Dr. Padma Rekha Jirge MRCOG (UK), FICOG, MBA (Healthcare Mx)

- **Clinical research fellow** – in ART; from University of Glasgow 1995 – 1997
- Trained in Operative Laparoscopy and Hysteroscopy, Glasgow 1994-1995

- **Scientific Director** – 1.Sushrut Assisted Conception Clinic, & Shreyas Hospital , Kolhapur
- **Publications** - 2 manuscripts on role of LH in ovulation induction - in Human Reproduction
  - infection and IVF - in Fertility Sterility
  - author of 15 chapters on various aspects of ART in textbooks
  - Stem Cells - FAQ and answers –FOGSI Focus Jan 2008
  - **Comparative study of Letrozole vs Clomiphene – Fertility Sterility, Jan 2010**
  - **Ovarian Reserve Tests – A review, Journal of Human Reproductive Sciences, Jan 2012**
  - **DHEA supplementation in poor responders…. JHRS, Sep 2014**
  - **Co-editor of World Clinics in O&G (Ovulation Induction) November 2015**
  - **Poor Ovarian Reserve – JHRS**
  - **Preparing and Publishing Scientific Manuscripts – A review - JHRS**

- **Sushrut IVF Clinic: Recognised by ICOG for fellowship course in IVF**
- Chairperson, Research Committee, PCOS Society of India
- Co-opted Member, Managing Committee, ISAR
- National corresponding Editor – Journal of O&G of India
- **On Editorial board of Austin Journal of Reproductive Medicine & Infertility; and Journal of IVF Lite**
- **Reviewer for 1. Journal of Human Reproductive Sciences**
  2. Reproductive Biology & Endocrinology Journal (RB&E)
  3. Journal of Assisted Reproduction & Genetics
- Clinical secretary, Maharashtra Chapter of ISAR
- Editor & Founder member of Fertility Preservation Society of India
- Elected Member – Representative Committee, West Zone, AICC-RCOG

What are the different Phenotypes of PCOS? Its importance in Management

Dr. Padma Rekha Jirge MRCOG(UK), FICOG, MBA (Healthcare Mx)

Shreyas Hospital & Sushrut Assisted Conception Clinic, Kolhapur

PCOS

- Multitude of symptoms – endocrine / metabolic
- Multifactorial in origin
- Diagnosis – important clinical implications for the individual and relatives

PCOS – Diagnosis
NIH Criteria (1999)

1999 criteria (both 1 and 2)

1. Chronic anovulation
2. Clinical and/or biochemical signs of hyperandrogenism, and exclusion of other aetiologies

Exclusion:

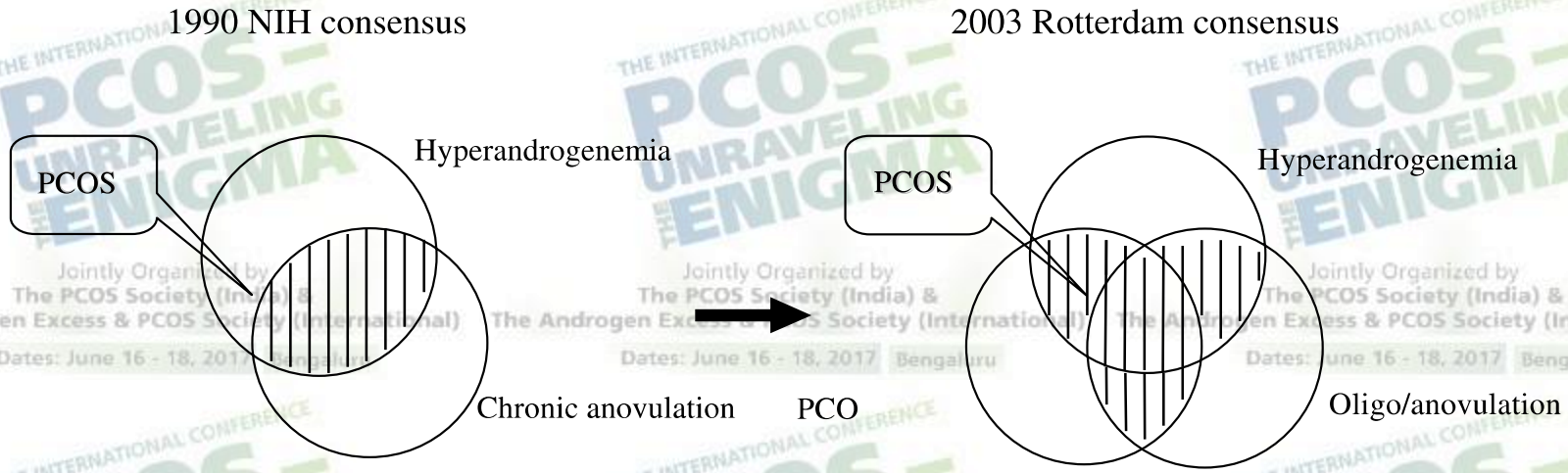
congenital adrenal hyperplasia, androgen secreting tumours, hyperprolactinaemia, and thyroid disorders.

PCOS – Rotterdam Criteria

Revised 2003 criteria (2 out of 3)

1. Oligo- and/or anovulation
2. Clinical and/or biochemical signs of hyperandrogenism
3. Polycystic ovaries, and exclusion of other aetiologies (congenital adrenal hyperplasias, androgen-secreting tumours, Cushing's syndrome)

NIH and Rotterdam Criteria



Rotterdam Criteria
expanded the definition of PCOS
Introduced different subgroups

PCOS: AE-PCOS Society Criteria

ANDROGEN EXCESS AND POLYCYSTIC OVARY SYNDROME SOCIETY: CRITERIA FOR THE DIAGNOSIS OF THE POLYCYSTIC OVARY SYNDROME

1- Hyperandrogenism: Hirsutism and/or hyperandrogenemia

and

2 – Ovarian Dysfunction: Oligo-anovulation and/or polycystic ovaries

and

3 - Exclusion of other androgen excess or related disorders[a]

Proposed criteria for the diagnosis of the PCOS. [a]Possibly including 21-hydroxylase deficient nonclassic adrenal hyperplasia, androgen-secreting neoplasms, androgenic/anabolic drug use or abuse, Cushing's syndrome, the Hyperandrogenic-Insulin Resistance-Acanthosis Nigricans syndrome, thyroid dysfunction, and hyperprolactinemia.

Diagnosis and Treatment of Polycystic Ovary Syndrome: An Endocrine Society Clinical Practice Guideline

Richard S. Legro, Silva A. Arslanian, David A. Ehrmann, Kathleen M. Hoeger, M. Hassan Murad, Renato Pasquali, and Corrine K. Welt

Category	Specific Abnormality	Recommended Test	NIH	Rotterdam (2 of 3 Met)	Androgen Excess PCOS Society (Hyper-Androgenism With 1 of 2 Remaining Criteria)
Androgen status	Clinical hyperandrogenism[a]	Clin a p			
	Biochemical hyperandrogenism[a]	Bioc s e G s a c re			
Menstrual history	Oligo- or anovulation	Ano i > d m h s			
Ovarian appearance	Ovarian size/morphology on ultrasound	The o i d			

- Use of Rotterdam Criteria for diagnosis
- Ovulation to be confirmed and not to be relied upon regularity of the cycle
- If clinical HA is present, then serum androgens need not be evaluated
- If HA and Ovulatory dysfunction, no need for USG

The Task Force suggests using the Rotterdam criteria for the diagnosis of PCOS, acknowledging the limitations of each of the three criteria (Table 2). All criteria require exclusion of other diagnoses (listed in Table 3) that cause the same symptoms and/or signs (6–9). X, may be present for diagnosis; XX, must be present for diagnosis.

[a] Clinical or biochemical hyperandrogenism is included as one criterion in all classification systems. If clinical hyperandrogenism is present with the absence of virilization, then serum androgens are not necessary for the diagnosis. Similarly, when a patient has signs of hyperandrogenism and ovulatory dysfunction, an ovarian ultrasound is not necessary.

Phenotypes of PCOS

- Oligo /anovulation+HA+PCO (Classic PCOS)
- Oligo / anovulation + HA (NIH PCOS)
- HA+PCO (Ovulatory PCOS)
- Oligo / anovulation+PCO (Non-androgenic PCOS)

Risks Associated with PCOS

- Infertility
- Hypertension, cardiovascular morbidity
- Insulin resistance and type 2 DM
- Dyslipidemia
- Metabolic syndrome
- Endometrial Carcinoma
- Implications for mothers, sisters, brothers and offspring

Phenotypes and Clinical Implications

- **Do different phenotypes influence the severity of the condition?**
- **Metabolic risks – Hyperandrogenism or Insulin resistance**
- Impact of obesity
- Age
- Influence of ethnicity

Characterizing Discrete Subsets of Polycystic Ovary Syndrome as Defined by the Rotterdam Criteria: The Impact of Weight on Phenotype and Metabolic Features

C. K. Welt,* J. A. Gudmundsson,* G. Arason,* J. Adams, H. Palsdottir, G. Gudlaugsdottir, G. Ingadottir, and W. F. Crowley

Context: The Rotterdam criteria for polycystic ovary syndrome (PCOS) defines discrete subgroups whose phenotypes are not yet clear.

Objective: The phenotypic characteristics of women in the PCOS subgroups defined by the Rotterdam criteria were compared.

Design: The study was observational.

Setting: Subjects were studied in an outpatient setting in Boston and Reykjavik.

Patients: Four subgroups of subjects with PCOS defined by 1) irregular menses (IM), hyperandrogenism (HA), and polycystic ovary morphology (PCOM, n = 298); 2) IM/HA (n = 7); 3) HA/PCOM (n = 77); and 4) IM/PCOM (n = 36) and a group of controls (n = 64), aged 18–45 yr, were examined.

Intervention: Subjects underwent a physical exam; fasting blood samples for androgens, gonadotropins, and metabolic parameters; and a transvaginal ultrasound.

Main Outcome Measures: The phenotype was compared between groups.

Results: Ninety-seven percent of women with IM/HA had PCOM. Therefore, the groups with and without PCOM were combined. The Ferriman-Gallwey score and androgen levels were highest in the hyperandrogenic groups (IM/HA and HA/PCOM), whereas ovarian volume was higher in all PCOS subgroups compared with controls, as expected based on the definitions of the PCOS subgroups. Body mass index and insulin levels were highest in the IM/HA subgroup.

