### Founder Members

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<tr>
<th><strong>Dr. Duru Shah</strong></th>
<th><strong>Founder President, The PCOS Society, India</strong></th>
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<td>MD, FRCOG, FCPS, FICS, FICOG, FICMCH, DGO, DFF</td>
<td>Gynaecologist</td>
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<td><strong>President of the Federation of Obstetric &amp; Gynecological Societies of India (FOGSI) (2006)</strong></td>
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<td><strong>Dr. Uday Thanawala</strong></td>
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<td><strong>Vice President of the Indian Academy of Diabetes</strong></td>
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<td>MD, DNB, DGO, FRCOG</td>
<td>Gynaecologist</td>
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### Patrons, 2015

- **Dr. Abha Majumdar**
- **Dr. Kanthi Bansal**
- **Dr. Nalini Mahajan**
- **Dr. P. C. Mahapatra**
- **Dr. Sujata Kar**
- **Dr. Sujata Misra**
Dear Friends,

Managing PCOS in an adolescent or a young woman was dedicated to managing her symptoms. Today, it is based on evidence accumulated from very diligent and precise research. As science rapidly progresses, so does excellence in the art of this science progress. To highlight the various aspects of this very much needed disorder, the PCOS Society, India, has been initiated.

It is my pleasure to inform you that a new Society has been created termed ‘The PCOS Society’ (India), which will focus on the subject of PCOS. The Society is a multi-disciplinary society and has a mix of Gynecologists, Endocrinologists, Dermatologists and all health specialties which deal with PCOS patients.

The Launch of the Society was held on Thursday 6th August 2015, by our Chief Guest Prof. Anuja Dokras, the current President of the Androgen Excess and PCOS Society (AEP COS) and the Executive team consists of Dr. Shashank Joshi, Dr. Rekha Sheth, Dr. Piyathakkar, Dr. Sangeeta Agrawal, Dr. Uday Thanawala, Dr. Madhuri Patil and myself.

The Society plans to:
Conduct and organize lectures, seminars and meetings by inviting well-known educationists and experts in the field of PCOS, serve as a forum for the interchange of ideas between professionals from different scientific and clinical backgrounds, strive to stimulate evidence-based studies that will assist in developing our own Recommendations and Guidelines, provide a forum to collect and disseminate recent findings in PCOS research which have been either published or presented at meetings, which will be updated regularly and made available on the PCOS website and through our Newsletter, Pandora. Through the ‘PCOS Connect Program’ we aim to educate patients and the lay public about PCOS.

I would like to take this opportunity to profusely thank my colleagues who joined me as founder members to create this Society and others who have contributed by becoming Patrons and Members. I would also like to thank my team members for the support I have received towards developing the Newsletter, the forthcoming International Conference material and the PCOS Website (www.pcosindia.org | www.pcosindia.com). I sincerely thank USV for supporting the launch of our Society and for being our corporate partner for this year’s Newsletter.

As President of this Society, I extend to you an opportunity to join this Society and help to grow it into a very strong and creditable organization, serving the cause of the Polycystic Ovary Syndrome.

Dr. Duru Shah
Founder President, The PCOS Society, India

Email: thepcossociety@gmail.com

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I had the privilege of inaugurating the PCOS Society of India in August 2015. PCOS is the commonest endocrine disorder in reproductive age women, yes even more common than hypothyroidism. It can affect a woman through a greater part of her life – adolescence, adulthood and peri-menopause. The diagnosis is complex due to the heterogeneous phenotypes and a number of patients are dissatisfied with the difficulties with establishing an accurate diagnosis and the fragmented counselling they receive. The establishment of a Society dedicated to PCOS allows clinicians and researchers to focus their efforts to optimize diagnosis and develop new treatments for this disorder. There is urgent need to establish normograms for the diagnostic criteria of PCOS in the Indian population. Increasing awareness and ongoing education is critical to the mission of this Society. I am pleased to be the guest speaker at the first meeting to be held in June 2016. As President of the AE-PCOS Society, the theme during my tenure has been to focus on our patients. I therefore hope the Indian Society will also seek opportunities to increase awareness of PCOS amongst the general population allowing for early diagnosis and timely therapeutic interventions and more importantly, clarify misconceptions associated with PCOS. Dr. Duru Shah has brought together highly acclaimed specialists from different medical fields to lead this Society. I applaud her efforts and strongly encourage you to join this multi-disciplinary Society and actively participate in its mission with the goal of bringing PCOS awareness to all women in India.

