

Comparison of Dietary Intake and Physical Activity between Women with and without Polycystic Ovary Syndrome: A Review^{1,2}

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ABSTRACT

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder affecting women of reproductive age worldwide. In addition to deleterious effects on fertility imparted by PCOS, women with PCOS are at increased risk of obesity, diabetes, cardiovascular disease, depression, and certain cancers. Hormonal and metabolic aberrations in PCOS have the potential to influence dietary intake and physical activity levels. There are emerging global data that women with PCOS have different baseline dietary energy intakes compared with women without PCOS. These alterations in diet may exacerbate clinical symptoms and compound risk of chronic disease in patients. Few studies have compared baseline physical activity levels between women with and without PCOS. Although comparisons between studies are confounded by several factors, the data point to no differences in activity levels among PCOS and non-PCOS groups. This review provides an assessment of the current literature on baseline dietary intake and physical activity levels in women with PCOS. Future recommendations to strengthen research in this area are provided, given the implications to aid in the development of effective nutrition-focused interventions for PCOS. *Adv. Nutr.* 5: 486–496, 2014.

Introduction

As a leading cause of anovulatory infertility and a risk factor for endometrial dysfunction and uterine cancer, polycystic ovary syndrome (PCOS) represents a serious health concern for women across the life span (1,2). PCOS is characterized by a heterogeneous collection of symptoms: infrequent or absent menstrual cycles, biochemical or clinical evidence of androgen excess, and polycystic ovarian morphology (3,4). PCOS occurs in a striking proportion of women of reproductive age, ranging from 6% to 15% worldwide, depending on the diagnostic criteria used (3,5,6). PCOS should be regarded as a broad-spectrum disorder because its consequences for patients extend beyond impairments of the reproductive system to include serious metabolic (i.e., metabolic syndrome, type 2 diabetes, and cardiovascular disease) and psychological sequelae (i.e., depression, anxiety, poor self-esteem, and reduced quality of life) (3,7,8).

Researchers have established that up to 80% of the PCOS population is overweight or obese with obesity prevalence rates, dependent on the ethnicity and geographical location

(3,9). Although PCOS can manifest in both normal weight and overweight women, some evidence supports that increased central adiposity is present across all BMI categories (10–12). It is debatable as to whether women with PCOS have a unique predisposition to obesity or whether obesity drives development of PCOS (13). Data supporting lower basal metabolic rate (14) and postprandial thermogenesis (15) in individuals with PCOS compared with age- and weight-matched controls may account for a higher prevalence of obesity among the PCOS population. However, reports on differences in basal metabolic rate among women with or without PCOS are inconsistent (16). There is also the potential for appetite circuits to be affected by the abnormal hormone profile in PCOS. Testosterone replacement was shown to increase meal frequency in male rodents (17), whereas anti-androgenic pharmaceutical therapy was found to reduce meal-related hunger in women with bulimia (18). The anti-androgenic finding may be particularly relevant because women with PCOS exhibit appetite indications similar to those in women with bulimia (19). Women with PCOS also demonstrated smaller reductions in postprandial ghrelin (i.e., an orexigenic hormone) and lower postprandial cholecystokinin concentrations (i.e., an anorexigenic hormone) compared with age- and/or weight-matched controls

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(20,21). Collectively, these findings are consistent with the hypothesis that women with PCOS have lower perceived satiety and greater appetite compared with women without PCOS. These findings are tempered by other studies that showed no differences or a blunted response in ghrelin concentrations among women with or without PCOS (22,23). Last, experimental and clinical evidence supports that testosterone promotes abdominal fat deposition in women (11,24,25). Increased abdominal adiposity has been linked to elevated leptin secretion and leptin resistance, which may result in impaired satiety and increased energy intake (26). Irrespective of whether PCOS causes obesity or a reverse causation exists, it is recognized that obesity, particularly abdominal obesity, worsens clinical and metabolic features of PCOS (3).

Lifestyle intervention is recommended as a first-line treatment in overweight and obese women with PCOS (27). Uncontrolled trials involving hypocaloric diets with physical activity (1200 kcal/d) and low-carbohydrate, ketogenic diets (<20 g carbohydrate/d, unlimited consumption of high-biologic-value protein and dairy) support improvements in hyperandrogenism, frequency of menses, ovulation, pregnancy rates, insulin resistance, and lipid profile when accompanied by modest weight reductions for women with PCOS (28,29). Randomized controlled trials with reduced-energy diets also support improvements in hyperandrogenism and insulin resistance in women with PCOS. Yet, data on ovulation and other reproductive outcomes are less clear (30–32). There are limited data on the feasibility or effectiveness of long-term weight-loss interventions for this population. Moreover, only a few studies examined diet alterations to improve cardiometabolic risk factors in normal weight women with PCOS (33,34). Understanding the baseline dietary intake and physical activity levels of the PCOS population is essential to aid in the development of effective lifestyle interventions in free-living settings. The primary aim of this review was to examine the current literature on baseline dietary intake and physical activity habits in women with PCOS. Furthermore, this review provides recommendations to strengthen future studies in this area of research.

Studies were identified by searching the electronic databases PubMed, CINAHL, and PsycINFO for studies published after 1990 and before January 2014. A search was performed by using a combination of keywords relevant to PCOS and diet, lifestyle, and nutrition assessment methods. Ten studies from various countries were included in the review based on a Population, Intervention, Comparison, Outcome framework established a priori by the authors. In short, studies included for review were limited to original research articles in which 1) the primary objective was to assess baseline diet and physical activity levels between adult women with and without PCOS, 2) enrollment exceeded 10 participants in each study arm, and 3) diet and physical activity were assessed in a free-living sample. A description and the main findings of each study included for review are summarized in [Table 1](#).