Conclusions: Subjects with PCOS defined by IM/HA are the most severely affected women on the basis of androgen levels, ovarian volumes, and insulin levels. Their higher body mass index partially accounts for the increased insulin levels, suggesting that weight gain exacerbates the symptoms of PCOS. (***J Clin Endocrinol Metab* 91: 4842–4848, 2006**)

IR highest in IM/HA group; HDL lowest
Also, highest MetS related to weight

J Clin Endocrinol Metab. 2006 Oct;91(10):3922-7

Oligoanovulation with polycystic ovaries but not overt hyperandrogenism.

Dewailly D1, Catteau-Jonard S, Reyss AC, Leroy M, Pigny P.

OBJECTIVES:
Rotterdam definition recognizes four PCO syndrome (PCOS) phenotypes: HA+OA+PCO (full-blown syndrome), HA+OA (former National Institutes of Health definition), HA+PCO (ovulatory PCOS), and OA+PCO. However, **the latter phenotype is controversial**, and it is not known to what extent it shares similarities with the others.
DESIGN:
The study was a comparative analysis of hormonal, metabolic, and ultrasound parameters obtained from patients and controls that were consecutively included in a database.
PATIENTS AND METHODS:
Sixty-six patients having OA+PCO without hirsutism or elevated serum androstenedione and testosterone levels were compared with 118 normally cycling nonhyperandrogenic age-matched women without PCO (controls). These patients (phenotype D) were also compared with patients with HA+OA+PCO (phenotype A, n = 246), HA+OA (phenotype B, n = 27), and HA+PCO (phenotype C, n = 67).
RESULTS:
Patients with phenotype D had higher mean values of waist circumference and higher mean levels of serum testosterone, androstenedione, and LH than controls. Conversely, they had lower mean serum levels of FSH and SHBG ($P < 0.05$ for each parameter). Variance analysis disclosed significant group effects between the different patients' phenotypes for all parameters, except age, BMI, and FSH. After multiple comparisons with post hoc analysis, **phenotype D had milder endocrine and metabolic abnormalities than phenotype A,** although it did not differ from phenotype C, except for androgen data, by definition. Phenotypes A and B were statistically similar, except for the ultrasound data, by definition.
CONCLUSION:
Oligoanovulatory patients with PCO but without HA have mild endocrine and metabolic features of PCOS.

Normo- and hyperandrogenic women with polycystic ovary syndrome exhibit an adverse metabolic profile through life

Pekka Pinola, Ph.D.,[a,b] Katri Puukka, Ph.D.,[b,c] Terhi T. Piltonen, Ph.D.,[a,b] Johanna Puurunen, Ph.D.,[a,b] Eszter Vanky, Ph.D.,[d,e] Inger Sundström-Poromaa, Ph.D.,[f] Elisabet Stener-Victorin, Ph.D.,[g] Angelica Lindén Hirschberg, Ph.D.,[h] Pernille Ravn, Ph.D.,[i] Marianne Skovsager Andersen, Ph.D.,[i] Dorte Glintborg, Ph.D.,[j] Jan Roar Mellembakken, Ph.D.,[k] Aimo Ruokonen, Ph.D.,[b,c] Juha S. Tapanainen, Ph.D.,[b,l] and Laure C. Morin-Papunen, Ph.D.[a,b]

1550 women with PCOS

- Raised BMI / Waist circumference
- Raised Testosterone
- Lower HDL
- Impaired OGTT
- Raised BP and hs CRP

Anthropometric and metabolic para

Metabolic parameter				Result	*P* value
Age (y)				30.0 (7.2)	<.001
BMI (kg/m^2)	447			29.2 (6.9)	<.001
Waist (cm)	312		1,204	92.9 (17.5)	<.001
Testosterone (nmol/L)	433	1.1 (0.5)	1,359	1.8 (1.0)	<.001[a]
Fasting glucose (mmol/L)	376	5.1 (0.9)	1,104	5.1 (0.6)	NS
Fasting insulin (mU/L)	372	7.4 (6.0)	1,093	12.3 (11.1)	<.001[a]
Total cholesterol (mmol/L)	364	4.6 (0.9)	982	4.8 (1.0)	.003[a]
HDL (mmol/L)	346	1.5 (0.3)	960	1.4 (0.4)	<.001[a]
LDL (mmol/L)	347	2.6 (0.8)	863	2.9 (0.9)	<.001[a]
Triglycerides (mmol/L)	366	0.9 (0.5)	974	1.3 (0.8)	<.001[a]
OGTT glucose, 2 h (mmol/L)	140	5.0 (1.3)	681	5.8 (1.8)	<.001
OGTT mean glucose (mmol/L)	140	5.0 (0.8)	681	5.5 (1.1)	<.001
OGTT insulin, 2 h (mU/L)	152	27.4 (20.5)	860	71.7 (73.9)	<.001[a]
OGTT mean insulin (mU/L)	152	17.2 (12.0)	840	42.6 (41.6)	<.001[a]
Systolic BP (mm Hg)	318	118.2 (15.6)	1,276	123.43 (16.2)	<.001[a]
Diastolic BP (mm Hg)	318	74.3 (12.1)	1,276	78.5 (12.1)	<.001[a]
hs-CRP (mg/L)	159	1.5 (3.0)	761	2.8 (3.8)	<.001

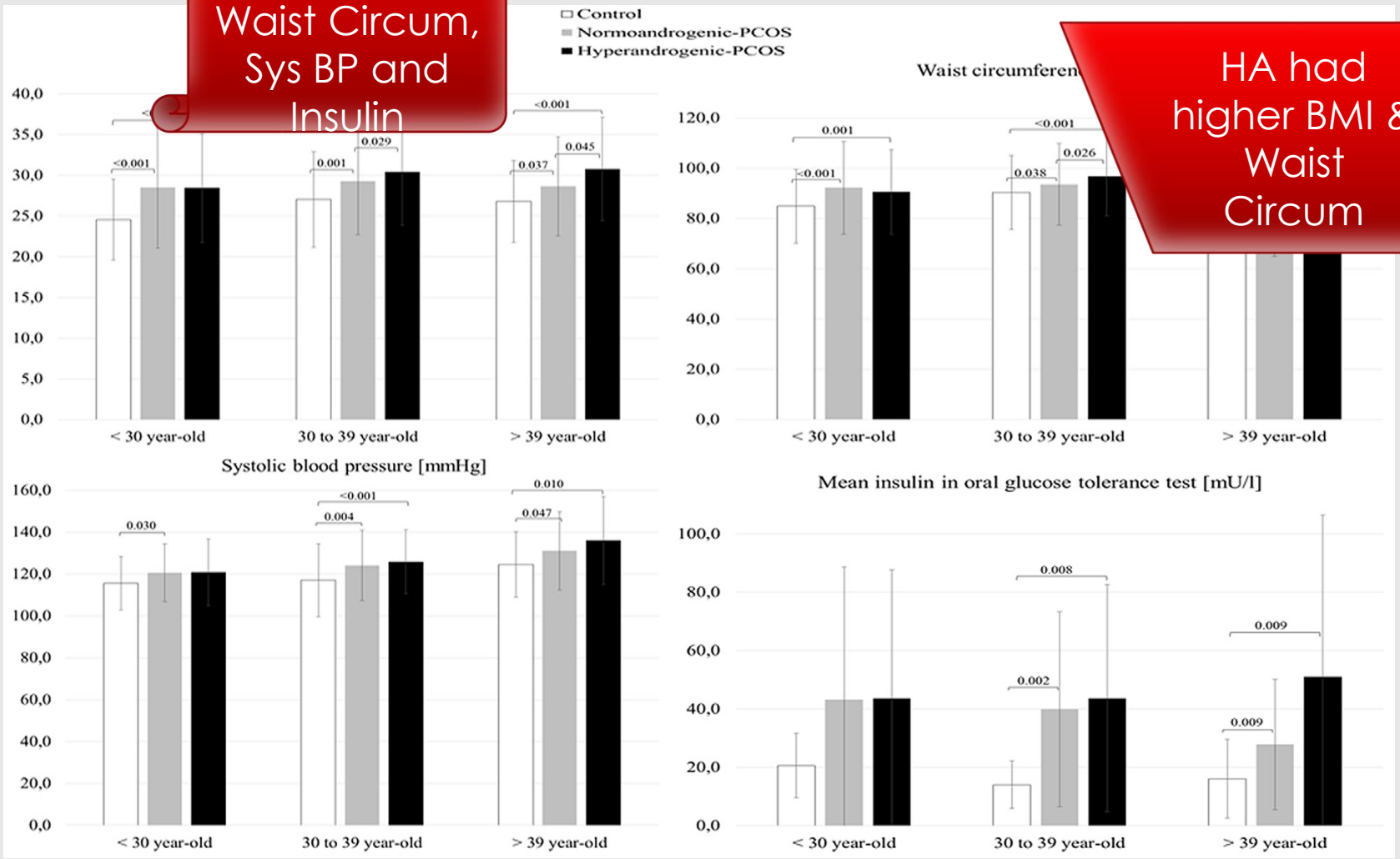
Note: Data presented as n or mean (SD), unless stated otherwise. BMI = body mass index; BP = blood pressure; HDL = high-density lipoprotein; hs-CRP = high-sensitivity C-reactive protein; LDL = low-density lipoprotein; OGTT = oral glucose tolerance test.

[a] Statistical significance ($P<.05$) remains after adjustment for age and BMI.

Pinola. Metabolic profile in women with PCOS. Fertil Steril 2016.