Anuja Dokras
MD., PhD.
President, AE-PCOS Society, Director, Penn PCOS Center
Professor of Obstetrics Gynecology, University of Pennsylvania, Philadelphia, USA
How does PCOS lead to early and recurrent pregnancy

Can we recommend bariatric surgery to a young girl,

The Metabolic Syndrome

Hypothyroidism – is it associated with PCOS?

Assessing Ovarian Reserve in PCOS / Ovarian Drilling?

Medical Management: What’s the

Vitamin D

Interaction

Endometrial Polyps and Endometrial

Endometrial Cancer

Do Genes Matter?

If we initiate Metformin to promote fertility, should we

Interaction

Which is the best diet program?

Newer Insulin Sensitizers

Assisted Reproduction for PCOS – are the results

Sleep Apnoea – how serious can it be?

PCOS and Obesity –

Concerns of tomorrow

2.30 pm to 5.00 pm – Workshop II

PCOS – Management of Cosmetic Concerns

We see young women in our clinical practice with concerns such as acne, facial hair, hair loss, obesity, stretch marks, pigmentation, etc. which affect them tremendously! They are concerned by their distressing symptoms which affects their self-confidence and body image, leading to a lot of anxiety and mood changes. As clinicians attending to these young adolescents, it should be our endeavor to look beyond their symptoms and manage them correctly in order to prevent serious problems in future.

To discuss the latest advances in the management of their manifestations we have a team of renowned experts in the field of dermatology and cosmetic surgery to interact with you in this extremely useful workshop.

It is a “must workshop” for all to attend.

Inaugural Lectures

Understanding the Science of Hyperandrogenaemia and Insulin Resistance.

Do Genes Matter?

7.30 – INAUGURATION

8.30 – pm onwards DINNER
Introduction
Polycystic ovary syndrome (PCOS) is a heterogeneous and complex disorder with varied collection of signs and symptoms so as no single test is diagnostic. It has both adverse reproductive and metabolic implications for affected women. It is a complex genetic disorder and may be familial. PCOS is seen to cluster in families and both female and male relatives can show stigmata of the syndrome, including metabolic abnormalities. It is a multi-gene environmental interaction, resulting in the signs and symptoms, most of them related to abnormal gonadotropin secretion, hyperandrogenemia and insulin resistance with hyperinsulinism.

We know that diagnosing a woman with PCOS implies an increased risk for infertility, dysfunctional bleeding, endometrial carcinoma, obesity, type 2 diabetes mellitus (DM), dyslipidemia, hypertension, and possibly cardiovascular disease (CVD), and may require lifelong treatment.

Prevalence
The overall incidence of PCOS varies from 8-10 %, but it has been observed that the incidence is higher in South Asian population and is about 50% PCOS as against the Caucasian population, where the incidence is 5-25% PCOS.

Pathophysiology
Exact pathophysiology and its initiating event have yet to be elucidated. However, various biochemical abnormalities are described, and associations and linkages of one to another have been established. Many abnormalities reinforce each other in vicious circles. It is a multifactorial disease with full clinical expression being the result of synergistic pathological interaction of genetic, epigenetic and environmental factors. External factors like nutrients, physical activity, pollutants, psychological stress and androgen excess can program and modify epigenome and lead to PCOS.