Current Status of Knowledge

Comparison of dietary intake between women with and without PCOS. Two studies compared baseline dietary intake between women with and without PCOS by using case-control study designs in the United States (35,36). Wright et al. (36) assessed dietary intake by using FFQs in mostly middle-aged women undergoing the perimenopausal transition. This was evidenced by the number of women in both control and PCOS groups who reported the absence of menses for 12 mo. By contrast, Douglas et al. (35) assessed the food records of reproductive-aged women who were ~20 y younger than the sample used by Wright et al. Both dietary assessment methods used by these studies have been commonly used to assess dietary intake (37,38), yet each has distinct strengths and weaknesses. Whereas diet records over several days are expected to reflect usual intake and have less reliance on participant memory, this approach may have limited accuracy because participants are aware that their dietary intake would be scrutinized on specific days. This may result in atypical dietary intake and provide misleading dietary information (38,39). The FFQ is an appropriate measure to assess usual dietary composition over a longer period of time; however, the accuracy of the data can be limited by the respondents' abilities to recall their diet habits (38). It is also impossible to discern whether a PCOS diagnosis may have been a catalyst to altered dietary intake due to the study design.

When the data were pooled without regard to body composition, both Wright et al. (36) and Douglas et al. (35) reported no significant differences in micro- and/or macronutrient intake among women with PCOS and controls. The data were consistent with studies conducted in Italy (40,41) and Spain (42), in which researchers either reported no differences in energy and/or nutrient intake among women with and without PCOS as evidenced by 24-h dietary recall, 7-d food records, and FFQ (35,36). However, Douglas et al. (35) noted that the PCOS group consumed more servings of white bread compared with the control group. When dietary intake was assessed with respect to BMI categories, Wright et al. (36) reported that normal weight women with PCOS (BMI <25 kg/m²) consumed significantly lower total energy diets compared with BMI-matched women without PCOS (~400 fewer kcal). This may be attributed to the lower reported intakes of carbohydrates (~43 g), protein (~15 g), total fat (~19 g), saturated fat (~5 g), monounsaturated fat (~7 g), polyunsaturated fat (~6 g), and cholesterol (~60 mg) by the normal weight PCOS group compared with controls. An examination of food servings also revealed that normal weight women with PCOS consumed less bread, cereal, rice, pasta, and meat products compared with BMI-matched controls. This may be considered clinically significant as it provided an energy difference of ≥250 kcal/d between the 2 groups. These findings led Wright et al. to hypothesize that women with PCOS within a normal weight range restricted their daily energy intake to a clinically significant margin to offset

TABLE 1 Characteristics of studies included in the review assessing baseline diet and/or physical activity in women with and without PCOS¹

Study (reference)	Sample, assessments used	Outcomes ²	Limitations
Wright et al. 2004 (36)	<p>Groups: n = 84, PCOS; n = 79, controls</p> <p>Age: 46.7 ± 5.8 y, PCOS; 48.2 ± 5.7 y, controls</p> <p>BMI (kg/m²): 32.1 ± 9.3, PCOS; 29.0 ± 6.0, controls</p> <p>Location: Pittsburgh, PA</p> <p>Race: Caucasian: 83%, PCOS; 90%, controls; non-Caucasian: 13%, PCOS; 10%, controls</p> <p>PCOS definition: oligoamenorrhea plus either hirsutism, hyperandrogenism and/or elevated LH/FSH</p> <p>No specific exclusion criteria were applied</p> <p>Assessments: FFQ, physical activity questionnaire</p>	<p>No differences in daily food and nutrient intake or physical activity between PCOS and control groups</p> <p>Lower nutrient intake in normal weight PCOS groups* (n = 21) vs. normal weight control (n = 33) groups*: total energy/d, CHO (g/d), protein (g/d), fat (g/d), SFAs (g/d), MUFAs (g/d), PUFAs (g/d), cholesterol (mg/d)</p> <p>Lower bread, cereal, rice, pasta, meat, fish, poultry, egg intake in normal weight PCOS vs. normal weight control groups (servings)*</p> <p>Lower milk product intake in overweight PCOS (n = 15) vs. overweight control (n = 19) groups (servings)*</p> <p>Lower meat, fish, poultry, egg intake in obese PCOS (n = 48) vs. obese control (n = 27) groups (servings)*</p> <p>Higher carbohydrate and lower fat intakes in PCOS group vs. Reaven study recommendations (49)</p> <p>No differences in nutrient intake between PCOS and control groups</p> <p>Greater white bread intake in PCOS vs. control groups (servings)*</p>	<p>Diagnostic criteria used yielded a heterogeneous PCOS group</p> <p>Population studied used medications known to influence endocrine profile (e.g., oral contraceptive, antiandrogens)</p> <p>No reported exclusion criteria on medications that may influence weight, appetite</p> <p>Older, potentially perimenopausal, populations studied, heterogeneous control group used with 41% reporting oligoamenorrhea; low generalizability to younger women with PCOS</p> <p>Power analysis not provided for post hoc comparisons among BMI-matched groups</p> <p>Did not report energy expenditure or energy balance</p> <p>Did not compare with U.S. DRI</p>
Douglas et al. 2006 (35)	<p>Groups n = 30, PCOS; n = 27, controls</p> <p>Age: 28.9 ± 6.3 y, PCOS; 28.9 ± 6.5 y, controls</p> <p>BMI (kg/m²): 29.1 ± 4.8, PCOS; 29.7 ± 4.8, controls</p> <p>Location: Birmingham, AL</p> <p>Race: Caucasian: 83%, PCOS; 85%, controls; black: 13%, PCOS; 11%, controls other: 4%, PCOS; 4% controls</p> <p>PCOS definition: oligoamenorrhea plus hirsutism and/or hyperandrogenism</p> <p>Exclusion criteria: diabetes, use of insulin sensitizers or glucose-lowering drugs and adherence to a modified diet</p> <p>Assessments: 4-d food records (Wed/Thu/Sat/Sun)</p>	<p>No differences in nutrient intake and physical activity between PCOS and control groups</p> <p>PCOS group intake vs. U.S. dietary recommended intake*:</p> <p>Above: total fat (g/d), SFAs (% of energy/d), MUFAs (% of energy/d), dietary cholesterol (mg/d), sodium (mg/d), vitamin C (mg/d), vitamin D (µg/d), calcium (mg/d), magnesium (mg/d)</p> <p>Below: fiber (g/d), potassium (mg/d), vitamin E (mg/d)</p>	<p>Population studied used drugs known to influence endocrine profile</p> <p>No reported exclusion criteria on other medications that may influence weight, appetite</p> <p>Comprehensive dietary intake not collected on all days of week</p> <p>Overall study groups not matched for BMI</p> <p>Power analysis not provided</p> <p>Data on physical activity not collected</p> <p>Did not compare with U.S. DRI</p>
Álvarez-Blasco et al. 2011 (42)	<p>Groups: n = 22, PCOS; n = 59, controls</p> <p>Age[‡]: 26.3 ± 7.6 y, PCOS; 32.2 ± 7.5 y, controls</p> <p>BMI (kg/m²): 35.2 ± 6.7, PCOS; 32.2 ± 6.1, controls</p> <p>Location: Madrid, Spain</p> <p>Race not reported</p> <p>PCOS definition: oligoamenorrhea plus hirsutism and/or hyperandrogenism</p> <p>Exclusion criteria: use of hormonal contraception and medications that interfere with metabolism, hypocaloric dieting, implausible energy intake, supplement use</p> <p>Assessments: FFQ, exercise habits assessed using interview</p>	<p>No differences in nutrient intake and physical activity between PCOS and control groups</p> <p>PCOS group intake vs. U.S. dietary recommended intake*:</p> <p>Above: total fat (g/d), SFAs (% of energy/d), MUFAs (% of energy/d), dietary cholesterol (mg/d), sodium (mg/d), vitamin C (mg/d), vitamin D (µg/d), calcium (mg/d), magnesium (mg/d)</p> <p>Below: fiber (g/d), potassium (mg/d), vitamin E (mg/d)</p>	<p>Study groups not matched for age</p> <p>Power analysis not provided</p> <p>Details on physical activity assessment tool not reported</p> <p>Did not report energy expenditure or energy balance</p> <p>Did not compare intake with EFSA-recommended intake, which is established for European countries</p>