Both HA and NA PCOS – higher BMI, Waist Circum, Sys BP and Insulin

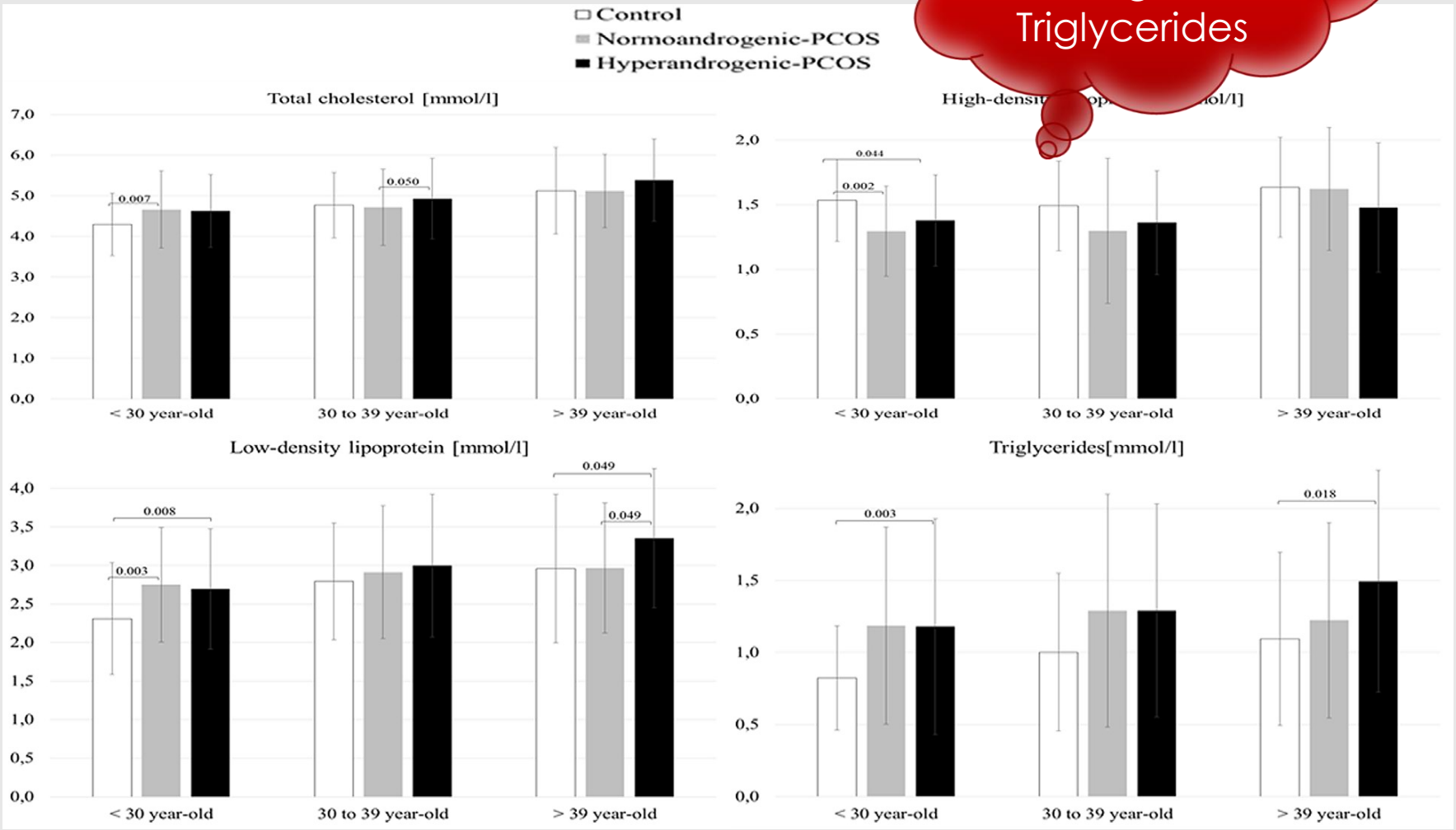
HA had higher BMI & Waist Circum



Body mass indices, waist circumferences, and parameters of glucose metabolism at different ages in the study populations. The *bars* represent means and the *error bars* standard deviations. Results are adjusted for body mass index. PCOS = polycystic ovary syndrome.

Pinola. Metabolic profile in women with PCOS. Fertil Steril 2016.

Low HDL &
High LDL
High
Triglycerides



Lipids at different ages in the study populations. The *bars* represent the means and the *error bars* the standard deviations. Results are adjusted for body mass index. PCOS = polycystic ovary syndrome.

Pinola. Metabolic profile in women with PCOS. Fertil Steril 2016.

Human Reproduction, Vol.30, No.10 pp. 2396–2403, 2015
Advanced Access publication on July 29, 2015 doi:10.1093/humrep/dev187

human reproduction

ORIGINAL ARTICLE *Reproductive endocrinology*

Maternal and neonatal outcomes in pregnant women with PCOS: comparison of different diagnostic definitions

M. Kollmann[1], P. Klaritsch[1,*], W.P. Martins[2], F. Guenther[1], V. Schneider[1], S.A. Herzog[3], L. Craciunas[4], U. Lang[1], B. Obermayer-Pietsch[5], E. Lerchbaum[5], and N. Raine-Fenning[4]

Table III Maternal and neonatal complications in PCOS pregnancies.

	NIH 1990 ($n = 85$)		AE-PCOS 2006 ($n = 14$)		ESHRE/ASRM 2003 ($n = 78$)		Within PCOS
	n	%	*n*	%	*n*	%	*P*-value
Maternal complications							
Gestational diabetes	16/85	18.82	2/14	14.3	21/78	26.9	0.36
PIH	8/85	9.4%	2/14	14.3	9/78	11.5	0.81
Pre-eclampsia	4/85	4.7	1/14	7.1	1/78	1.3	0.32
Operative delivery	34/83	41.0	9/14	64.3	50/77	64.9	0.007[a]
Total complication rate	42/85	49.4	9/14	64.3	47/78	60.3	0.31
Neonatal complications							
Preterm birth $<34+0$	4/83	4.8	1/14	7.1	1/77	1.3	0.32
Preterm birth $<37+0$	11/83	13.3	1/14	7.1	8/77	10.4	0.72
SGA (<10th percentile)	6/81	7.4	1/12	8.3	8/76	10.5	0.97
LGA (>90th percentile)	5/81	6.2	1/12	8.3	4/76	5.3	
Fetal acidosis	3/62	4.8	0/10	0	1/59	1.7	0.72
ICU	6/83	7.2	4/14	28.6	4/75	5.3	0.02[b]
Pre- and perinatal mortality	0/83	0	1/14	7.1	2/77	2.6	0.06
Total complication rate	23/85	27.1	5/14	35.7	18/78	23.1	0.62

Table IV **Maternal and neonatal complications PCOS versus control.**

	PCOS (n = 177)		Control (n = 708)		PCOS versus Control	
	n^a	%	n^a	%	OR (95% CI)[a]	*P*-value
Maternal complications						
Gestational diabetes	39/171	22.8	18/708	2.5	10.97 (6.02–20.72)	<0.001
PIH	19/171	11.1	9/708	1.3	8.25 (3.60–20.23)	<0.001
Pre-eclampsia	6/171	3.5	11/708	1.6	1.91 (0.63–5.20)	0.221
Operative delivery	89/168	53.0	282/706	39.9	1.70 (1.21–2.42)	0.003
Total complication rate	95/171	55.6	229/708	32.3	2.57 (1.82–3.64)	<0.001
Neonatal complications						
Preterm birth <34 + 0	6/168	3.6	30/708	4.2	0.87 (0.32–2.01)	0.758
Preterm birth <37 + 0	19/168	11.3	66/708	9.3	1.26 (0.71–2.15)	0.408
SGA (<10th percentile)	15/163	9.2	93/701	13.3	0.76 (0.46–1.21)	0.260
LGA (>90th percentile)	10/163	6.1	37/701	5.3		
Fetal acidosis	4/125	3.2	17/668	2.5	1.11 (0.31–3.14)	0.854
ICU	/166	8.4	39/422	9.2	0.93 (0.47–1.76)	0.827
Pre- and perinatal mortality	3/168	1.8	7/708	1.0	1.88 (0.39–7.04)	0.373
Total complication rate	45/171	26.3	206/708	29.1	0.83 (0.56–1.21)	0.343

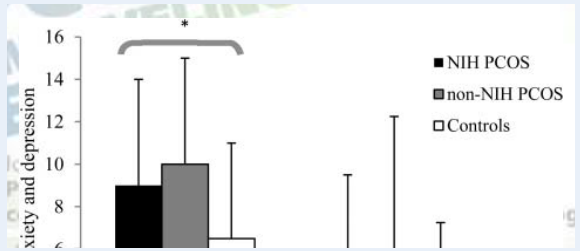
Human Reproduction, Vol.27, No.7 pp. 2082–2088, 2012
Advanced Access publication on April 4, 2012 doi:10.1093/humrep/des114

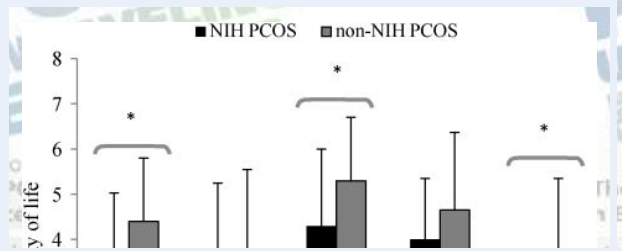
human reproduction

ORIGINAL ARTICLE *Psychology and counselling*

Psychological parameters in the reproductive phenotypes of polycystic ovary syndrome

L.J. Moran [1,2,*], A.A. Deeks [2], M.E. Gibson-Helm [1,2], and H.J. Teede [1,2,3]





CONCLUSIONS: PCOS is associated with anxiety and depression. Non-NIH phenotypes present with similar psychological profiles to NIH PCOS, indicating increased psychological dysfunction in PCOS, even in milder reproductive phenotypes. However, women with NIH PCOS appear to have worse HRQoL in some areas than women with non-NIH PCOS. Psychological function and HRQoL should be considered in all women with PCOS.

factor. *Significant difference $P = 0.028$ between PCOS phenotypes and controls such that trend for differences between NIH PCOS and controls ($P = 0.054$) and non-NIH PCOS and controls ($P = 0.076$) but no difference between NIH and non-NIH PCOS ($P = 0.994$). HADS, Hospital Anxiety and Depression Scale; NIH, National Institute of Health; PCOS, polycystic ovary syndrome.

Figure 2 HRQoL in women with different PCOS phenotypes. Data are presented as median ± IQR and were assessed by one-way ANOVA with PCOS phenotype as the between subject factor. *Significant difference $P < 0.05$ between NIH PCOS and non-NIH PCOS. NIH, National Institute of Health; PCOS, polycystic ovary syndrome.

Prevalence of the metabolic syndrome in women with a previous diagnosis of polycystic ovary syndrome: long-term follow-up

Miriam Hudecova, M.D., Ph.D.,[a] *Jan Holte, M.D., Ph.D.,*[a,b] *Matts Olovsson, M.D., Ph.D.,*[a] *Anders Larsson, M.D., Ph.D.,*[c] *Christian Berne, M.D., Ph.D.,*[c] *and Inger Sundstrom-Poromaa, M.D., Ph.D.*[a]

Anthropometric measures and metabolic variables in all PCOS patients, in resolved and persisting PCOS patients, in different PCOS phenotypic subgroups, and in healthy controls at the follow-up investigation.