Pathogenesis
Hyperandrogenism, abnormal feedback of gonadotrophins (higher mean concentration of LH, low or low-normal FSH, increased circulating estrone levels), insulin resistance (IR) and dysregulation of genes encoding enzymes in androgen biosynthesis pathway and insulin secretion and action (INS VNTR, CYP17, P450c17alpha) are responsible for the for this metabolic disorder. (Figure 1)

Hypothalamic-pituitary abnormalities
Elevated LH and low-normal FSH: In PCOS, there is increased frequency and amplitude of pulses of luteinizing hormone (LH) (normal is 10% to 20%), while that of follicle-stimulating hormone (FSH) is unchanged or muted. Thus, LH values may be elevated, and the LH:FSH ratio can be increased to more than 2.5, even in ovulatory cycles. Elevated GnRH. The inappropriate secretion of gonadotropins is thought to be due to an abnormality of the gonadotropin-releasing hormone (GnRH) pulse generator in the hypothalamus. It remains unclear whether this is a primary abnormality or a secondary one.

Elevated prolactin. Elevated prolactin is seen in 25% of patients and this elevation of prolactin may stimulate adrenal production of dehydroepiandrosterone sulfate (DHEA-S).

Ovarian Abnormalities
Hyperandrogenism
All patients with PCOS have an increased sensitivity to androgens and up to 70% have elevated androgen levels, and the other 30% are in the high-normal range. Excess androstenedione in the circulation is converted to estrone, which exerts a tonic effect on LH production while contributing to a relative suppression of FSH production. In the face of a high LH: FSH ratio, more androstenedione is synthesized but is not aromatized, thus perpetuating a vicious cycle driving LH production and some prolactin production. (Figure 2). The ovary converts some androstenedione to testosterone, and in PCOS this is amplified. Thus, the increased circulating testosterone comes from the ovaries and from peripheral conversion of estrone to testosterone.

Androgen levels are elevated in both obese as well

Abnormalities of estrogen secretion
Estrogen secretion is usually abnormal in PCOS. Estradiol levels may be low to normal, and in the anovulatory cycle there is tonic production without increase in levels before ovulation or in the midluteal phase, as in normal women. Estrone, androstenedione levels increase due to extraglandular conversion of androstenedione in adipose tissue.

Adrenal abnormalities
Excess adrenal androgen generated during stress or adolescence or due to congenital adrenal hyperplasia because of enzyme defects might initiate the cycle of abnormal LH:FSH stimulation and lead to PCOS.

Continued on page 08
Mechanisms of Anovulation in PCOS

For many years chronic anovulation and excessive androgen secretion have been considered the main components of clinical pattern of PCOS, both necessary for diagnosing this disorder. However, it is now well understood that PCOS may present with anovulatory but also with ovulatory phenotypes.10, 11 The anovulatory phenotype, while being the most common in patients who are referred to specialized clinics,6, 7 may not be the most common in general population.6, 8 Understanding the cause of anovulation in PCOS may represent a key step in finding the link between genetics and clinical expression of the disorder.

There is evidence that anovulation in PCOS is not the consequence of increased androgen ovarian secretion. In fact, although patients with the classic NIH anovulatory phenotype tend to have higher androgen levels than patients with the hyperandrogenic ovulatory phenotype,1, 9 elevated androgen levels may be found in non PCOS patients without determining anovulation.10, 11

Instead, most data suggest that in PCOS the anovulation is the consequence of the derangement of early follicle development that is characteristic of this disorder.12 Studies in cultures of follicles derived from anovulatory women with PCOS have shown that their granulosa cells are hyper-responsive to FSH in terms of estradiol production and tend to respond to LH also when the follicles are still small (3-4 mm).13, 14, 15 When granulosa cells from ovulatory PCOS patients were evaluated, these cells behaved normally in terms of estradiol response to FSH and responded to LH only when taken from a larger dominant follicle.15 It has been hypothesized that the inappropriate response in small follicles to LH could result in terminal differentiation of the granulosa cells and thence in premature arrest of follicle growth and anovulation.16 Because in the same studies, there was a large heterogeneity in the behavior of studied follicles of anovulatory women, with granulosa cells of some follicles responding normally to LH17, mathematical models have been developed suggesting that in a heterogeneous population of small follicles, if a group of follicles is relatively more mature, anovulatory arrest will develop.12 However, it is unlikely that the derangement of early follicle development is the only cause of chronic anovulation in PCOS. While the cause of the early follicle alteration in PCOS remains unclear, it is probable that is linked in some way to the genetic alteration that has been found in these patients. However, genetic studies have been unable to differentiate between ovulatory and anovulatory PCOS patients.18 Mostly important, the majority of anovulatory women with PCOS become ovulatory when they lose weight and at the contrary increase of body weight may transform an ovulatory PCOS woman in an anovulatory PCOS patient.1, 9 It is unclear how it could be possible if the problem is just depending on an inherited alteration affecting the early follicle development.