(Continued)

TABLE 1 (Continued)

Study (reference)	Sample, assessments used	Outcomes ²	Limitations
Barr et al. 2011 (43)	<p>Group $n = 198$, PCOS</p> <p>Age: 32.6 ± 6.3 y</p> <p>BMI (kg/m^2): 27.4 ± 7.3</p> <p>Location: London, UK</p> <p>Race: Caucasian: 97%, PCOS; unknown: 3%, PCOS</p> <p>PCOS definition not provided</p> <p>Exclusion criteria: pregnancy, breastfeeding, eating disorders, and use of weight-loss medications</p> <p>Assessments: 7-d food and activity record</p>	<p>Greater daily nutrient intake in PCOS vs. controls (national survey reference)*: total energy/d, CHO (g/d), protein (g/d), fat (g/d), fat (% of energy/d), SFAs (g/d), MUFAs (g/d), PUFAs (g/d), total sugar (g/d), fiber (g/d)</p> <p>Lower daily nutrient intake in PCOS vs. controls (national survey reference)*: CHO (% of energy/d) vs. overweight PCOS ($n = 100$) groups* PCOS group intake vs. UK recommended intake*:</p> <p>Above: total energy/d, protein (g/d), fat (g/d), SFAs (g/d), MUFAs (g/d), PUFAs (g/d)</p> <p>Below: CHO (g/d), fiber (g/d)</p> <p>Greater activity in moderate-intensity physical activity (min/d) in normal weight ($n = 80$) vs. overweight ($n = 100$) PCOS groups*</p> <p>No differences in nutrient intake between PCOS and control groups</p> <p>PCOS group intake vs. U.S. recommended intake*:</p> <p>below: fiber (g/d), MUFAs (% of energy/d), PUFAs (% of energy/d)</p> <p>No associations between androgen status and nutrients</p>	<p>Diagnostic criteria for PCOS not provided, heterogeneous PCOS group studied</p> <p>Recruitment based on self-reported diagnosis of PCOS</p> <p>Reference population may contain women with PCOS</p> <p>No reported exclusion criteria on medications that may influence endocrine profile</p> <p>Older, potentially perimenopausal, women included</p> <p>Did not report energy expenditure or energy balance</p>
Toscani et al. 2011 (41)	<p>Groups: $n = 43$, PCOS; $n = 37$, controls</p> <p>Age³: 22.7 ± 5.6 y, PCOS; 29.7 ± 4.9 y, controls</p> <p>BMI (kg/m^2): 30.9 ± 5.5, PCOS; 29.7 ± 5.2, controls</p> <p>Location: Porto Alegre, Brazil</p> <p>Race: Caucasian: 90%, PCOS; 74%, controls; African-European: 10%, PCOS; 26%, controls</p> <p>PCOS definition: oligomenorrhea plus either hirsutism and/or hyperandrogenism</p> <p>Exclusion criteria: medications known to interfere with hormone concentrations, BMI >40 kg/m^2 and diabetes</p> <p>Assessments: 24-h dietary recall</p>	<p>Greater daily nutrient intake in PCOS vs. control groups*: fat (% of energy/d)</p> <p>Lower daily nutrient intake in PCOS vs. control groups*: total energy/d, CHO (g/d), CHO (% of energy/d)</p> <p>Positive associations among hormones and nutrients in PCOS*: FSH and CHO (g/d), FSH and CHO (% of energy/d)</p> <p>No differences in daily nutrient intake between hyperandrogenic ($n = 21$) and nonandrogenic ($n = 24$) PCOS groups</p>	<p>Study groups not matched for age</p> <p>No reported exclusion criteria on medications that may influence weight and appetite</p> <p>High reporting bias because participants may alter diet before scheduled visit</p> <p>Data on physical activity not collected</p> <p>Comparisons with U.S. recommended intake may not be appropriate for Brazilian populations</p>
Tsai et al. 2012 (47)	<p>Groups: $n = 45$, PCOS; $n = 161$, controls</p> <p>Age: 32.7 ± 4.2 y, PCOS; 34.7 ± 3.6 y, controls</p> <p>BMI (kg/m^2): 23.0 ± 4.4, PCOS; 21.3 ± 2.9, controls</p> <p>Location: Taipei, Taiwan</p> <p>Race not reported</p> <p>PCOS definition: 2 of 3 symptoms: 1) oligomenorrhea, 2) hirsutism and/or hyperandrogenemia, 3) polycystic ovaries</p> <p>Exclusion criterion: hormonal therapy</p> <p>Assessments: 3-d food record (2 weekdays, 1 weekend day)</p>	<p>Greater daily nutrient intake in PCOS vs. control groups*: fat (% of energy/d)</p> <p>Lower daily nutrient intake in PCOS vs. control groups*: total energy/d, CHO (g/d), CHO (% of energy/d)</p> <p>Positive associations among hormones and nutrients in PCOS*: FSH and CHO (g/d), FSH and CHO (% of energy/d)</p> <p>No differences in daily nutrient intake between hyperandrogenic ($n = 21$) and nonandrogenic ($n = 24$) PCOS groups</p>	<p>Diagnostic criteria used yielded heterogeneous PCOS group</p> <p>Control group comprised infertile women with various etiologies including unexplained infertility</p> <p>Study groups not matched for BMI</p> <p>No reported exclusion criteria on medications that may influence weight and appetite</p> <p>Power analysis not provided for post hoc comparisons between PCOS phenotypes</p> <p>Data on physical activity not collected</p>

