From PCOS to PMOS: Reframing a Common Female Endocrine Disorder in the GCC

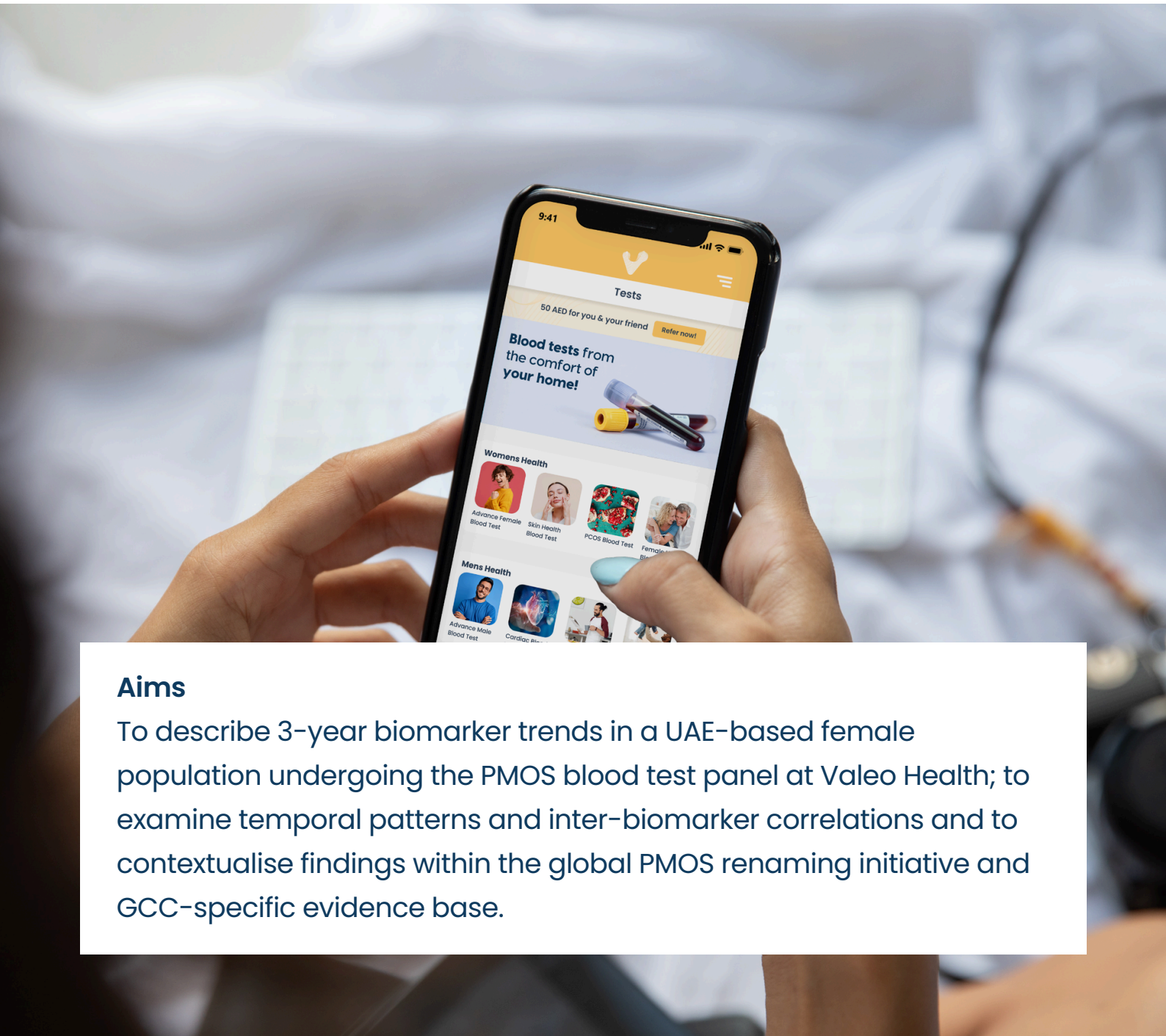
Temporal Patterns & a 3-Year Hypothesis-Generating Observational Report from a UAE-Based Testing Program

