IODINE DEFICIENCY AND RISK OF PCOS IN ADOLESCENTS: A POPULATION-BASED STUDY OF PREVALENCE, MENSTRUAL IRREGULARITIES, AND THYROID DYSFUNCTION

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Abstract. This thesis presents a population-based study framework for assessing PCOS prevalence and clinical–endocrine phenotypes in iodine-deficient regions. It integrates WHO-recommended iodine status indicators with adolescent-appropriate PCOS diagnostics, proposes practical sampling and measurement strategies, and outlines analytic approaches to disentangle associations among iodine nutrition, thyroid dysfunction, menstrual irregularities, and PCOS risk without inflating prevalence through pubertal overlap.
Keywords: adolescent health; polycystic ovary syndrome; iodine deficiency; urinary iodine concentration; thyroid dysfunction.

INTRODUCTION

Adolescence is the “noisiest” phase of reproductive endocrinology: cycles are stabilizing, ovarian follicle dynamics are active, acne is common, and weight trajectories can change rapidly. In that physiological background, PCOS is easy to over-call and just as easy to miss. The problem is not simply academic—labeling a teenager with a chronic syndrome can shape self-image, medical trajectories, and treatment exposure, while delayed recognition can allow metabolic risk and distress to accumulate. For this reason, the 2023 International Evidence-Based Guideline places strong emphasis on adolescent-specific diagnostic rules, including the requirement for both persistent ovulatory/menstrual dysfunction and hyperandrogenism, and the explicit recommendation that pelvic ultrasound (for polycystic ovarian morphology) and AMH should not be used to diagnose PCOS in adolescents [1].

MAIN PART

At the same time, environmental and nutritional endocrinology can reshape the clinical landscape in which PCOS is assessed. Iodine deficiency remains a public-health concern in multiple regions, especially those far from marine food sources and where iodized salt coverage is incomplete. WHO describes iodine status at the population

level using median UIC; for school-age children (≥6 years), a median UIC of 100–199 μg/L indicates adequate iodine intake, while a median <100 μg/L indicates insufficient intake (with <20, 20–49, and 50–99 μg/L reflecting severe, moderate, and mild deficiency categories, respectively) [2].

Population estimates of adolescent PCOS prevalence vary widely across studies primarily because diagnostic criteria differ. The most recent global meta-analysis designed specifically for adolescents shows that prevalence shifts substantially depending on whether the original Rotterdam criteria are used (which include polycystic ovarian morphology) or whether the International Evidence-Based Guideline criteria are applied (which are more conservative for adolescents). In meta-analysis across 20 studies (n≈14,010), global prevalence was 9.8% by original Rotterdam criteria versus 6.3% by guideline criteria, with the authors explicitly noting that excluding ovarian morphology—overlapping with normal puberty—should deter over-diagnosis. This difference is not a technical footnote; it fundamentally changes how many adolescents are counted as having PCOS in community surveys and, consequently, how health systems interpret "burden."

Iodine deficiency is best understood as a population exposure that shapes thyroid hormone synthesis capacity. WHO's iodine deficiency indicators (median UIC and the distribution of UIC below key thresholds) allow regions to be categorized reliably for public-health monitoring. For PCOS epidemiology, iodine deficiency becomes relevant because thyroid dysfunction can affect menstrual regularity and general endocrine-metabolic status, thereby complicating attribution of menstrual disturbance to PCOS. In adolescents with menstrual disorders, thyroid dysfunction can be substantially more common than in adolescents without menstrual disorders; a case–control study reported thyroid dysfunction in 13.6% of girls with menstrual disorders compared with 3.5% in controls, alongside differences in biochemical hyperandrogenism. These data do not prove that iodine deficiency increases PCOS prevalence, but they do demonstrate that thyroid dysfunction is a nontrivial contributor to menstrual pathology in adolescence and must be incorporated into diagnostic pathways—especially in settings where iodine deficiency may elevate thyroid-related problems [3].

Therefore, a population-based study in an iodine-deficient region should treat thyroid dysfunction as both (1) an exclusion diagnosis when defining PCOS cases (to avoid misclassification), and (2) a phenotype modifier potentially influencing severity, symptom combinations, and healthcare-seeking patterns. If thyroid dysfunction is not measured, a study risks inflating "PCOS prevalence" simply by counting thyroid-related menstrual irregularity as PCOS-like ovulatory dysfunction [4].

CONCLUSION

Estimating PCOS prevalence in adolescents requires diagnostic restraint because puberty naturally reproduces some PCOS-like features. The 2023 International Evidence-Based Guideline and adolescent recommendations provide a clear framework: diagnose adolescent PCOS only when both persistent ovulatory/menstrual dysfunction and hyperandrogenism are present, and avoid pelvic ultrasound and AMH for diagnosis in adolescents to reduce overdiagnosis. Consistent with this, the strongest adolescent meta-analytic evidence shows that global prevalence differs markedly by criteria: original Rotterdam criteria yield higher estimates (about 9.8%) than guideline-aligned criteria (about 6.3%), supporting the rationale for stricter adolescent definitions.

In iodine-deficient regions, thyroid dysfunction becomes a pivotal contextual factor, both as a confounder of menstrual irregularity and as a phenotype modifier that can reshape symptom patterns and healthcare needs. Population iodine status should be defined using WHO urinary iodine indicators (median UIC and deficiency categories), rather than assumptions about geography, to ensure valid exposure classification.

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