Characteristic	All PCOS patients[a] (n = 84)	PCOS patients with persisting symptoms at follow-up[a] (n = 27)	PCOS patients with resolved symptoms at follow-up[a] (n = 27)	PCOS patients with hirsutism, oligomenorrhea, and PCO at index assessment[a] (n = 40)	PCOS patients with oligomenorrhea and PCO at index assessment[a] (n = 32)	Control subjects (n = 87)
Age	43.0 ± 5.8	40.6 ± 4.2[b]	43.3 ± 4.6	43.2 ± 5.6	42.2 ± 5.9	43.7 ± 6.2
BMI (kg/m^2)	28.3 ± 6.0[c]	28.7 ± 6.3[b]	26.4 ± 5.0	29.6 ± 6.0[b,d]	26.7 ± 5.8	25.7 ± 4.4
Waist (cm)	89 ± 15[c]	91 ± 16[b,e]	83 ± 13	92 ± 15[b,d]	85 ± 15	82 ± 11
Fasting glucose (mg/dL)	90.1 ± 16.2[c]	90.1 ± 19.8	86.5 ± 14.4	86.5 ± 10.8	84.7 ± 10.8	84.7 ± 9.0
Triglycerides (mg/dL)	125.7 ± 82.3[c]	116.8 ± 57.5	135.4 ± 106.2[b]	126.5 ± 79.6[b]	123.9 ± 88.5[b]	90.3 ± 42.5
HDL cholesterol (mg/dL)	61.8 ± 19.3	57.9 ± 15.4	65.6 ± 23.2	61.8 ± 19.3	61.8 ± 15.4	65.6 ± 15.4
Systolic BP (mm Hg)	133 ± 21[c]	129 ± 20	131 ± 20	131 ± 21	136 ± 22[b]	125 ± 17
Diastolic BP (mm Hg)	83 ± 13[c]	82 ± 13	81 ± 12	82 ± 13	84 ± 12	79 ± 10

[a] Women with diabetes not included.
[b] Significantly different from control subjects, ANOVA post hoc Tukey HSD, $P<.05$–$.001$.
[c] Significantly different from control subjects, Student's t test, $P<.05$–$.001$.
[d] Significantly different from PCOS patients with oligomenorrhoea and PCO at index assessment, ANOVA post hoc Tukey HSD, $P<.05$–$.001$.
[e] Significantly different from resolved PCOS patients, ANOVA post hoc Tukey HSD, $P<.05$.

Hudecova. Metabolic syndrome in women with PCOS. Fertil Steril 2011.

Conclusion(s): The MetS occurred more often in patients with PCOS than in controls and did not depend on phenotypic presentation at the index assessment or the persistence of PCOS at follow-up. (Fertil Steril® 2011;96: 1271–4. ©2011 by American Society for Reproductive Medicine.)

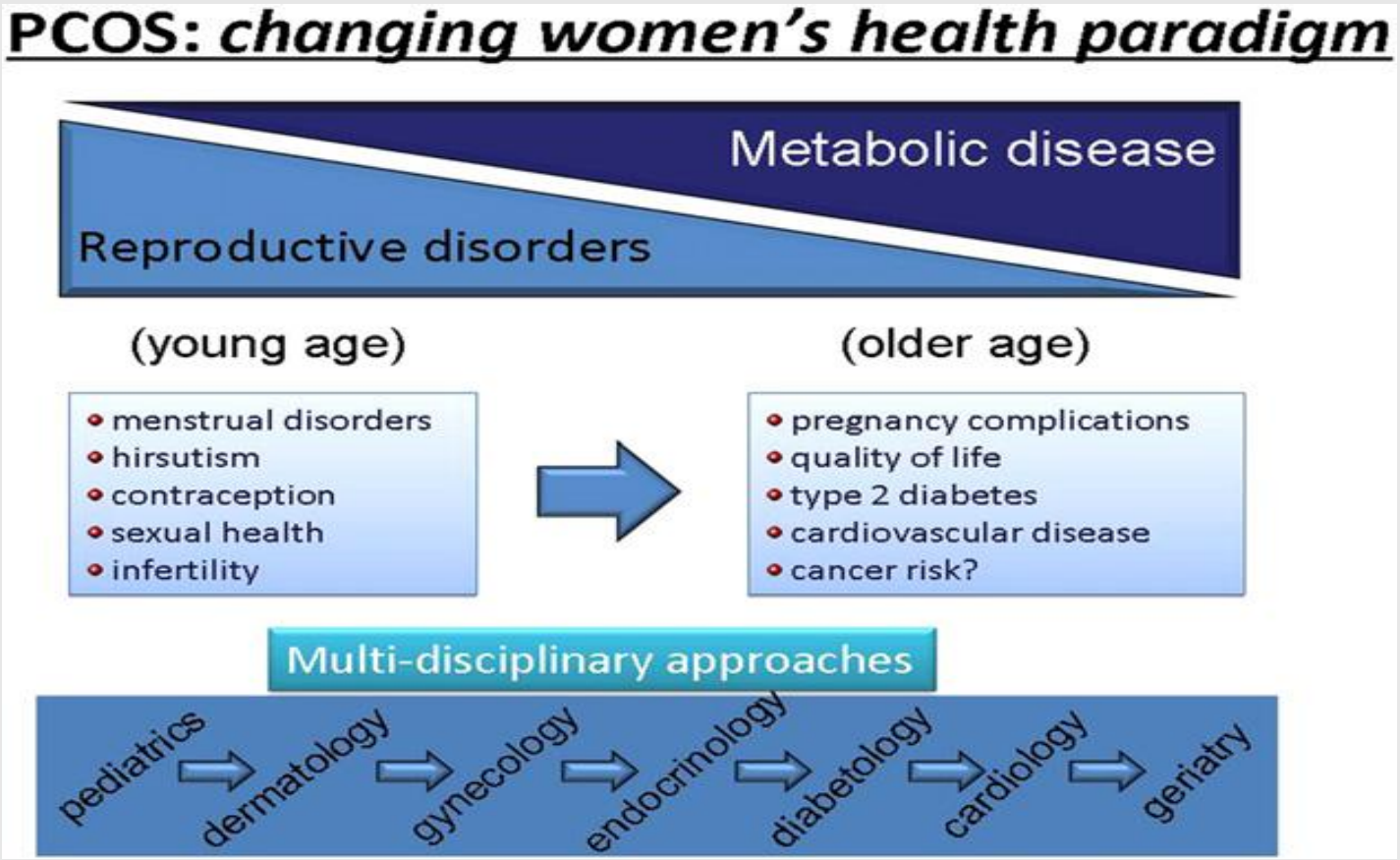
Ethnicity & Phenotypes

- **Asian women** – lower BMI, central obesity, milder HA but high prevalence of MetS and T2D
- African and Hispanic – more obese, Africans more prone to hypertension and cardiovascular disease; Hispanics more prone to MetS and T2D
- High prevalence of hirsutism in Mediterranean and Middle Eastern women

Clinical Implications

- Different phenotypes may exhibit different range of metabolic dysfunction.
- Those with HA have more severe metabolic abnormalities
- The phenotypic dysfunction may become less obvious with age and in particular following menopause
- Obesity impacts the severity of metabolic dysfunction
- Ethnicity

(Endocrine Society and Amsterdam ESHRE / ASRM Consensus



Schematic representation of the change in emphasis from early age reproductive disorders to long-term metabolic and cardiovascular health.

Fauser. ESHRE/ASRM PCOS Consensus. Fertil Steril 2012.

Management Implications

- **Adolescent PCOS** – Establishing diagnosis can be challenging; AVOID over-diagnosis
- **Hirsuitism:** (underlying hyperandrogenism); needs long term treatment
- **Oligomenorrhoea:** Severe form of HA in amenorrhoea; associated with metabolic abnormalities (Level B); cycles may become regular with increasing age
- **Contraception:** OCP use does not increase metabolic risk (Level B)
- **QoL:** increased prevalence of psychological disorders in all phenotypes of PCOS; (?disorder in itself ?its manifestations); should be considered, counseled and treated

Pregnancy:

- Women with PCOS who desire a pregnancy may be at increased risk for adverse pregnancy outcomes, and this may be exacerbated by obesity and/or insulin resistance (level B).
- Health should be optimized before conception, with advice about smoking cessation, lifestyle, diet, and appropriate vitamin supplementation (e.g., folic acid) (GPP).
- Women with PCOS should be observed closely during pregnancy as they may be at increased risk for the development of GDM, gestational hypertension, and associated complications (level B).
- Pregnancy-associated risks are greater in women diagnosed by more classic (NIH) criteria as opposed to nonhyperandrogenic women (level B).

Obesity

- Prevalence is increasing and has an important influence on the phenotype of PCOS.
- Is associated with greater insulin resistance, IM and HA
- Lifestyle management results in weight loss and improves surrogate markers of MetS (Level A)
- (Screening: BMI and waist circumference)

Infertility

- Should be managed along the standard clinical practice
- Obesity adversely affects the clinical outcomes and lifestyle management is of importance
- Those with HA and chronic amenorrhoea – more resistant to ovarian stimulation and ?lower pregnancy rates

IR, DM and Met S

- IR is an important component of PCOS; most often seen in classic / NIH PCOS phenotype
- Precursor for various metabolic consequences including T2D and metabolic syndrome.
- Screening for DM – in adolescent and adult PCOS

– in those with obesity / visceral adiposity / FH of DM

- Fasting and 2 hr OGTT (HbA1C); Repeat every 3-5 years.
- Diet and lifestyle are important preventive measures
- Metformin

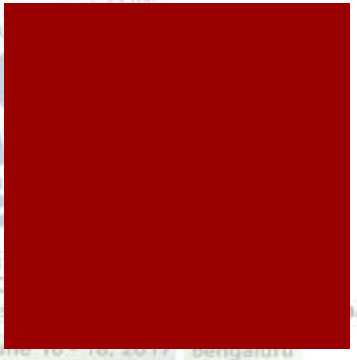
Cardiovascular Disease

At risk—PCOS women with any of the following risk factors:
- Obesity (especially increased abdominal adiposity)
- Cigarette smoking
- Hypertension
- Dyslipidemia (increased LDL-cholesterol and/or non-HDL-cholesterol)
- Subclinical vascular disease
- Impaired glucose tolerance
- Family history of premature cardiovascular disease (<55 y of age in male relative; <65 y of age in female relative)

At high risk–PCOS women with:
- Metabolic syndrome
- T2DM
- Overt vascular or renal disease, cardiovascular diseases
- OSA

- The recommended CVD risk assessment at any age is for psychosocial stress, blood pressure, glucose, lipid profile (cholesterol, triglycerides, HDL, LDL, and non-HDL cholesterol), waist circumference, physical activity, nutrition, and smoking (level C).
- Because CVD risk increases with age and accompanying additive environmental insults, periodic reassessment for CVD risk is recommended (GPP).

Endometrial Carcinoma



- No specific recommendations for screening
- Based on age, length of amenorrhea, dysfunctional uterine bleeding and endometrial thickness.

Conclusions

- PCOS may present with different phenotypes in young adulthood.
- Metabolic dysfunctions are more severe in those with HA.
- However, obesity and increasing age may obliterate the distinction between various phenotypes.
- Pregnancy complications and psychological disorders occur with similar frequency in all phenotypes.