May Chalhoub, M.Sc Human Nutrition

Abstract

Background

Polycystic ovary syndrome (PCOS), recently renamed Polyendocrine Metabolic Ovarian Syndrome (PMOS) by global consensus (Teede et al., The Lancet, 2026), affects an estimated one in eight women worldwide. In the Gulf Cooperation Council (GCC), up to 30% of infertile women carry the diagnosis (Alam et al., Heliyon, 2024), yet real-world, community-based biomarker data from this region remain scarce.



Aims

To describe 3-year biomarker trends in a UAE-based female population undergoing the PMOS blood test panel at Valeo Health; to examine temporal patterns and inter-biomarker correlations and to contextualise findings within the global PMOS renaming initiative and GCC-specific evidence base.

Methods

Retrospective analysis of 176 de-identified orders from 166 unique female patients across three chronological cohorts . Eleven core biomarkers were analysed. Statistical tests: Kruskal-Wallis H, Spearman rank correlations, and multivariable OLS regression.

Results

Five biomarkers showed significant temporal variation. FT4 demonstrated the strongest upward trend (Spearman $\rho = +0.49$, $p < 0.0001$; regression $\beta = +0.13$ ng/dL/period, $p < 0.0001$). LH showed a significant declining trajectory ($\rho = -0.19$, $p = 0.010$). Free testosterone rose modestly ($\rho = +0.22$, $p = 0.004$). Classical PMOS correlations were confirmed: SHBG inversely associated with insulin ($\rho = -0.38$) and free testosterone ($\rho = -0.40$). Body weight independently predicted HbA1c and fasting insulin.

Conclusion

This report provides the first real-world, direct-to-consumer PMOS biomarker dataset from the UAE. A thyroid-PMOS axis — signalled by the FT4 trend and its mechanistic support in the literature — warrants prospective investigation. Findings support adoption of the PMOS framework and the value of longitudinal community-based hormonal monitoring in high-risk GCC populations.

Background

The Global Naming Problem

Polycystic ovary syndrome (PCOS) is the most prevalent endocrine disorder in reproductive-age women, yet its name is a misnomer — the term implies pathological ovarian cysts, a feature that is not definitively present in most cases. In May 2026, Teede and colleagues published a landmark global consensus in The Lancet, formally renaming the condition Polyendocrine Metabolic Ovarian Syndrome (PMOS). The new name was selected through iterative Delphi surveys with 14,360 responses from 56 organisations worldwide, endorsed by 84% of patients and health professionals alike.

> "The term polycystic ovary implies pathological ovarian cysts, which are not a feature of the condition. This misnomer contributes to misunderstandings among patients, clinicians, policy makers, and the public." — Teede et al., The Lancet, 2026

PMOS encompasses diverse endocrine, metabolic, reproductive, psychological, and dermatological features driven by dysregulation across insulin, androgen, neuroendocrine, and ovarian hormone axes. Valeo Health adopted the PMOS rebranding ahead of the global consensus, making it among the first commercial health platforms to implement the new nomenclature in practice.

Background

The GCC Context

Middle Eastern women have among the highest PCOS prevalence rates globally (16.0%, 95% CI: 13.8–18.6%). Alam et al. (Heliyon, 2024) conducted the first systematic review of PCOS among infertile women in the GCC, analysing seven studies across Saudi Arabia, Qatar, Kuwait, and Oman. Their meta-analysis found a pooled prevalence of 30.0% (95% CI: 29.0–38.0%), rising to 59% in women aged ≥35. Critically, no UAE or Bahrain data existed in this review — a gap this report directly addresses.