(Continued)

TABLE 1 (Continued)

Study (reference)	Sample, assessments used	Outcomes ²	Limitations
Altieri et al. 2013 (40)	<p>Groups: $n = 100$, PCOS; $n = 100$, controls</p> <p>Age: 27.7 ± 5.2 y, PCOS; 28.4 ± 5.8 y, controls</p> <p>BMI (kg/m^2): 34.7 ± 5.5, PCOS; 34.8 ± 5.4, controls</p> <p>Location: Bologna, Italy</p> <p>Race: not reported</p> <p>PCOS definition: 2 of 3 symptoms: 1) oligoamenorrhea, 2) hirsutism and/or hyperandrogenemia, 3) polycystic ovaries</p> <p>Exclusion criteria: endocrine or metabolic disorders; medications that influence appetite, reproduction, glucose, or lipid concentrations; psychoactive drugs; eating disorders; intensive lifestyle interventions</p> <p>Assessments: 7-d food records</p>	<p>Greater daily nutrient intake in PCOS vs. control groups*: fiber (g/d)</p> <p>Lower daily nutrient intake in PCOS vs. control groups*: lipids (% of energy/d)</p> <p>Greater starchy sweets (g/d), cheese (g/d), oil (g/d) in PCOS vs. control groups*</p> <p>Lower cooking fats (g/d) in PCOS vs. control groups*</p> <p>Positive associations between hormones and nutrients in PCOS*: A4 and total energy, A4 and protein (g/d), A4 and cholesterol (mg/d)</p> <p>Negative associations between hormones and nutrients in PCOS*: SHBG and total energy/d, SHBG and CHO (g/d), SHBG and CHO (% of energy/d), SHBG and oligosaccharides (g/d)</p> <p>Greater daily nutrient intake in PCOS vs. control groups*: total energy/d, fiber (g/d), folate ($\mu\text{g}/\text{d}$), iron (mg/d), magnesium (mg/d), phosphorus (mg/d), vitamin E (mg/d), sodium (mg/d)³, zinc (mg/d)³, calcium (mg/d)³, potassium (mg/d)³, niacin (mg/d)³</p> <p>Lower daily nutrient intake in PCOS vs. control groups*: SFAs (% of energy/d), glycemic index, retinol ($\mu\text{g}/\text{d}$)</p> <p>PCOS group reported higher diet quality than control group</p> <p>PCOS group intake vs. U.S. DRI*: above: SFAs (% of energy)</p> <p>No differences in self-reported physical activity between PCOS and control groups</p> <p>PCOS group reported greater amount of sitting time compared with controls*</p> <p>Greater daily nutrient intake in PCOS vs. control groups*: total energy/d, glycemic index³, glycemic load³, sodium (mg/d)³</p> <p>Greater energy intake/d and glycemic index diet between classic PCOS ($n = 39$) and control ($n = 44$) groups*</p> <p>Higher glycemic index diet between classic PCOS ($n = 39$) and ovulatory PCOS ($n = 22$) groups*</p> <p>No differences in total energy intake and glycemic index diet between ovulatory PCOS ($n = 22$) and control ($n = 44$) groups</p> <p>No differences in physical activity between PCOS and control groups</p>	<p>Diagnostic criteria used yielded a heterogeneous PCOS group</p> <p>Data on physical activity not collected</p> <p>Did not compare intake with EFSA-recommended intake, which is established for European countries</p>
Moran et al. 2013 (44)	<p>Groups: $n = 409$, PCOS; $n = 7057$, controls</p> <p>Age³: 33.5 ± 1.4 y, PCOS; 33.7 ± 1.5 y, controls</p> <p>BMI (kg/m^2): 29.3 ± 7.5, PCOS; 25.6 ± 5.8, controls</p> <p>Location: Australia (national survey)</p> <p>Race: not reported</p> <p>PCOS definition: not provided</p> <p>No specific exclusion criteria were applied</p> <p>Assessments: FFQ, physical activity 1-wk recall</p>	<p>Recruitment based on self-reported diagnosis of PCOS</p> <p>Control group may contain undiagnosed women with PCOS</p> <p>No reported exclusion criteria on medications that may influence weight, appetite, or reproduction</p> <p>Groups included women who are pregnant women and using hormones</p> <p>Study groups not matched for age or BMI</p> <p>Did not compare intake with Australian nutrient reference values</p>	<p>Recruitment based on self-reported diagnosis of PCOS</p> <p>Control group may contain undiagnosed women with PCOS</p> <p>No reported exclusion criteria on medications that may influence weight, appetite, or reproduction</p> <p>Groups included women who are pregnant women and using hormones</p> <p>Study groups not matched for age or BMI</p> <p>Did not compare intake with Australian nutrient reference values</p>