Conclusions

- Lifestyle modification with diet and exercise
- Metformin
- Screening – BMI, waist circumference, BP, acanthosis, OGTT, (lipid profile) and re-assessment at regular intervals
- Symptomatic treatment
- Vigilance for long term risks
- Improve awareness regarding risk for mothers, siblings and offspring.

Thank You

Diabetes Care. 2008 June ; 31(6): 1237–1241. doi:10.2337/dc07-2190.

Metabolic Phenotype in the Brothers of Women with Polycystic Ovary Syndrome

Susan Sam, MD[1], **Andrea D. Coviello, MD**[2], **Yeon-ah Sung, MD**[3], **Richard S. Legro, MD**[4], and **Andrea Dunaif, MD**[1]

Male Phenotype

OBJECTIVE—Hyperandrogenemia, insulin resistance, and dyslipidemia demonstrate familial aggregation in the female first-degree relatives of women with polycystic ovary syndrome (PCOS), suggesting that these defects are heritable. Hyperandrogenemia also appears to be the male reproductive phenotype. We performed this study to test the hypothesis that brothers of women with PCOS have metabolic defects similar to those of their proband sisters.

RESEARCH DESIGN AND METHODS—This was a prospective case-control study performed at four academic medical centers in the U.S. Fasting blood was obtained from 196 non-Hispanic white brothers of women with PCOS and 169 control men of age, BMI, and ethnicity comparable to those of brothers. A separate analysis was performed by study site to assess potential regional variations in metabolic parameters.

RESULTS—Overall, brothers of women with PCOS had significantly higher total ($P = 0.001$) and LDL cholesterol ($P = 0.01$) as well as triglyceride levels ($P = 0.01$) compared with control men, although there were regional variations in these differences. There were significant positive correlations between brothers and their sisters with PCOS for total ($\rho = 0.2$, $P = 0.009$) and LDL cholesterol ($\rho = 0.3$, $P = 0.001$) and triglyceride ($\rho = 0.2$, $P = 0.05$) levels. Brothers also had significantly higher fasting insulin levels and homeostatic index of insulin resistance ($P = 0.02$ for both comparisons) compared with control men.

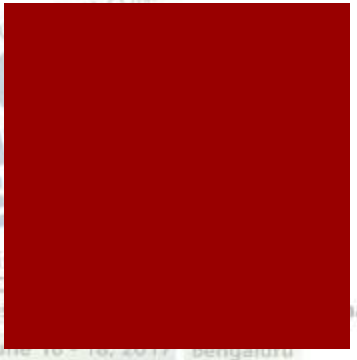
CONCLUSIONS—Brothers of women with PCOS have dyslipidemia as well as evidence for insulin resistance similar to that of their proband sisters with PCOS. These findings are consistent with the hypothesis that some metabolic abnormalities in PCOS are heritable and are not sex specific.

Dyslipidemia and Metabolic Syndrome in the Sisters of Women with Polycystic Ovary Syndrome

Susan Sam,* Richard S. Legro,* Rhonda Bentley-Lewis, and Andrea Dunaif

Conclusions: Low-density lipoprotein levels are increased in affected sisters of women with PCOS consistent with a heritable trait. The prevalence of metabolic syndrome is increased in affected sisters.
(***J Clin Endocrinol Metab*** **90: 4797–4802, 2005)**

PCOS and Sisters



- J Clin Endocrinol Metab. 2005 May;90(5):2545-9. Epub 2005 Feb 22.
- **Phenotypic variation in hyperandrogenic women influences the findings of abnormal metabolic and cardiovascular risk parameters.**
- Carmina E1, Chu MC, Longo RA, Rini GB, Lobo RA.
- **Author informatio**
- **Abstract**
- In hyperandrogenic women, several phenotypes may be observed. This includes women with classic polycystic ovary syndrome (C-PCOS), those with ovulatory (OV) PCOS, and women with idiopathic hyperandrogenism (IHA), which occurs in women with normal ovaries. Where other causes have been excluded, we categorized 290 hyperandrogenic women who were seen consecutively for this complaint between 1993 and 2004 into these three subgroups. The aim was to compare the prevalence of obesity, insulin resistance, and dyslipidemia as well as increases in C-reactive protein and homocysteine in these different phenotypes with age-matched ovulatory controls of normal weight (n = 85) and others matched for body mass index (BMI) with women with C-PCOS (n = 42). Although BMI affected fasting serum insulin and the Quantitative Insulin-Sensitivity Check Index, these markers of insulin resistance were greatest in C-PCOS (n = 204), followed by OV-PCOS (n = 50) and then IHA (n = 33). Androgen levels were similar in OV-PCOS and IHA but were higher in C-PCOS, whereas gonadotropins were similar in all groups. Lipid abnormalities were highest in C-PCOS and OV-PCOS and were normal in IHA. C-reactive protein was elevated in C-PCOS and OV-PCOS but not IHA. Homocysteine was elevated only in C-PCOS. Overall, the prevalence of obesity (BMI > 30) was 29% in C-PCOS, 8% in OV-PCOS, and 15% in IHA and insulin resistance (Quantitative Insulin-Sensitivity Check Index < 0.33) was 68% in C-PCOS, 36% in OV-PCOS, and 26% in IHA. The prevalence of having at least one elevated cardiovascular risk marker was 45% in C-PCOS 38% in OV-PCOS and was not increased on IHA (6%). These results suggest that among hyperandrogenic women the prevalence of abnormal metabolic and cardiovascular risk parameters is greatest in C-PCOS, followed by OV-PCOS and then women with IHA. Moreover, in that in OV-PCOS and IHA, ages and weights were similar yet the prevalence of metabolic and cardiovascular risk was greater in OV-PCOS, the finding of polycystic ovaries may be a significant modifying factor.

J Clin Endocrinol Metab. 2005 May;90(5):2571-9. Epub 2005 Feb 15.

Polycystic ovaries are common in women with hyperandrogenic chronic anovulation but do not predict metabolic or reproductive phenotype.

Legro RS1, Chiu P, Kunselman AR, Bentley CM, Dodson WC, Dunaif A.

Author information

Abstract

Polycystic ovary syndrome (PCOS) is a heterogeneous disorder of unexplained hyperandrogenic chronic anovulation. Experts have recommended including the morphology and volume of the ovary in the diagnostic criteria for PCOS. We performed this study to determine whether there was an association between the morphology and size of the ovaries and markers of insulin sensitivity as determined by dynamic testing within women with PCOS or compared with a group of control women. We then examined reproductive parameters. We studied 88 unrelated PCOS women and 21 control women, aged 17-45 yr. All were in the early follicular phase or its equivalent (no follicle with > 10 mm diameter and anovulatory serum progesterone level < 3 ng/ml). Subjects underwent on the same day a phlebotomy for baseline hormones, a 2-h oral glucose tolerance test, and transvaginal ultrasound to determine the morphology and volume of the ovaries. Ninety-five percent (84 of 88) of women with PCOS and 48% (10 of 21) of the control women had polycystic ovaries using the criteria of at least one ovary greater than 10 cm3 (PCOV) and/or polycystic ovary morphology (PCOM) using the criteria of 10 or more peripheral follicular cysts 8 mm in diameter or less in one plane along with increased central ovarian stroma. PCOM was a better discriminator than PCOV between PCOS and control women. The odds of women with PCOS having PCOM were elevated 50-fold compared with controls (odds ratio, 50; 95% confidence interval, 10-240; P < 0.0001), whereas the odds of PCOV were elevated 5-fold in women with PCOS (odds ratio, 4.6; 95% confidence interval, 1.7-12.6; P = 0.003). Neither the insulin sensitivity index, fasting or 2-h values, or any integrated measures of glucose and insulin varied in women according to either morphology or volume, nor was there an association with circulating androgen levels. Women with PCOS and PCOM had lower FSH levels than women with PCOS and non-PCOM. Women with PCOS and PCOV had a higher LH to FSH ratio than women without PCOV and PCOS. These data support the hypothesis that polycystic ovaries are an abnormal finding. However, neither the morphology nor the volume of the ovaries is associated with distinctive metabolic or reproductive phenotypes in women with PCOS.

Diagnosis of PCOS: New Consensus

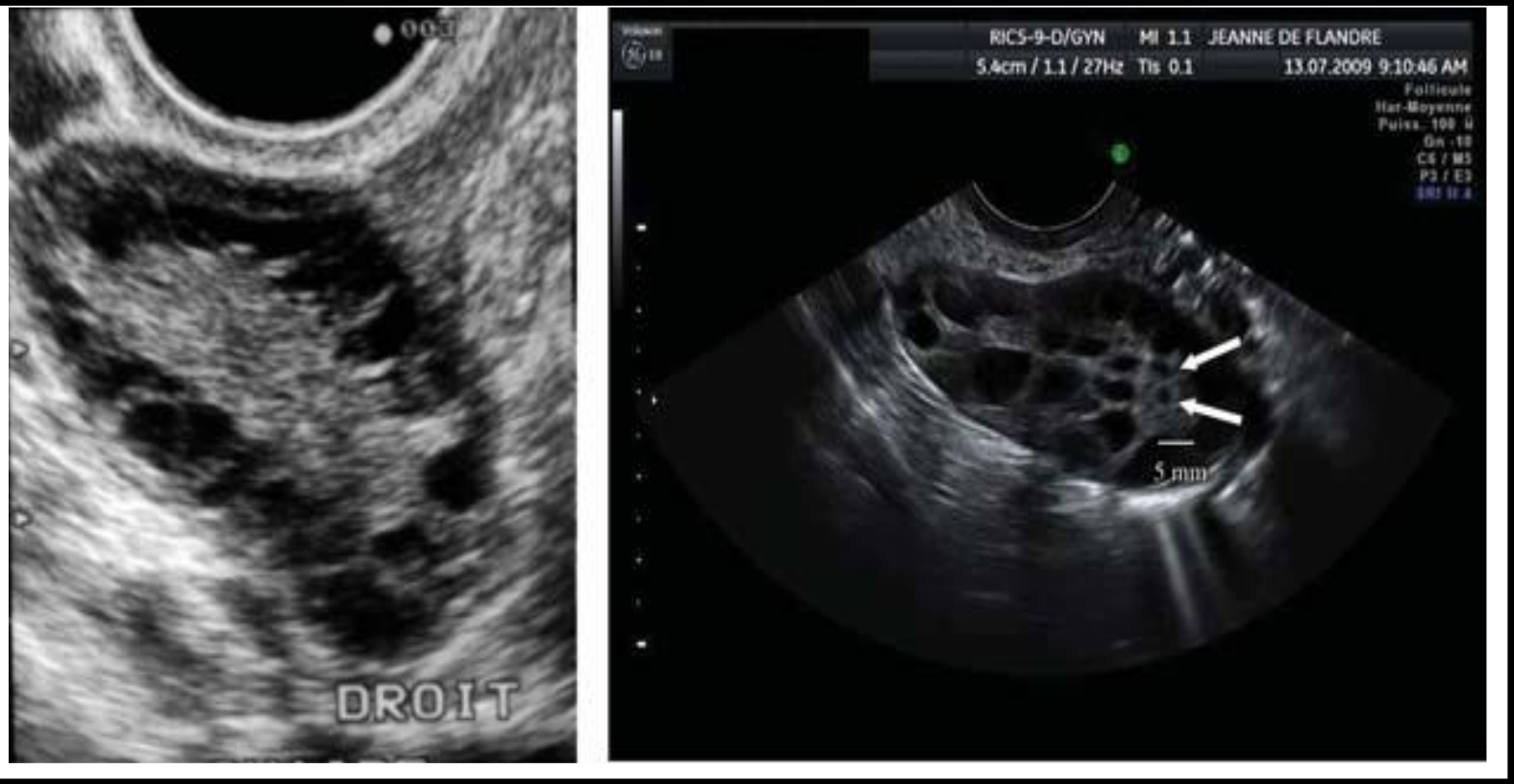
Dr. Padma Rekha Jirge MRCOG(UK), FICOG, MBA (Healthcare Mx)