Interestingly, anovulatory PCOS patients present an endocrine character that may be partially reverted when they lose weight: these patients have higher insulin levels than ovulatory PCOS patients.10, 17 In anovulatory PCOS women, ovulation may also occur after administration of most insulin-sensitizing drugs, including metformin and thiazolidinediones. All it suggests that increased insulin circulating levels play a major role in the anovulation of women with PCOS. It is confirmed by in vitro studies showing that increased insulin levels, also in a condition of insulin resistance, may affect ovarian steroidogenesis and determine arrest of follicle growth.18, 19 On the other hand, the majority of obese women have normal follicular cycles20, 21 and it suggests that hyperinsulinemia alone is not able to induce anovulation but affects follicle growth only in presence of some previous derangement of follicle development. In conclusion, in the majority of women with PCOS the mechanism of anovulation requires two concomitant alterations, one mainly genetically induced, the early follicle development, and the second, mainly environmentally determined, a chronic hyperinsulinemia.

In figure 1, the possible pathophysiological mechanism of anovulation in PCOS is summarized. In this model, different gene variants may produce two essential characters of PCOS: derangement of early follicle development and ovarian androgen excess production. The consequence is (generally) the ovulatory PCOS. Increased insulin levels (probably secondary to obesity or altered fat function determined by environmental factors) determine the arrest of follicle growth and transform the ovulatory PCOS to an anovulatory (classic) PCOS. Other mechanisms of anovulation may be operative and may be important in subgroups of PCOS patients. About one third of classic hyperandrogenic anovulatory PCOS patients has normal body weight and 50% of them have normal fat distribution, normal insulin levels and no insulin resistance.22 Also PCOS patients with the normoandrogenic phenotype (chronic anovulation, polycystic ovaries and normal androgen levels) generally present normal body weight, normal insulin and insulin sensitivity, too.2, 10, and 23 Therefore, mechanisms independent on insulin have to exist and may determine arrest of follicle growth and anovulation. No evidence on what may be these mechanisms exist but it is possible that in some patients the genetic alteration is particularly severe and able to induce anovulation also in absence of hyperinsulinemia.

In fact, it has been suggested that increased AMH values may play a role in the arrest of follicle growth by inhibiting the recruitment of primordial follicles and diminishing the response of recruited follicles to FSH, thus impairing the selection of the dominant follicle.24 It is possible that, in some patients, a particularly severe alteration of folliculogenesis may determine very high levels of AMH sufficient to impair the selection of the dominant follicle. However, in the majority of PCOS patients, increased AMH does not seem to play a main role in blocking the selection and growth of the dominant follicle and the ovulation.20 According to it, patients with anovulatory normoandrogenic PCOS have relatively low AMH values.24 More studies are needed to understand the mechanism of anovulation in the different subgroups of PCOS but chronic anovulation in PCOS seems to be generally the consequence of an interplay between genetic mechanisms affecting the early follicle development and environmental factors inducing obesity or altered fat distribution and chronic hyperinsulinemia.