Graff et al. 2013 (46)	<p>Groups $n = 61$, PCOS; $n = 44$, controls</p> <p>Age: 22.7 ± 6.2 y, PCOS; 25.0 ± 6.3 y, controls</p> <p>BMI (kg/m^2): 28.9 ± 5.6, PCOS; 27.1 ± 5.7, controls</p> <p>Location: Porto Alegre, Brazil</p> <p>Race: Caucasian: 88% of sample; African-European: 12% of sample</p> <p>PCOS definition: 1) classic PCOS: oligoamenorrhea, hirsutism and/or hyperandrogenemia with or without polycystic ovaries; 2) ovulatory PCOS: hirsutism and polycystic ovaries in the presence of regular menstrual cycles and normal androgens</p> <p>Exclusion criteria: diabetes, medications that alter hormone concentrations, pregnancy, BMI ≥ 40 kg/m^2</p> <p>Assessments: FFQ, 6-d pedometer use</p>	<p>Diagnostic criteria used yielded a heterogeneous PCOS group</p> <p>Included both adolescents and adults with PCOS</p> <p>No reported exclusion criteria on medications that may influence weight and appetite</p> <p>Power analysis not provided for post hoc comparisons between PCOS phenotypes</p> <p>Pedometer may not comprehensively capture physical activity data</p> <p>Did not report energy expenditure or energy balance</p>	<p>Diagnostic criteria used yielded a heterogeneous PCOS group</p> <p>Included both adolescents and adults with PCOS</p> <p>No reported exclusion criteria on medications that may influence weight and appetite</p> <p>Power analysis not provided for post hoc comparisons between PCOS phenotypes</p> <p>Pedometer may not comprehensively capture physical activity data</p> <p>Did not report energy expenditure or energy balance</p>

(Continued)

TABLE 1 (Continued)

Study (reference)	Sample, assessments used	Outcomes ²	Limitations
Ahmadi et al. 2013 (45)	<p>Groups: $n = 65$, PCOS; $n = 65$, controls</p> <p>Age: 25.1 ± 6.1 y; PCOS; 26.1 ± 6.5 y; controls</p> <p>BMI (kg/m^2): 23.4 ± 3.6, PCOS; 23.1 ± 3.8, controls</p> <p>Location: Shiraz, Iran</p> <p>Race not reported</p> <p>PCOS definition: 2 of 3 symptoms: 1) oligomenorrhea, 2) hirsutism and/or hyperandrogenemia, 3) polycystic ovaries</p> <p>Exclusion criteria: liver, kidney, and heart disease; hormone use; medications that influence metabolism or body composition; incomplete FFC; implausible energy intake; and intensive lifestyle interventions</p> <p>Assessments: Three 24-h recalls (2 weekdays, 1 weekend day), exercise habits assessed by using demographic questionnaire</p>	<p>Greater daily nutrient intake in PCOS vs. control groups*:</p> <p>total kcal/d, fat (% of energy/d), SFAs (g/d), PUFAs (g/d)</p> <p>No significant self-reported physical activity differences between PCOS and control groups</p> <p>No differences in daily nutrient intake between normal weight ($n = 49$) and overweight ($n = 16$) PCOS groups</p>	<p>Diagnostic criteria used yielded a heterogeneous PCOS group</p> <p>Power analysis not provided for post hoc comparisons among PCOS groups</p> <p>Details on physical activity assessment tool not reported</p> <p>Did not report energy expenditure or energy balance</p>

¹ $^{*}p < 0.05$. A4, androstenedione; CHO, carbohydrate; EFSA, European Food Safety Authority; FSH, follicle-stimulating hormone; LH, luteinizing hormone; PCOS, polycystic ovary syndrome; SHBG, sex hormone-binding globulin.

² Only data pertaining to diet and/or physical activity are reported.