Shreyas Hospital & Sushrut Assisted Conception Clinic,

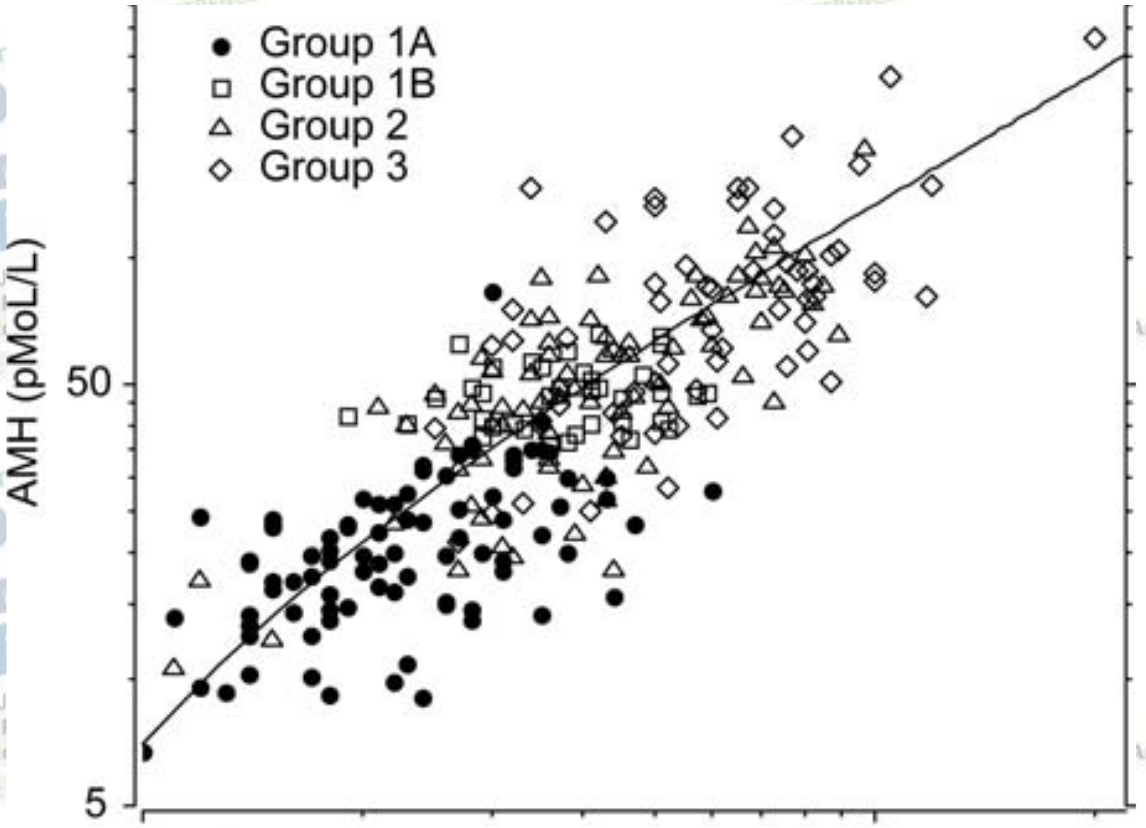
Kolhapur

KISAR 30 Apr-1 May 2016, Bangalore

Follicular numbers



Correlation bet FN and AMF



Correlation between FN and serum AMH

duction, Vol.27, No.8 pp. 2494–2502, 2012
publication on June 12, 2012 doi:10.1093/humrep/des213

ction

ORIGINAL ARTICLE *Reproductive endocrinology*

Anti-Mullerian hormone in the diagnosis of polycystic ovary syndron can morphologic description be replaced?

Tina B. Eilertsen[1,2,*], Eszter Vanky[2,3], and Sven M. Carlsen[4,5]

[1]Department of Obstetrics and Gynaecology, Hospital of Namsos, Nord-Troendelag Hospital Trust, Namsos, Norway [2]Depart Laboratory Medicine, Children's and Women's Health, Norwegian University of Science and Technology, Trondheim, Norway [3]D of Obstetrics and Gynaecology, University Hospital of Trondheim, Trondheim, Norway [4]Unit for Applied Clinical Research, Dep Cancer Research and Molecular Medicine, Faculty of Medicine, Norwegian University of Science and Technology, Trondheim, N [5]Department of Endocrinology, St. Olavs Hospital, University Hospital of Trondheim, Trondheim, Norway

In conclusion, PCOM can be replaced by AMH when diagnosing PCOS, both according to the PCOS-R criteria and the PCOS-AES criteria. Sensitivity and specificity is high even at low AMH levels. Future studies should use universally accepted methods for AMH measurements and international standards should be established. If a high sensitivity and specificity is confirmed by others, AMH may replace US examination of the ovaries in PCOS diagnosis.

Table II Sensitivity and specificity of newly proposed diagnostic thresholds for FNPO, FNPS and OV.

Criterion	Area under ROC curve (95% CI)	Threshold	Sensitivity (%)	Specificity (%)
FNPO	0.969* (0.948, 0.990)	12[a]	100	36
		15[b]	99	54
		19[c]	96	77
		20[d]	96	79
		26	**85**	**94**
FNPS	0.880* (0.830, 0.930)	**9**	**69**	**90**
		10[d,e]	58	94
OV (cm^3)	0.873* (0.817, 0.930)	7[c,f]	95	53
		8[g]	93	61
		9[h]	88	71
		10	**81**	**84**
		11[i]	74	86
		13[j]	60	90

ROC curve, receiver operating characteristic curve.
Comparison with previously reported thresholds provided:
[a]Jonard *et al.* (2003);
[b]Fox (1999);
[c]Dewailly *et al.* (2011);
[d]Allemand *et al.* (2006);
[e]Adams *et al.* (1985);
[f]Jonard *et al.* (2005);
[g]Chen *et al.* (2008);
[h]Atiomo *et al.* (2000); [i]van Santbrink *et al.* (1997);
[j]Fulghesu *et al.* (2001).
*$P < 0.0001$ compared with chance alone.

Table III **Adaptation of the previous classifications for the diagnosis of PCOS, proposing an excessive FN of >19 or serum AMH concentration >35 pmol/l or >5 ng/ml as a surrogate when either oligo-anovulation or HA is missing.**

Oligo -anovulation	Clinical and/or biological HA	FN > 19 and/or serum AMH[a] > 35 pmol/l (5 ng/ml)	Diagnosis
+	+	(+/−)[b]	PCOS
+	−	+	PCOS
−	+	+	PCOS
−	−	+	Normal woman with PCOM[c]
+	−	−	Idiopathic anovulation
−	+	−	Idiopathic hyperandrogenism

As with the previous classifications, other causes of oligo-anovulation and/or HA must be excluded before applying this classification.

[a]To be used preferentially.

[b]Not necessary for the diagnosis.

[c]Consider the risk for OHSS.

Reproduction, Vol.29, No.4 pp. 791–801, 2014
Access publication on January 16, 2014 doi:10.1093/humrep/det469

man
production

ORIGINAL ARTICLE *Reproductive endocrinology*

The prevalence of polycystic ovary syndrome in a normal population according to the Rotterdam criteria versus revised criteria including anti-Müllerian hormone

M.P. Lauritsen[1,*], J.G. Bentzen[1], A. Pinborg[1], A. Loft[1], J.L. Forman[2], L.L. Thuesen[1], A. Cohen[3], D.M. Hougaard[3], and A. Nyboe Andersen[1]

[1]The Fertility Clinic, Section 4071, Copenhagen University Hospital, Rigshospitalet, DK-2100 Copenhagen, Denmark [2]Department of Biostatistics, University of Copenhagen, DK-1014 Copenhagen, Denmark [3]Department of Clinical Biochemistry and Immunology, Statens Ser Institute, DK-2300 Copenhagen, Denmark

To conclude, our data confirm that AMH is a reliable marker of polycystic ovaries in PCOS. Furthermore, prevalence estimates in our study population indicate a need of revision of the Rotterdam criterion for polycystic ovaries. The AFC and AMH criteria proposed by Dewailly *et al*. diminished the prevalence of PCOS in our study population to a more appropriate figure. However, future studies are required to validate the AMH threshold level. A revision of the Rotterdam criteria should also include age adjustments to avoid overdiagnosis of PCOS in young

Additional Assessment

- Insulin Resistance
- Metabolic Syndrome

Conclusions

- Rotterdam criteria and AE-PCOS society criteria have expanded the diagnosis of PCOS
- USG parameters have been under constant scrutiny with changing technological aspects
- With the availability of fully automated assays for AMH, there is a valid reason for it to be included as a diagnostic criteria.
- Any defined phenotype should be a guiding factor regarding long-term health concerns

Areas of Concern

- USG criteria - ? Need revised

No stromal measurement / doppler parameters

?3D USG

Lam et al. **Human Reproduction Vol.21, No.9 pp. 2209–2215, 2006**

- AMH

mark-type=disc
Chronic anovulation
Clinical and/or biochemical signs of hyperandrogenism

(Exclusion of other etiologies) Both criteria are necessary to establish diagnosis list-behavior=unordered prefix-word= mark-type=disc
Oligo- and/or anovulation
Clinical and/or biochemical signs of hyperandrogenism
Polycystic ovaries