References on page 11
Pathophysiology of PCOS

Insulin resistance and hyperinsulinemia

Insulin resistance (IR) probably plays a pathogenic role in PCOS and is a forerunner of several metabolic alterations. The incidence of IR is higher in obese, irrespective of ethnicity. Adipose tissue dysfunction may be a major factor contributing to IR. Because hyperandrogenism and hyperinsulinemia coexist in PCOS, the important question is whether one causes the other. We know that that exogenous androgens and androgen producing tumors result in hyperandrogenism, which can result in glucose intolerance and elevated insulin levels.15

There is abundant evidence that indicates that hyperinsulinemia begets hyperandrogenism.16 Insulin may increase androgen synthesis by various mechanisms. It may directly increase ovarian androgen synthesis by interacting with its own receptor or with the receptor for insulin-like growth factor-1, thereby increasing P450c17-alpha enzyme activity. Insulin amplifies the LH response of granulosa factor-1, thereby increasing P450c17-alpha enzyme mechanisms. It may directly increase ovarian SHBG, which increases free testosterone levels. Thus, insulin alters normal folliculogenesis by increasing intra-ovarian androgens, altering gonadotropin release, or by direct effects on the ovary. (Figure 4)

While PCOS is associated with insulin resistance and hyperinsulinemia, the ovary itself is not insulin-resistant and, in fact, possibly responds excessively to the hyperinsulinemia. Cell surface insulin receptors are at normal levels, but there is a postreceptor defect in signal transduction, causing a decrease in glucose transport. The post-receptor binding defect is an increase in insulin receptor-mediated serine phosphorylation with a concomitant decrease in protein kinase activity and necessary tyrosine kinase activity, thereby interfering with transduction of the insulin signal and causing it to be defective.10

Adipose tissue dysfunction and PCOS

Adipose tissue dysfunction may be a central factor in the pathogenesis of PCOS. There exists a complex interaction between the pituitary gland, pancreas and ovary that results in a changed hormonal secretion pattern. PCOS is thought to be mediated by ghrelin, (a gastric peptide) which is orexigenic and adipogenic. Obesity is known to increase androgen, insulin & leptin levels, insulin resistance and risk of early pregnancy loss. It also decreases SHBG, GH, IGFBP-1, response to COS thus requiring higher doses of GT with longer duration of stimulation and cycle cancellation rate. It also increases the peak E2 concentrations, number of oocytes retrieved and can affect the endometrial receptivity and corpus luteum function along with early embryo development. Subcutaneous and omental fat gene expression studies have shown altered expression of genes related to obesity, insulin resistance, inflammation and steroidogenesis. Epigenetic modification is linked to metabolic disease and genetic and environmental factors affect human muscle and adipose tissue epigenetics in PCOS and Type 2 Diabetes.

Evidence of possible dysregulation in the secretion of several adipocytokines, including leptin, adiponectin, tumor necrosis factor alpha (TNF-a), IL-6, monocyte chemoattractant protein-1 (MCP-1), visfatin, and retinol-binding protein 4 (RBP4), were reviewed. Data suggested, however, that the main factor perturbing adipocyte function in PCOS was the degree of abdominal obesity.11 Importantly, ovariolan women with PCOS generally exhibit smaller changes in the expression of adrenal androgen, insulin & leptin levels, insulin resistance, inflammation and steroidogenesis. Epigenetic modification is linked to metabolic disease and genetic and environmental factors affect human muscle and adipose tissue epigenetics in PCOS and Type 2 Diabetes.

Leukocyte telomere length (LTL) plays an important role in the pathophysiology of PCOS and has potential importance for our understanding of the etiology of the disease. LTL is strongly associated with PCOS and that there is a significant negative correlation between LTL and serum DHEAS concentrations in healthy controls.12 In vitro studies have demonstrated theca cell CYP17 expression and androgen synthesis were inhibited by FSH.13 Differential gene expression in subcutaneous fat and genetic association at the FOS locus in PCOS subjects implicates a role for this transcription factor in PCOS. FOS dysregulation may be a common factor between hyperandrogenism and insulin resistance.14