³ NS after adjustment for energy intake or age and BMI.

weight gain. In the higher BMI categories, Wright et al. noted no differences in overall macronutrient and energy intake among overweight and obese women with PCOS compared with their respective BMI-matched controls. Overweight women with PCOS reported lower intakes of milk products compared with overweight controls, whereas obese women with PCOS reported consuming more servings of meat, fish, poultry, and eggs than the obese controls. Most of these differences were less than 1 serving apart. This may or may not be considered clinically significant, depending on the type of protein consumed.

By using the 7-d food records from a large cohort of women ($n = 198$) with a self-reported PCOS diagnosis, Barr et al. (43) reported that women with PCOS in the United Kingdom had higher total energy intakes (~350 kcal) compared with a reference population. They noted that women with PCOS consumed higher amounts of total carbohydrates (229.0 vs. 198.0 g), protein (78.0 vs. 66.3 g), dietary fat (85.0 vs. 61.1 g), saturated fat (26.5 vs. 22.2 g), monounsaturated fat (29.7 vs. 21.7 g), polyunsaturated fat (16.2 vs. 12.6 g), sugar (102.0 vs. 87.4 g), and dietary fiber (16.5 vs. 13.0 grams) compared with a reference population (43). On the basis of these results, it can be recommended that sugar intake should be monitored when conducting dietary assessments in patients with PCOS in the United Kingdom. Barr et al. also reported that overweight women with PCOS consumed higher-glycemic-index diets compared with normal weight women with PCOS. These findings were consistent with reports from Australia involving a cohort of women ($n = 409$) with a self-reported diagnosis of PCOS (44). Moran et al. (44) noted a small, but statistically significant difference in total daily energy intake (~50 kcal) between women with PCOS and controls on the basis of a validated FFQ. The PCOS group consumed higher amounts of iron (12.3 vs. 11.6 mg), magnesium (272 vs. 258 mg), phosphorus (1471 vs. 1401 mg), and vitamin E (5.9 vs. 5.6 mg) when adjusted for total daily energy intake and lower amounts of saturated fat (15.1% vs. 15.4% of energy) and retinol (295 vs. 311 μg). Although the studies by Barr et al. (43) and Moran et al. (44) represent the largest studies that assessed baseline dietary intake in PCOS to date, both were limited by their reliance on a self-reported diagnosis of PCOS. It is possible that the control populations contained women with PCOS and/or other endocrine issues because Barr et al. did not exclude PCOS features from their control population survey. Moran et al. used a diagnostic question within a survey that restricted PCOS diagnosis and treatment to within 3 y. This may have classified women with PCOS who were diagnosed earlier in their lives or not seeking treatment as controls. Collectively, there is the potential for differences in dietary intake between groups to be underestimated by these studies.

Higher energy diets were also reported in Iranian (45) and Brazilian (46) women with PCOS. Ahmadi et al. (45) compared the 3-d, 24-h dietary recalls of Iranian women with and without PCOS and noted that overall daily energy intake was higher (~300 kcal) in women with PCOS. Iranian

women with PCOS also reported higher total fat (~2% kcal), polyunsaturated fat (0.6 g), and saturated fat (0.8 g) intakes compared with controls. This was contradicted by Altieri et al. (40), who reported that the Italian PCOS group consumed a lower-fat diet (~1% kcal) compared with healthy controls. The 24-h dietary recall used by Ahmadi et al. has similar disadvantages to a FFQ because it relies on participant memory. However, it is a convenient method that can provide accurate dietary information when collected by a trained interviewer using standardized approaches. Similar to Ahmadi et al., Graff et al. (46) reported that Brazilian women with PCOS had higher total daily energy intakes (~250 kcal) compared with controls with the use of an FFQ. Brazilian women with PCOS reported consuming a higher-glycemic-index (2 units), glycemic load (~33 units), and sodium (~430 mg) diet. However, these differences disappeared after adjusting for age and BMI. Graff et al. (46) recognized the heterogeneous composition of their PCOS population and performed an assessment of dietary intake on the basis of PCOS phenotypes. They found that women with a classical form of PCOS (i.e., chronic anovulation and hyperandrogenism), but not those with ovulatory PCOS (i.e., hyperandrogenism, polycystic ovaries but regular menstrual cycles), had significantly higher total daily energy intake compared with controls. These differences became negligible after adjusting for age and BMI.

Last, Tsai et al. (47) investigated baseline dietary intake in Taiwanese women with PCOS by using 3-d food records. Taiwanese women with PCOS reported lower total daily energy intakes (110 kcal) compared with infertile women without PCOS. The PCOS group consumed more total dietary fat (~3% of energy) but lower amounts of total daily carbohydrates (~4% of energy; 30 g), which likely accounted for the energy difference between groups. Comparing the results of this study with others is challenging because groups were not matched for BMI and their control population comprised infertile women (including those with unexplained infertility).

