with obesity. We reran the model in Table 2 for PCOS, stratified by obesity at year 2 (Table 3). Similar to the nonstratified findings, the only statistically significant risk of PCOS was associated with low parental/high personal education, although this risk was considerably stronger among obese compared to nonobese women (OR 4.4, 95%CI 1.0-18.4 for obese and OR 2.0, 95% CI 1.1-3.9 for nonobese women).

When considering an alternate, more restrictive measure of PCOS, the results were similar, with a higher OR of PCOS for those with low parental/high personal education among obese women (OR 5.9, 95% CI 1.1-31.7) (Table 4). In additional sensitivity analyses, we considered finer categories of SES; however, the results yielded the same trends, and, thus, to ensure adequate power, we present the main findings.

Discussion

Our results indicate a strong association between low childhood SES and PCOS, primarily among women with high SES in adulthood. After adjusting for age, race, BMI, waist circumference, and OC use, this group was over twice as likely to have PCOS as women with both high parental and high personal education. We further found that this association was strongest among obese women, with >4 times the risk associated with PCOS for obese women with low

Table 1. Prevalence of Polycystic Ovary Syndrome and Individual Components by Select Characteristics

	PCOS[a] n = *938*	*Oligomenorrhea*[b] n = *939*	*Hirsutism*[c] n = *1157*	*High testosterone*[d] n = *800*
Overall, %	10.7	29.0	23.3	10.3
Race, %	*n* = 938	*n* = 939	*n* = 1157	*n* = 800
White	10.7	27.7	24.1	7.3
Black	10.6	30.2	22.5	12.6
p value	0.9	0.4	0.5	0.02
Parental education (baseline), %	*n* = 889	*n* = 890	*n* = 1093	*n* = 751
≤High school	13.7	32.7	25.1	14.2
>High school	9.0	26.5	23.3	8.4
p value	0.03	0.05	0.5	0.01
Education (year 15), %	*n* = 886	*n* = 887	*n* = 1087	*n* = 748
≤High school	8.1	29.4	20.2	12.5
>High school	12.1	28.3	25.1	8.1
p value	0.05	0.7	0.06	0.05
BMI (year 2),%	*n* = 900	*n* = 901	*n* = 1111	*n* = 785
Underweight (<18.5)	11.3	25.7	27.3	0
Normal (18.5–24)	9.5	29.4	20.4	7.8
Overweight (25–29)	10.6	27.8	24.7	13.7
Obese (≥30)	14.4	31.0	28.5	14.3
p value	0.4	0.9	0.08	0.01
Obesity (year 2),%	*n* = 900	*n* = 901	*n* = 1111	*n* = 785
Obese (BMI≥30)	14.4	31.0	28.5	14.3
Nonobese (<30)	9.9	28.8	21.9	9.1
p value	0.09	0.6	0.04	0.04
High waist circumference (year 2), (%)	*n* = 897	*n* = 898	*n* = 1107	*n* = 781
>88 cm	15.4	32.2	25.8	18.1
≤88 cm	9.7	28.5	22.5	8.5
p value	0.04	0.4	0.3	0.0006

[a]PCOS: Defined as irregular menses (≥34 or <20 days between cycles) or taking oral contraceptive (OC) pills to regulate periods) during ages 20–30 and either unwanted hair growth (during their 20–30s) or high free (0.65) or total testosterone (T) (>80) measured at year 2 for those not on OC. These measures were also considered for the past year (year 16) for those aged <40 who had no PCOS based on recall information from ages 20–30; for these same women, current OC use (year 16) to regulate periods/vaginal bleeding was considered a marker of oligomenorrhea.

[b]Oligomenorrhea: Defined as ≥34 or <20 days between cycles or taking OC pills to regulate periods during ages 20–30 or ≥34 or <20 days between cycles in past year if <40 years old and no menopause or hysterectomy) for women <40 years of age. Current OC pill use (year 16) to regulate periods/vaginal bleeding was also considered a marker of oligomenorrhea.

[c]Hirsutism: Self-reported unwanted hair growth at ages 20–30 or in past year (only for women <40 years).

[d]High testosterone: Total T>80 or free T>0.65 at year 2 or year 16 (restricted to those not on OC pills during those years and to those aged <40 for year 16 values).

BMI, body mass index; PCOS, polycystic ovary syndrome.

parental/high personal education. Women with low parental/low personal education, as well as those with high parental/low personal education did not show an increased risk of PCOS. There was no statistically significant interaction between race and SES with regard to PCOS.

