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Exploring the physiological barriers to weight management in women with polycystic ovary syndrome: a scoping review

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Polycystic ovary syndrome (PCOS) is a common endocrine condition occurring in 8–13% of reproductive-aged women associated with reproductive, metabolic and psychological dysfunction.[1,2] Higher rates of weight gain and obesity have been observed in PCOS which exacerbate its features. Weight management is a first-line strategy for the management of PCOS. Mechanistic reasons for increased weight gain remain unclear but may include dysregulation of intrinsic factors such as energy homeostasis. The aim of this study is to examine the extent of current research evaluating the physiological barriers to weight management in women with PCOS and generate recommendations for future research. A scoping systematic literature search was performed using MEDLINE, EMBASE, PsycInfo, AMED, CINAHL and Cochrane Central Register of Controlled Trials up to June 2022. Search terms included physiological factors affecting energy intake (e.g. gastrointestinal appetite hormones, adipokines, subjective appetite, brain metabolism measured by functional brain imaging (fMRI), neuropeptides) and energy expenditure (e.g. total energy expenditure (TEE), resting energy expenditure (REE), metabolic flexibility) in women with PCOS. Abstracts, non-English language and reviews were excluded. A total of $n = 55$ energy intake and $n = 23$ energy expenditure papers were included. For the energy intake papers, we focused specifically on studies assessing the impact of food, nutrient or supplements on these outcomes to better capture the accurate relationship with energy homeostasis (papers with multiple outcomes were counted as independent studies). Energy intake studies ($n = 91$ were grouped into broad outcomes of gastrointestinal appetite hormones ($n = 44$), adipokines ($n = 35$), subjective appetite ($n = 9$), fMRI ($n = 3$) and neuropeptides ($n = 0$). Energy expenditure studies ($n = 29$) were grouped into TEE ($n = 1$), REE ($n = 15$), meal induced thermogenesis ($n = 3$), nutrient oxidation ($n = 6$) and metabolic flexibility ($n = 4$). Across both energy intake and expenditure papers, sixty percent of the studies compared outcome responses in women with PCOS to a control group. Results were inconsistent with 57% reporting no differences and 43% reporting altered responses in PCOS compared with controls, including blunted appetite hormone responses, metabolic inflexibility and reduced energy expenditure, which all may be potential barriers to weight loss. An additional 456 papers with relevant outcomes but outside the context of energy homeostasis were identified and grouped into the categories of cardiometabolic risk, receptors and gene expression, medication and pharmaceuticals, fasting measures and reproduction, highlighting the complex interplay of these systems. This comprehensive review has identified several priorities for further research, firstly in primary studies where research is limited such as TEE, fMRI and neuropeptides and secondly where systematic reviews and meta-analyses could be conducted to synthesise the current evidence including REE and gastrointestinal hormones. Addressing these research gaps will contribute significantly to understanding the aetiology of weight gain and obesity in women with PCOS and assist with informing future interventions.

References

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