Immune dysfunction

Leukocyte telomere length (LTL) plays an important role in the pathophysiology of PCOS and has potential importance for our understanding of the etiology of the disease. LTL is strongly associated with PCOS and that there is a significant negative correlation between LTL and serum DHEAS concentrations in healthy controls.12 In women with PCOS, the mean expression of GAB1 is reduced as compared with normal fertile women. Endometrial GAB1 protein and mRNA expression are reduced in women with PCOS, suggesting that the endometrium of PCOS women has a defect in insulin signaling due to GAB1 down-regulation.15

Three TGF-isoforms (TGF-1, TGF-2, and TGF-3) have been identified in humans.16 The ovaries of women with PCOS show all the characteristics of TGF-hyperactivity including increased vascularity and increased deposition of collagen in ovarian stroma and theca.17,18 There are some genetic studies

Continued from page 11
CONFERENCE REGISTRATION FORM

Dr. First Name Middle Name Last Name

Age Sex  M  F  Designation

Institution / Hospital

Address for correspondence

City State

Pin code Country

STD Code Phone (Resi.)

Phone (Clinic / Hospital)

Fax Mobile

Email

Medical Council No.

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DD/ Cheque in Favour of ‘The PCOS International Conference - 2016’

DD/ Cheque No. For Rs.

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Bank name and address – Bank of India, Dell St, NS Patkar Marg, Hughes Road, Mumbai 400026

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THE PCOS SOCIETY (India)

MEMBERSHIP FORM

Membership criteria and types:

For Life Members: Doctors with a minimum of post graduate degree or diploma in any speciality of medicine can apply.

For Associate Members: Nutritionists, physiotherapists, counsellors etc. can apply.

Membership is open to any physician or allied health professional with an interest in research, teaching or development activities in the areas of PCOS.

Name

Date of Birth

Address for correspondence

City State

Pincode

Mobile number Telephone No (with STD code)

Email (Mandatory)

Profession / Occupation

MCI registration no. and date

Qualifications: (Certificates attached)

University / College / Institution

Year of Qualifying Speciality

Current Position: Affiliation with Institutions / Societies

Community / Extension Services

(Please include any membership of Rotary/Lions/Jaycees/Ladies Organization)

MEMBERSHIP FEE

■ Life Members (Diploma / Degree holders in speciality of Medicine / Surgery): ₹ 5,000/-

■ Associate Member – (Allied professionals like physiotherapists, nutritionists, counsellors, etc.) – No voting rights.

■ Patron Members

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10% discount on PCOS Society Membership if registered by 15th March 2016.
Pathophysiology of PCOS

supporting the role of TGF- dysregulation in PCOS and showing that allele 8 of D19S8584 within intron 55 of the fibrillin-3 gene is associated with PCOS and increased insulin resistance (IR) in women with PCOS.24,25 Fibrillins are matrix components of extracellular microfibrils that are regulated by TGF- This may contribute to the metabolic disturbances in women with PCOS.26

Vitamin D and PCOS

Decreased vitamin D levels27 have been correlated with increased insulin resistance, body mass index (BMI), total testosterone, and DHEAS in PCOS women.20,21 Beneficial effects of vitamin D treatment in women with PCOS may be mediated via Vitamin D’s effects on TGF-1 and for SEng.

Conclusion

PCOS is a polygenic disorder representing a quantitative trait in which small number of key genes contribute in conjunction with environmental factors to produce the observed clinical & biochemical heterogeneity. Cellular signaling abnormalities in PCOS are due to hyperinsulinemia. Insulin acts synergistically with LH to stimulate ovarian androgen production. Excess effect of insulin on SHBG production also contributes to higher levels of free or bioavailable testosterone and indirectly to the abnormalities in GT secretion.

Key Points

- PCOS is characterized by amenorrhea, hirsutism and infertility. It is caused by a complex interaction of abnormalities in gonadotropins, androgens and estrogens. Insulin resistance and hyperinsulinemia contribute significantly to its underlying pathophysiology.
- Intrauterine environment together with genetic predisposition affects the offspring.
- Prenatal androgen excess may predispose to PCOS via alteration of the epigenome.
- Obesity contributes to the development of PCOS.
- Lifelong monitoring for cancer, diabetes and coronary artery disease is crucial.

References


References

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