Comparison with national dietary guidelines. Six of the 10 studies compared nutrient intake in women with PCOS with established dietary guidelines (35,36,41–43,48). Wright et al. (36) noted that women with PCOS in the United States had slightly higher carbohydrate and lower fat intakes compared with the dietary recommendations for insulin-resistant individuals established by Reaven (49) (i.e., diet consisting of 45% carbohydrates, 15% protein, 10% polyunsaturated fat, 20% monounsaturated fat, and <10% saturated fat). The Reaven recommendations may not be an optimal comparator for this population because certain PCOS phenotypes may not be prone to insulin resistance (50) and the low carbohydrate recommendation may be difficult to achieve in a free-living setting. When compared with the 2010 *Dietary Guidelines for Americans* (51), women with PCOS in the United States consumed excessive saturated fat (12% of total daily energy intake vs. <10% of total daily energy intake). The PCOS group consumed amounts

within the Acceptable Macronutrient Distribution Ranges for carbohydrate and protein (51), which was similar to the results of the U.S. study conducted by Douglas et al. (35). Douglas et al. (35) determined that the PCOS group consumed more than the recommended amount of saturated fat as established by the National Cholesterol Education Program (<7% kcal/d). The PCOS group also exceeded American Heart Association recommendations for sodium (≤ 2400 mg/d) and did not meet dietary fiber recommendations (25–30 g/d). When compared with the 2010 Dietary Guidelines, their reported values are consistent with the conclusion that American women with PCOS consume excessive sodium and insufficient fiber in their diets (35,51). When the PCOS group was stratified by BMI, normal weight and obese women with PCOS exceeded dietary fat Acceptable Macronutrient Distribution Range recommendations by 2% and 5%, respectively, whereas overweight women with PCOS consumed within the normal range (36). This emphasizes the importance of accounting for BMI when assessing baseline nutrient intake within the PCOS population.

Barr et al. (43) used the UK's Reference Nutrient Intake (RNI) guidelines to determine whether their PCOS group met dietary guidelines. On the basis of the results, women with PCOS exceeded the reference intakes for fat (i.e., total fat, saturated fat, polyunsaturated fat) and mean dietary glycemic index but did not meet fiber recommendations. The reported values also indicated that women with PCOS in the United Kingdom consumed more protein and but did not meet carbohydrate recommendations. The RNI established in the United Kingdom may not be an appropriate measure to determine nutrient adequacy (52). There is significant potential to overestimate the percentage of women with PCOS who are not meeting dietary guidelines because the RNI values are defined as nutrient intakes required to meet the recommendations for 97.5% of a national population.

Álvarez-Blasco et al. (42), Toscani et al. (41), and Moran et al. (44) used DRIs established in the United States to assess nutrient intake in Spanish, Brazilian, and Australian populations, respectively (Table 1). The dietary recommendations designed to meet the needs of the American population may not be a useful reference for countries that have different dietary patterns, food environment, and cultural beliefs and the potential for genetic variations in metabolism. The European Food Safety Association has established dietary reference values for the intake of carbohydrates, fats, and water that are likely more appropriate for European countries, including Spain (53). Similarly, the Australian National Health and Medical Research Council and the New Zealand Ministry of Health have established nutrient reference values specifically for the Australian and New Zealand populations (54). To the best of our knowledge, there are no established South American nutrient value recommendations.

When comparing the dietary intake results with the corresponding national dietary guidelines, we concluded that Spanish, British, and Australian women with PCOS exceeded the recommended intakes for total, saturated, and/or

monounsaturated fats when compared with women without PCOS (42–44). Álvarez-Blasco et al. (42) reported that women with PCOS in Spain exceeded the U.S. recommended dietary cholesterol intake, while not meeting the fiber, potassium, and vitamin E recommendations. Both Spanish (42) and Italian (40) women with PCOS consumed excessive total fat but inadequate fiber when compared with European Food Safety Association recommendations. Similarly, Australian women with PCOS had inadequate fiber and vitamin E intakes compared with the Australian nutrient reference values (44). The global data indicate that women with PCOS exceed total fat and saturated fat recommendations, while not meeting recommended amounts of dietary fiber in their diet. Women without PCOS included in these studies appear to have similar results when comparing nutrient intake to national nutrient reference values across countries. Meeting nutrient recommendations may be a key public health issue for clinicians and researchers to resolve across both PCOS and non-PCOS populations.

Biomarkers and diet. Two of the studies included in this review determined associations between biochemical markers and nutrients (Table 1). Tsai et al. (47) reported a positive association between carbohydrate intake (g and % of energy) and follicle-stimulating hormone. Follicle-stimulating hormone is a hormone that promotes follicular growth in the ovaries and is typically lower compared with its companion gonadotropin (luteinizing hormone) in a subset of women with PCOS (55,56). Altieri et al. (40) observed positive associations of total energy, protein (g), and cholesterol intakes with androstenedione (a precursor of testosterone). Although androstenedione is not a diagnostic marker of PCOS, a subgroup of women with PCOS exhibit elevated concentrations of this androgen (57). Collectively, these data are consistent with the hypothesis that PCOS symptoms may be related to dietary intake. Further research is needed to corroborate these findings and to determine the physiologic mechanisms behind these associations.

Physical activity. Six of the 10 studies performed an evaluation of physical activity levels in women with PCOS (36,42–46). By using a validated physical activity questionnaire, Wright et al. (36) did not detect any differences in self-reported physical activity levels between American women with PCOS and healthy controls. Both the PCOS and control groups reported similar amounts of time engaged in various activities, including vigorous, moderate, and light activity, as well as sleeping or reclining, for typical weekdays or weekend days. These findings were consistent with those of Álvarez-Blasco et al. (42), Ahmadi et al. (45), and Graff et al. (46), who also noted no differences in overall physical activity among Spanish, Iranian, and Brazilian women with or without PCOS. Wright et al. (36) noted that women with PCOS reported greater sitting time. Unlike the findings in an Australian cohort (44), this difference did not reach significance ($P = 0.064$). Wright et al. (36) did not detect differences in physical activity among

PCOS and control groups when the data were analyzed by BMI categories (i.e., normal weight, overweight, and obese).