(Exclusion of other etiologies) Two of three criteria are necessary to establish diagnosis list-behavior=unordered prefix-word= mark-type=disc
Clinical and/or biochemical signs of hyperandrogenism
Ovarian dysfunction (oligo- and/ or anovulation) and/or polycystic ovaries

(Exclusion of other etiologies) Both criteria are necessary to establish

Fasting and post−oral glucose load glucose and insulin levels and the insulin resistance indexes in PCOS women with GI states and in obese and normal-weight PCOS women with NGT

Parameters	GI	OB-NGT	NW-NGT
n	22	80	19
Fasting values			
Glucose (mmol/l)	5.52 ± 0.57	4.7 ± 0.57*	4.5 ± 0.43*
Insulin (pmol/l)	136 ± 73.9	100 ± 59.6†	57.2 ± 59.9*‡
C-peptide (nmol/l)	1.60 ± 0.58	1.34 ± 1.08	0.65 ± 0.22§‡
AUC values			
Glucose (mmol · l^{-1} · min^{-1})	1,580 ± 173	1,064 ± 130*	1,062 ± 186*
Insulin (pmol · l^{-1} · min^{-1})	154,334 ± 94,069	78,618 ± 54,915*	56,065 ± 34,301*
C-peptide (nmol · l^{-1} · min^{-1})	689 ± 212	529 ± 189§	440 ± 146*‖
QUICKI	0.30 ± 0.02	0.33 ± 0.24§	0.37 ± 0.05*¶
$HOMA_{OGTT}$	2.05 ± 1.08	4.40 ± 2.25*	7.58 ± 3.96*¶

Data are means ± SD. †$P < 0.05$, §$P < 0.01$, *$P < 0.001$ for GI vs. OB-NGT or NW-NGT; ‖$P < 0.05$, ‡$P < 0.01$, ¶$P < 0.001$ for OB-NGT vs. NW-NGT.

Phenotypes				Exclusion
1	2	3	4	
Clinical HA +Oligo-ovulation	Biochemical HA + Oligo-ovulation	-	-	CAH, androgen secreting tumours, hyperprolactinemia, Cushing's syndrome
Clinical/biochemHA+ Oligo-ovulation	Clinical/biochem HA+ Oligoovulation+PCOM	Clinical/Biochem HA + PCOM	Oligo-ovulation +PCOM	CAH, androgen secreting tumours, Cushing's syndrome
Clinical/biochem HA+Oligo-ovulation	Clinical/biochem HA+PCOM	-	-	As in NIH + androgenic drugs, syndromes ofsevere insulin resistance, thyroid dysfunction

- hyperplasia, thyroid dysfunction, hyperprolactinemia, neoplastic androgen secretion,
- or drug-induced androgen excess.

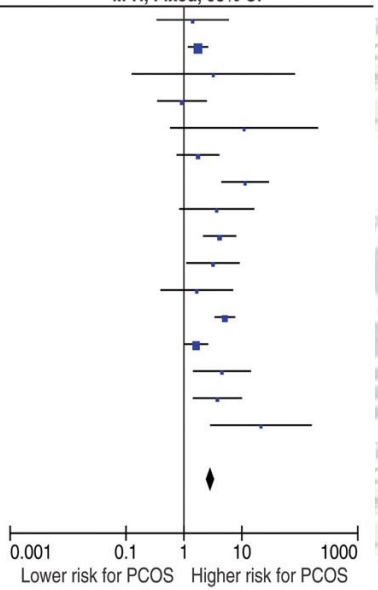
Metabolic syndrome prevalence (A) in women with and without PCOS and subgroup meta-analysis (B) of metabolic syndrome prevalence in women with and without PCOS with BMI-matched study populations.

A

Study or Subgroup	PCOS Events	PCOS Total	Control Events	Control Total	Weight	Odds Ratio M-H, Fixed, 95% CI	Year
Faloia 2004	10	50	3	20	2.4%	1.42 [0.35, 5.80]	2004
Dokras 2005	45	129	442	1887	26.0%	1.75 [1.20, 2.55]	2005
Vrbikova 2005	1	69	0	73	0.3%	3.22 [0.13, 80.36]	2005
Alvarez-Blasco 2006	8	32	19	72	6.2%	0.93 [0.36, 2.42]	2006
Leibel 2006	7	36	0	21	0.4%	10.93 [0.59, 201.92]	2006
Welt 2006	74	418	7	64	7.1%	1.75 [0.77, 3.99]	2006
Coviello 2006	18	49	8	165	1.6%	11.40 [4.55, 28.53]	2006
Carmina 2006	23	282	2	85	2.0%	3.69 [0.85, 15.96]	2006
Shroff 2007a	92	258	13	110	8.3%	4.14 [2.20, 7.78]	2007
Caliskan 2007	15	182	5	182	3.2%	3.18 [1.13, 8.94]	2007
Shroff 2007b	6	24	4	24	2.1%	1.67 [0.40, 6.87]	2007
Cussons 2008	62	168	91	883	13.0%	5.09 [3.48, 7.45]	2008
Shaw 2008	58	104	125	286	20.8%	1.62 [1.03, 2.55]	2008
Attuoua 2008	17	107	4	100	2.5%	4.53 [1.47, 13.98]	2008
Gulcelik 2008	20	60	7	60	3.3%	3.79 [1.46, 9.82]	2008
Cheung 2008	52	288	1	98	0.9%	21.37 [2.91, 156.79]	2008
Total (95% CI)		**2256**		**4130**	**100.0%**	**2.88 [2.40, 3.45]**	
Total events	508		731				

Heterogeneity: Chi² = 45.40, df = 15 (P < 0.0001); I² = 67%

Test for overall effect: Z = 11.51 (P < 0.00001)

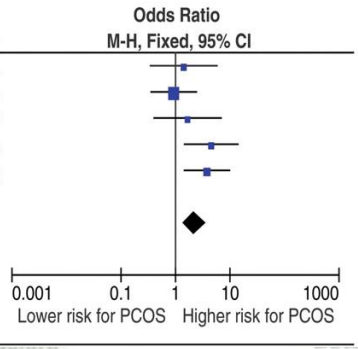


B

Study or Subgroup	PCOS Events	PCOS Total	Control Events	Control Total	Weight	Odds Ratio M-H, Fixed, 95% CI	Year
Faloia 2004	10	50	3	20	14.7%	1.42 [0.35, 5.80]	2004
Alvarez-Blasco 2006	8	32	19	72	37.6%	0.93 [0.36, 2.42]	2006
Shroff 2007b	6	24	4	24	12.9%	1.67 [0.40, 6.87]	2007
Attuoua 2008	17	107	4	100	14.9%	4.53 [1.47, 13.98]	2008
Gulcelik 2008	20	60	7	60	20.0%	3.79 [1.46, 9.82]	2008
Total (95% CI)		**273**		**276**	**100.0%**	**2.20 [1.36, 3.56]**	
Total events	61		37				

Heterogeneity: Chi² = 6.47, df = 4 (P = 0.17); I² = 38%

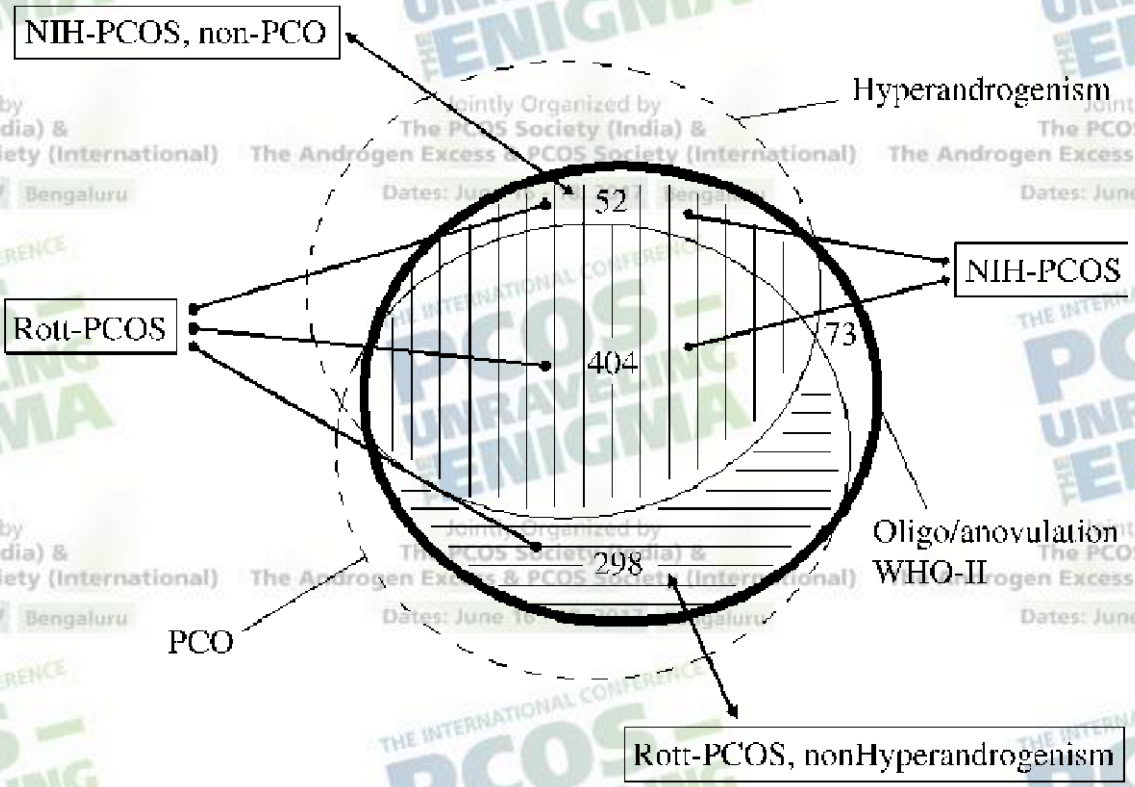
Test for overall effect: Z = 3.23 (P = 0.001)



Moran L J et al. Hum. Reprod. Update 2010;16:347-363

human reproduction update

PCOS according to the Rotterdam consensus criteria



PCOS and Phenotypes

Comparison of hormonal and metabolic parameters between the three PCOS phenotypes.

