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Phytotherapy for Polycystic Ovarian Syndrome: A review of the literature and evaluation of practitioners' experiences

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ABSTRACT

Background: Polycystic Ovarian Syndrome (PCOS) is a common, complex, endocrine condition with potential long-term cardiovascular, endocrine and metabolic health implications. Currently, there exists no medical treatment that addresses all clinical presentations. Herbal practitioners often treat women with PCOS; yet, there is a lack of research investigating PCOS and herbal treatment. This study aimed to examine the current scientific literature on PCOS and phytotherapy, explore practitioners' experiences treating women with the syndrome and evaluate whether there is a role for phytotherapy in the treatment of PCOS.

Methods: A literature search was conducted using the terms; 'Polycystic Ovarian Syndrome'/'PCOS' and 'phytotherapy'/'herbal medicine'/'herbs'. The published research identified by these terms was then reviewed. A brief questionnaire with a mix of eleven open and closed questions was sent to herbal practitioners on the National Institute of Medical Herbalists (NIMH) 2012 register.

Results: The literature review found promising results for the use of *Mentha spicata*, *Cinnamomum verum* and berberine containing herbs. There was a weaker evidence base for the use of *Glycyrrhiza glabra* and *Paeonia lactiflora*. The questionnaire was completed by 72 practitioners, 71% had treated women with PCOS. The majority (38%, $n = 50$) felt herbal medicine was 'quite successful' in treating PCOS. The average time until herbal treatment started to be successful was 3.2 months. The most frequently used herbs were *P. lactiflora*, *G. glabra* and *Vitex agnus castus*. Dietary and lifestyle advice emerged as frequently identified therapeutic interventions.

Conclusions: This exploratory study suggests that there is a role for phytotherapy in the treatment of PCOS. Further investigation, consisting of well-designed clinical trials and monitoring the successful use of herbal medicine by practitioners, is clearly necessary. This future research would serve to substantiate positive empirical evidence, constructing a more robust evidence base for the effective use of phytotherapy as a therapeutic option for women with PCOS.

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1. Introduction

'Polycystic ovary syndrome is a frustrating experience for women, often complex for managing clinicians and a scientific challenge for researchers' (Teede et al., 2010, p. 1).

PCOS was first identified by Irving Stein and Michael Leventhal in 1935, whilst conducting a case study of seven women exhibiting the collection of symptoms of hirsutism, obesity, amenorrhea, and enlarged ovaries. Today, PCOS is recognized as a complex, heterogeneous, endocrine and metabolic condition with multiple aetiologies and clinical presentations with long term health implications. Although the exact aetiology is unknown, insulin resistance and compensatory hyperinsulinemia are now understood to play key roles (Dunaif, 1997).

Definition and diagnosis: PCOS is one of the most common endocrine disorders in women of reproductive age. However, its definition and diagnosis remains controversial. The prevalence of PCOS is dependent on which definition is used; the National Institute of Health (NIH, 1990) at 6% or the Rotterdam, 2013 at 15% (Fauser et al., 2012).

At present, the Rotterdam definition is the most widely accepted stating that women with PCOS will have a combination of **two** of the following:

- Chronic anovulation
- Clinical and/or biochemical signs of hyperandrogenism
- Polycystic ovaries on ultra sound with the exclusion of other androgen excess or related disorders. These may include hyperprolactinema; abnormal thyroid function; non classical 2-hydroxylase deficiency and androgen secreting tumours.[1]

The NIH definition is the strictest, whilst the Rotterdam incorporates a broader spectrum of symptoms, notably polycystic morphology and the inclusion of women with hirsutism and regular menstruation. The Androgen and PCOS Society (Azziz et al., 2006) have proposed a further definition which concentrates on androgen excess. Of note is the fact that the choice of definition used subsequently affects the validity of the outcomes of clinical trials and research analysis (Welt, 2006; Di Marcantonio, 2008).

Standard biochemical tests to assist in the diagnosis of PCOS measure total testosterone, free testosterone, dehydroepionadrosterone (DHEA), glucose tolerance, insulin sensitivity, cholesterol, thyroid hormones and prolactin. Sex hormone binding globulin (SHBG) is sometimes tested. Historically, luteinising hormone (LH) and follicle stimulating hormone (FSH) ratios were tested but are rarely used now due to their poor specificity (Dunaif, 1997).

[1] Chronic anovulation can be defined as absence of ovulation. Clinical signs of hyperandrogenism may include hirsutism, alopecia, acanthosis nigricans and acne. Biochemical signs of hyperandrogenism include raised levels of androgens. Polycystic ovaries are defined as presence of 12 or more follicles in each ovary measuring 2 ± 9 mm in diameter, and/or increased ovarian volume (>10 ml).

Recently there have been calls to change the name of PCOS as it has been perceived as confusing to both women with the syndrome and healthcare providers (Dunaif and Fauser, 2013). It has been proposed to re-name the syndrome to reflect the broader clinical significance and enhance recognition of its multi-faceted features (Teede et al., 2014

Aetiology: The aetiology of PCOS remains elusive as currently there are no specific genetic or metabolic markers. However, there is evidence of a genetic link in first degree female relatives (Kahsar-Miller et al., 2001) and that physical and environmental factors, such as obesity, nutrition and pollutants may be influential (Kandaraki et al., 2009).

Pathophysiology: The pathophysiology is not well established. Different ideas on its development may well be interlinked:

- Metabolic derangements leading to insulin resistance and hyperandrogenism.
- A primary neuroendocrine defect leading to elevated LH, Gonadotrophin releasing hormone (GnRH) pulse frequency and amplitude.
- Inflammation and role of adipose tissue: adipokines, adiponectin and resistin, hormones that are secreted by adipose tissue have been indicated in the pathogenesis of PCOS (Carmina et al., 2005).

Insulin resistance resulting in hyperinsulinemia was first identified by Burghen et al. (1980) as playing a key role in the pathogenesis of PCOS. Dunaif (1997) developed this finding to show that insulin resistance has a variety of circulatory, metabolic, hormonal and inflammatory consequences on the body. Hyperinsulinemia is understood to be the pathogenic mechanism involved in the development of hyperandrogenism. High levels of insulin stimulate the ovaries and adrenals to over produce androgens. This results in the absence of ovulation and consequent hyperandrogenism. Hyperinsulinemia decreases the hepatic production of the sex hormone binding globulin, which in turn further increases the amounts of circulating androgens. Insulin resistance is present in skeletal muscle and adipose tissue. With PCOS, there are defects in both insulin action and secretion and the molecular mechanisms differ from those found in obesity and NIDDM (Dunaif, 1997). The GnRH frequency in the hypothalamus is higher than normal. This results in increased levels of LH relative to FSH. This hormonal imbalance further results in increased levels of androgens.

Clinical manifestations: PCOS is often multifactorial in terms of signs and symptoms and these may vary in character and intensity during the different stages of a woman's lifetime.

- **Anovulatory infertility**: 50–70% of women with anovulatory infertility have PCOS (Pangariuan 2011). Approximately 90%–95% of anovulatory women presenting to infertility clinics have PCOS (Sirmans and Pate, 2014). The economic burden of diagnosing and treating this is estimated to be up to £22 million annually in the UK (University of Nottingham PCOS Research Group, 2014).