By using 7-d activity records, Barr et al. (43) showed that the majority of women with PCOS (74%) in their UK study reported achieving 30 min of daily moderate-intensity activity. This was consistent with the UK national recommendations for healthy living (58). Approximately half of the overweight and obese women with PCOS did not achieve the recommended 60 min of daily moderate-intensity activity (43). The authors admit that a self-selected sample might not have yielded a representative sample of women with PCOS because their approach may have overestimated physical activity due to the inclusion of highly motivated individuals. This study did not include comparisons with healthy age- and BMI-matched controls. Rather, Barr et al. (43) examined the potential for differences in physical activity among normal weight and overweight women with PCOS. They noted that normal weight women with PCOS reported longer durations of moderate-intensity physical activity compared with overweight and obese women with PCOS of the same age. Coupled with their findings of lower-glycemic-index diets in normal weight women with PCOS, this study supported that diet and physical activity behaviors were associated with BMI among women with PCOS.

The emerging data on baseline physical activity levels in women with PCOS are challenging to interpret because none of the studies used the same physical activity evaluation tool. The limitations for the methods used merit consideration. Wright et al. (36) used the Paffenbarger Physical Activity Questionnaire, which has been primarily validated in mixed-gender populations (59). It is uncertain whether this tool provides an accurate measure of physical activity for pre- and perimenopausal women with PCOS. Álvarez-Blasco et al. (42) and Ahmadi et al. (45) evaluated physical activity on the basis of an assessment of exercise habits by using interview questions. The validity of this approach is uncertain because the details regarding the validation of their interview tools were not provided. The 7-d activity records used by Barr et al. may be considered a more accurate quantification of physical activity since because is minimal dependence on memory, in contrast to the 7-d self-recall implemented in the Australian study (43). However, 7-d activity records place greater burden on participants, which can impact the reporting accuracy. Graff et al. (46) was the only research group to eliminate recall bias and use pedometers, which objectively quantified 6 d of baseline physical activity among participants. Graff et al. reported no difference in physical activity levels between women with or without PCOS. Although these data are strengthened by their inclusion of an objective measure of physical activity, we are unaware of any validation study on the pedometer model that was used. Moreover, pedometers may have low accuracy when assessing energy expenditure (60). Future studies would benefit from a combination of objective and subjective instruments in the situation that the objective tool may malfunction in the field. Information on perceptions of physical activity

may also have relevance when used in conjunction with objective measures.

Summary and Future Recommendations

It is important to recognize that studies assessing diet and physical activity of women with PCOS used broad definitions for PCOS. This creates a challenge when interpreting the literature because the PCOS group comprise of several distinct clinical phenotypes. Most research groups used criteria supported by the American Society for Reproductive Medicine and the European Society of Human Reproduction and Embryology, known as the Rotterdam criteria (61), which yield heterogeneous PCOS phenotypes. Hormonal and metabolic differences exist among these clinical phenotypes, which may serve as confounding factors when examining lifestyle variables (62,63). As Graff et al. (46) demonstrated, there may be distinct differences in dietary intake among clinical phenotypes of PCOS. This is consistent with repeated reports that women with milder variants of PCOS have improved metabolic status and different health risks compared with those with more severe phenotypes (64,65). Researchers must establish a clear distinction between PCOS status to provide an accurate comparison of lifestyle habits between women with and without clinical variants of PCOS.

Energy balance is an important determinant of weight that has not been adequately explored in women with PCOS. Few studies performed concomitant assessments of physical activity when examining dietary intake in women with PCOS. Future studies would be strengthened by the addition of objective tools to measure physical activity (e.g., accelerometers), which can provide an unbiased account of energy expenditure. Although there are emerging data on the associations between biochemical markers and dietary intake, more of these analyses are needed in PCOS populations to develop hypotheses related to potential predictors of dietary intake and physical activity in women with PCOS. Dietary interventions featuring weight loss were shown to have a positive effect on reproductive outcomes (27,30,31,48). However, methods to maintain weight loss should be further examined. Researchers should consider the interaction between environmental influences, personal beliefs, and biological variables in women with PCOS to fully understand drivers of diet and physical activity behaviors. Experts have suggested that depression and/or low self-esteem place women with PCOS at higher risk of emotional eating and decreased exercise, which contribute to a long-term positive energy balance and weight gain (66). By examining and understanding these associations, it may be possible to identify potential key intervention targets with a high likelihood for success in the PCOS population. The roles of race and ethnicity also merit further consideration. There is existing evidence supporting racial disparities in reproductive function among women with PCOS (67,68). Because only a few studies disclosed the race of their participants, we were unable to draw any conclusions regarding any potential influence of race on dietary intake or physical activity.

Conclusions

This review is the first to our knowledge to summarize the literature on baseline dietary intake and physical activity in women with and without PCOS and to provide recommendations to strengthen research within this area. There are emerging global data that women with PCOS have different baseline dietary intakes compared with women without PCOS. Although the limited number of studies in the United States suggest that dietary intake is similar to that of women without PCOS irrespective of BMI (35,36), both studies recommend that diet and its effect on metabolic outcomes be more thoroughly examined in this population. These recommendations were based on the observation that differences existed in the consumption of certain foods among women with PCOS (e.g., high glycemic index), despite similarities in overall energy or nutrient intake. Moreover, notable differences in dietary intake were evident in women with PCOS when BMI was taken into consideration. Internationally, most studies indicate higher energy intakes in women with PCOS, with excessive saturated fat and inadequate fiber consumption. However, there appears to be no significant differences in self-reported physical activity between women with and without PCOS. The use of objective tools may be the next step to determine energy expenditure in this population. Moving forward, we recommend that researchers incorporate life stage and clinical phenotypes into their analysis when examining baseline dietary intake and physical activity in the PCOS population. Larger sample sizes with sufficient power to discern the impact of BMI and clinical phenotype will also serve to strengthen future studies.

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