	Oligo+HA+Hirsutism (n = 153)	Oligo+HA (n = 92)	Oligo+Hirsutism (n = 71)
Total testosterone (ng/dL)	97.3 ± 52.9	93.5 ± 32.8	52.2 ± 15.8[a]
Free testosterone (ng/dL)	1.08 ± 0.50	0.97 ± 0.30	0.55 ± 0.15[a]
SHBG (nmol/L)	164.4 ± 56.9	169.5 ± 45.9	167.1 ± 43.0
DHEAS (ng/mL)	2,286.9 ± 1202.6	2,062.4 ± 943.5	1360.5 ± 569.7[a]
Fasting glucose (mg/dL)	89.0 ± 20.7	91.5 ± 12.3	88.2 ± 11.3
Fasting insulin (mcU/mL)	23.8 ± 18.0[b]	19.3 ± 14.3	17.6 ± 10.9[b]
HOMA-IR (mol μU/mL)	5.21 ± 4.25	4.48 ± 3.49	3.92 ± 2.63
HOMA-β-cell (%)	148.0 ± 111.9[b]	120.4 ± 89.4	109.2 ± 67.9[b]

Note: SHBG = sex hormone–binding globulin; DHEAS = dehydroepiandrosterone sulfate; HOMA-IR, HOMA-β-cell = insulin resistance and percent β-cell function estimated from the homeostatic assessment model, as previously described (16). See Table 1 for key to remaining abbreviations. All comparisons for continuous variables, other than age, were performed using ANOVA with Tukey post-hoc tests, using age-adjusted variables.

[a] Different from the other two phenotypes ($P<.002$).

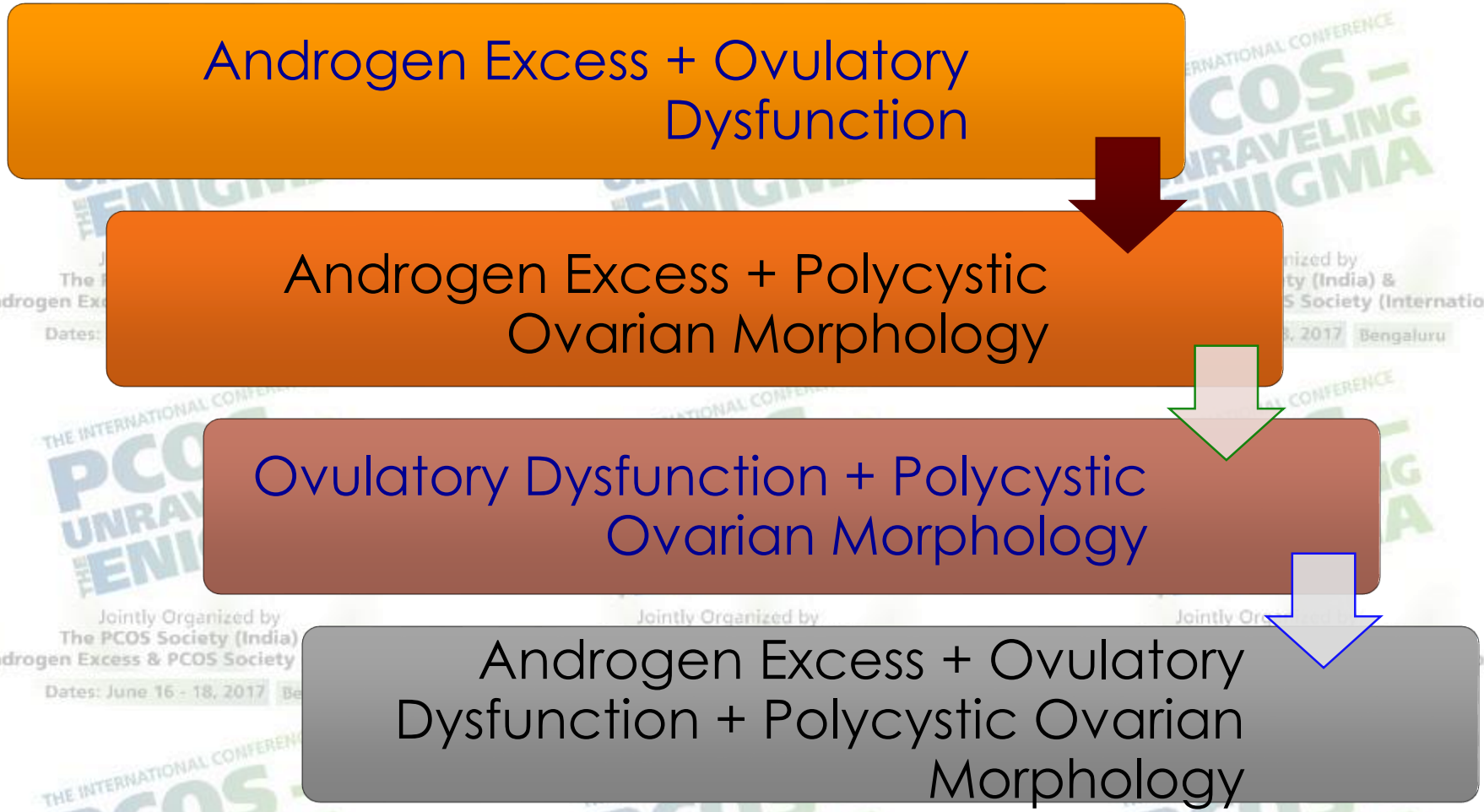
[b] Oligo+HA+Hirsutism different from Oligo+Hirsutism patients ($P<.05$); Oligo+HA not different from other phenotypes.

Chang. Phenotypes of PCOS. Fertil Steril 2005.

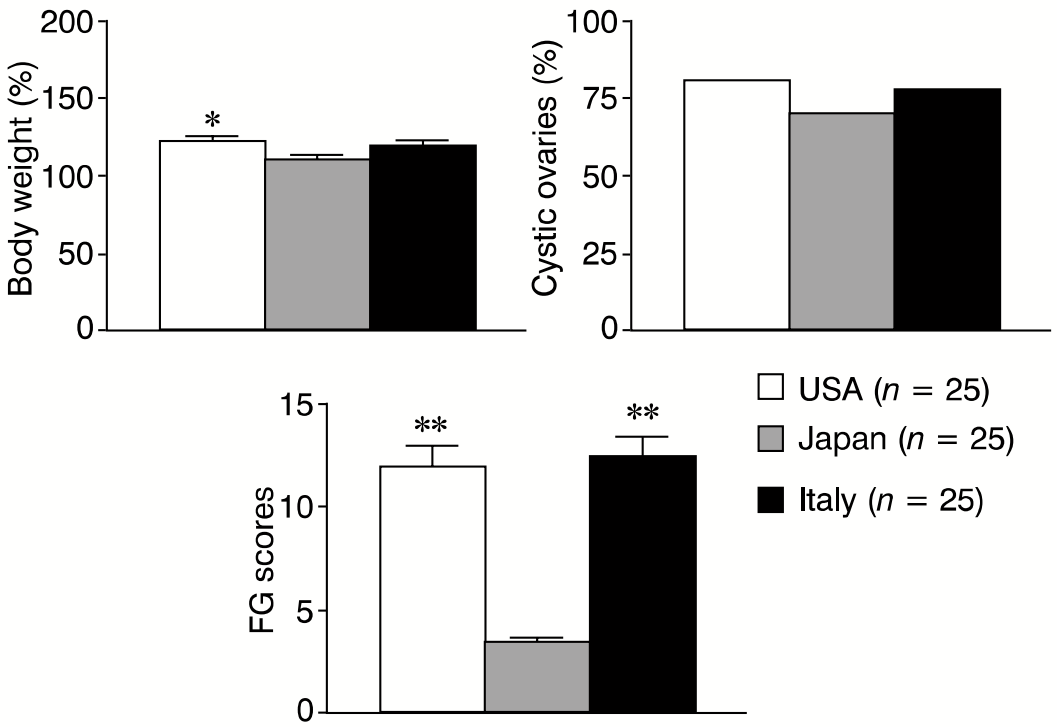
severe form of metabolic dysfunction
at an early age.
Strong correlation between

NIH workshop 2012, Draft statement

Recommend maintaining the broad, inclusionary diagnostic criteria of Rotterdam (which includes the "classic NIH" and AE-PCOS criteria) while specifically identifying the phenotype:



Ethnic Differences



Carmina et al 1992

J Clin Endocrinol Metab. 2006 Dec;91(12):4842-8. Epub 2006 Sep 26.

Characterizing discrete subsets of polycystic ovary syndrome as defined by the Rotterdam criteria: the impact of weight on phenotype and metabolic features.

Welt CK1, Gudmundsson JA, Arason G, Adams J, Palsdottir H, Gudlaugsdottir G, Ingadottir G, Crowley WF.

CONTEXT:
The Rotterdam criteria for polycystic ovary syndrome (PCOS) defines discrete subgroups whose phenotypes are not yet clear.

OBJECTIVE:
The phenotypic characteristics of women in the PCOS subgroups defined by the Rotterdam criteria were compared.

DESIGN:
The study was observational.

SETTING:
Subjects were studied in an outpatient setting in Boston and Reykjavik.

PATIENTS:
Four subgroups of subjects with PCOS defined by 1) irregular menses (IM), hyperandrogenism (HA), and polycystic ovary morphology (PCOM, n = 298); 2) IM/HA (n = 7); 3) HA/PCOM (n = 77); and 4) IM/PCOM (n = 36) and a group of controls (n = 64), aged 18-45 yr, were examined.

INTERVENTION:
Subjects underwent a physical exam; fasting blood samples for androgens, gonadotropins, and metabolic parameters; and a transvaginal ultrasound.

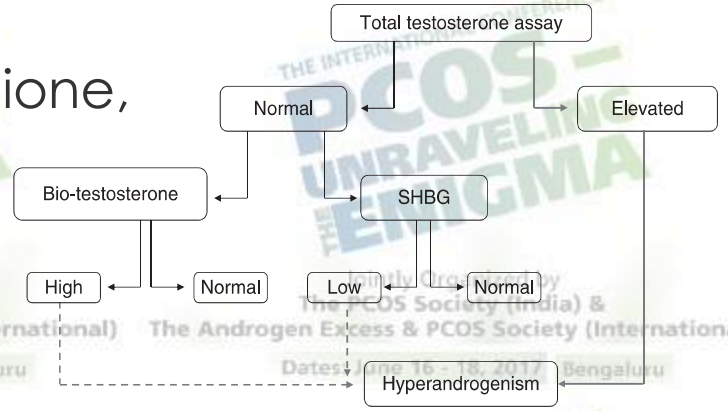
MAIN OUTCOME MEASURES:
The phenotype was compared between groups.

RESULTS:
Ninety-seven percent of women with IM/HA had PCOM. Therefore, the groups with

Concerns and Criticism

- Oligo/anovulation: role in evaluating in non-infertile women
- Hyperandrogenism: hirsuitism, acne, alopecia – not universal
- Hyperandrogenaemia: which tests to do?

(Testo, SHBG, FAI; 17-OH P, androstenedione, DHEAS)



Concerns and Criticism Contd

- PCO: follicular number vary with age.
- Prognostic features – obesity, IR
- Role of age and ethnicity