Although other studies have not explored the association between PCOS and SES, there is abundant evidence that among women, low SES is associated with many conditions related to PCOS, including obesity[29] and insulin resistance.[30] Low childhood SES, in particular, has been found associated with related conditions, including diabetes,[31] insulin resistance,[30,32] obesity,[29] and coronary heart disease (CHD).[32–34] Moreover, as in our analysis, these studies found that the association between these conditions and low childhood SES persisted even among those who moved up into higher levels of SES during adulthood.[30,33,34] The mediating factors that might link low childhood SES to a higher risk of developing PCOS include poor intrauterine or childhood nutrition,[32] excessive weight gain in infancy or early childhood,[31,35,36] and adopting adverse health behaviors.[11] Research suggests that these factors might influence later metabolic dysfunction,[31,35,36] and there is growing evidence that prenatal androgen excess plays a role in the development of PCOS.[37] A measure of childhood SES might thus be more influential than personal education or other measures of SES that are attained later in life, when PCOS may already have developed.

Although other childhood factors were not available in this dataset, we explored some of the potential adverse health behaviors that may have influenced the development of PCOS, including diet (total caloric intake, fat and carbohydrate intake), weight change, smoking, and physical activity, as measured at the CARDIA year 2 follow-up (data not shown). Although adjusting for these factors did not change the relationship between low SES and PCOS, it is possible that these measures did not adequately reflect prepubescent exposures that would most influence the development of PCOS.

In these analyses, we found that the low childhood SES-PCOS association was most pronounced among obese women. Several studies have shown that obesity exacerbates insulin resistance and hyperandrogenism, and there is evidence suggesting that obese women with PCOS are more insulin resistant then their lean counterparts.[28,38] Our findings

Table 2. Odds Ratios and 95% Confidence Intervals for Life Course Socioeconomic Status Trajectory with Risk of Polycystic Ovary Syndrome and Related Components

		Joint parental and personal education categories			
	n	*Low parental/low personal education* n = *161*	*Low parental/high personal education* n = *133*	*High parental/low personal education* n = *138*	*High parental/high personal education* n = *362*
PCOS[a]	794	0.9 (0.5-1.8)[b] *n* = 162	2.5 (1.4-4.4) *n* = 133	0.6 (0.3-1.4) *n* = 138	1.0 *n* = 362
Oligomenorrhea[a]	795	1.3 (0.9-2.0) *n* = 193	1.4 (0.9-2.2) *n* = 173	1.0 (0.6-1.5) *n* = 176	1.0 *n* = 428
Hirsutism[a]	970	0.7 (0.5-1.2) *n* = 131	1.6 (1.1-2.4) *n* = 109	0.9 (0.6-1.4) *n* = 146	1.0 *n* = 291
High testosterone[a]	677	2.3 (1.2-4.6)	2.0 (0.9-4.2)	1.1 (0.5-2.4)	1.0

[a]For definitions, see Table 1.
[b]OR (95% CI).
Models are additionally adjusted for age at year 16, BMI at year 2, waist circumference at year 2, race, OC use at year 2, and OC use at year 16.
OR, odds ratio; CI, confidence interval.

highlight the important role that lifestyle factors and environmental conditions may play in the development of PCOS, particularly in the development of the PCOS phenotype most vulnerable to negative metabolic sequelae, as we described earlier. This is also consistent with epigenetic studies that have suggested that genetic predisposition for PCOS can be influenced by environmental factors,[39,40] including diet and exercise. Our findings with regard to obesity could indicate that the principal driver of low parental education on PCOS is through development of poor nutritional habits and the subsequent development of obesity.

It is less clear why the low childhood SES-PCOS association might be limited to those who attain a higher level of education. We speculate that this may be the result of how PCOS was determined in this study. Aside from measuring T levels, PCOS was primarily determined based on self-reported information about menstrual cycles, hair growth, and OC use. It is possible that those with higher education were more likely to recall and report past and current health conditions and that those women might be more likely to use the healthcare system and, thus, have a diagnosis. Moreover, women with higher education may be more likely to complain of hirsutism or use OC to regulate menstrual cycles. Consequently, the group with the most risk associated with poorer childhood conditions and more ability to recall because of higher attained education might indeed be at greater risk of being assigned as having PCOS in this study. This might also explain the protective (although not statistically significant) effect for those with high parental education/low personal education, that is, a combination of better childhood socioeconomic conditions and perhaps less likelihood of recalling these health measures. This hypothesis is supported by the fact that the one measure that did not show this effect was high T, a measure that did not require recall information. Those with low parental/low personal education had, as expected, the highest association with T, whereas the risk was reduced (and borderline significant) for those with low parental/high personal education, and further reduced and not significant for those with high parental/low personal education (Table 2). Those results show that low parental education is clearly a more influential factor in its association with high T, although low personal education confers some risk as well.

Notwithstanding these concerns with the self-reported elements of our PCOS measure, our results suggest an additional hypothesis that might explain why the risk of PCOS associated with low parental education is limited to women with higher education. It is possible that women who achieve a higher level of education distinct from their parents (i.e., upwardly mobile) suffer more from peripubertal stress, a greater incidence of obesity, and thus early (and perhaps persistent) disruption in their menstrual cyclicity.[41] Although no other study has examined the effects of upward socioeconomic mobility and PCOS, some studies have found upward mobility to be associated with persistent risk of CVD and mortality despite its overall advantage compared to downward socioeconomic mobility.[42,43] This hypothesis warrants further research.

The present study has a number of strengths, including the use of a diverse, young, population-based sample, but there are some limitations. The outcome measure we used to identify PCOS was primarily determined via self-reported menstrual irregularities, OC use, and excessive hair growth.

Table 3. Odds Ratios and 95% Confidence Intervals for Life Course Socioeconomic Status Trajectory with Risk of Polycystic Ovary Syndrome, by Obesity at Year 2

PCOS[a]	n	*Low parental/low personal education*	*Low parental/high personal education*	*High parental/low personal education*	*High parental/high personal education*
Obese	142	0.7 (0.1-3.3)	4.4 (1.0-18.4)	0.5 (0.1-2.3)	1.0
Nonobese	652	0.9 (0.4-2.0)	2.0 (1.1-3.9)	0.6 (0.2-1.6)	1.0

[a]For definition, see Table 1.
Models are additionally adjusted for age at year 16, OC use at year 2, and OC use at year 16.

Table 4. Odds Ratios and 95% Confidence Intervals for Risk of Polycystic Ovary Syndrome Using an Alternate Measure

PCOS 2[a]	n	*Low parental/low personal education*	*Low parental/high personal education*	*High parental/low personal education*	*High parental/high personal education*
Prevalence 7.1%	938				
		n = 161	n = 133	n = 138	n = 362
Overall	794	0.8 (0.4-1.8)	2.1 (1.1-4.0)	0.3 (0.1-1.0)	1.0
Obese	142	0.1 (0.01-1.5)	5.9 (1.1-31.7)	0.1 (0.02-1.4)	1.0
Nonobese	652	1.0 (0.4-2.5)	1.7 (0.8-3.6)	0.3 (0.1-1.3)	1.0

[a]PCOS 2: Defined as irregular menses (>45 days between cycles or taking OC pills to regulate periods) during ages 20–30 and either unwanted hair growth (during their 20–30s) or high free (0.65) or total T (>80) measured at year 2 for those not taking OC pills. These measures were also considered for the past year (year 16) for those aged <40 who had no PCOS, based on recall information from ages 20–30; for these same women, current OC use (year 16) to regulate periods/vaginal bleeding was considered a marker of oligomenorrhea.

Models are additionally adjusted for age at year 16, BMI at year 2, waist circumference at year 2, race, OC use at year 2, and OC use at year 16.

Aside from the fact that the validity of recalled information about menstrual cycle length and variability may be differential by SES, studies have also shown that self-reports may be somewhat unreliable.[44,45] An ideal analysis would include recorded menstrual cycle length. Whereas clinical measures of hirsutism would also be ideal, studies have found that even among women with minimum excess hair growth, 50% were diagnosed with PCOS,[46] and, thus, self-reported hair growth is likely an adequate indicator of hirsutism. Additional limitations include missing values, with about 30% of the study sample missing T measures, mostly because of concurrent OC use. Although these women were able to contribute information about the other PCOS components, further analysis showed that they had a higher prevalence of oligomenorrhea and low parental/high personal education than women not missing T. The exclusion of these missing data potentially dilutes our findings.

Conclusions

This study points to an association between low childhood SES and PCOS, particularly among those who later achieve high personal education and especially for obese women in this category. Our findings suggest that future research is warranted to explore early life exposures related to low SES and possibly to upward mobility, including intrauterine and postnatal nutrition and growth, and how they may influence the development of PCOS. These studies should strive to include validated measures of PCOS symptoms to avoid any self-reported bias that might be associated with social factors.

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Disclosure Statement

The authors have no competing financial interests.

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Address correspondence to:
Sharon Stein Merkin, Ph.D.
Division of Geriatrics
Geffen School of Medicine at UCLA
10945 Le Conte Avenue, Suite 2339
Los Angeles, CA 90095-1687

E-mail: smerkin@mednet.ucla.edu