30%

Pooled PCOS prevalence among infertile GCC women

59%

Prevalence in women aged ≥35 in the GCC

The Thyroid-PMOS Connection

Fan et al. (Frontiers in Endocrinology, 2023) established that the risk of thyroid disease in PCOS/PMOS patients is 2.5× higher than controls. Subclinical hypothyroidism (SCH) affects up to 43.5% of PMOS patients vs. 20.5% of controls. Thyroid hormones regulate SHBG synthesis, androgen metabolism, LH pulsatility, and ovarian folliculogenesis. Despite this, free thyroxine (FT4) is rarely included in standard PMOS panels. Our observational data — showing FT4 as the most robust temporal signal — provides grounds to reconsider this omission.

STUDY METHODOLOGY

Study Design

Retrospective observational analysis of PMOS blood test conducted via Valeo Health. Presented as a hypothesis-generating report per observational study conventions.

166	176	11
Unique female pateitns	Total orders analyzed	Core biomarkers measured

Timeframe

January, 2022 — June, 2026

Biomarker Panel

Biomarker	Abbreviation	Unit	Axis
Follicle Stimulating Hormone	FSH	mIU/ml	Gonadotropin
Luteinizing Hormone	LH	mIU/ml	Gonadotropin
Free Testosterone	Free-T	pg/ml	Androgen
Sex Hormone Binding Globulin	SHBG	nmol/L	Androgen transport
Prolactin	PRL	uIU/ml	Neuroendocrine
Glucose Fasting	Glucose	mg/dL	Metabolic
Hemoglobin A1C	HbA1c	%	Metabolic
Insulin Fasting	Insulin	uIU/mL	Metabolic
Thyroid Stimulating Hormone	TSH	uIU/mL	Thyroid
Free T3	FT3	uIU/mL	Thyroid
Free T4	FT4	uIU/mL	Thyroid

Statistical Analysis

- Between-period differences: Kruskal-Wallis H test
- Temporal trends: Spearman ρ vs. order ID
- Age-biomarker associations: Spearman ρ vs. patient age
- Inter-biomarker associations: Spearman correlation matrix
- Multivariable associations: OLS regression (period + weight)
- Significance threshold: $\alpha = 0.05$, two-tailed

Results

Patient Demographics & Age Distribution

33.2	32	18–56
Mean age (years) ± SD 6.8	Median age (years)	Age range (years)

The cohort is predominantly composed of women in their prime reproductive years. The modal age decade is 30–34 years, accounting for 36.9% of the cohort. Seventy-five percent of patients fall within the 25–39 age band, consistent with the peak PMOS/PCOS diagnostic window. The age distribution narrows slightly over time (mean age declining from 34.3 in Year 1 to 32.1 in Year 3), suggesting progressive engagement of younger reproductive-age women with direct-to-consumer hormonal testing in the UAE.

Note on Female Hormonal Biomarkers

Female sex hormones (e.g. estrogen, progesterone, LH, FSH) were measured; however, interpretation is limited due to variability related to menstrual cycle phase, hormonal contraception, and peri- or post-menopausal status. These markers are therefore reported descriptively and interpreted with caution.

Results

Age-Biomarker Correlations

Biomarker	p-value	Direction	Clinical Interpreation
Free Testosterone	< 0.0001	↓ with age	Expected: free androgen levels decline across 4th-5th decade
Luteinizing Hormone	0.6035	not significant	No significant age effect — LH elevation in PMOS independent of age
SHBG	0.0400	↑ with age	SHBG rises with age as androgens decline and sex hormone binding increases
FSH	0.0422	↑ with age	Rising FSH with age — approaches menopausal transition in oldest patients
HbA1C	0.0643	Trending	Glycaemic index rises slightly with age; not significant
Free T4	0.2753	not significant	FT4 not significantly age-dependent — strengthens temporal trend hypothesis
Insulin	0.0242	↓ with age	Younger women show higher insulin — may reflect insulin-resistance phenotype prevalence in younger PMOS

Four of eleven biomarkers showed statistically significant Spearman correlations with patient age, consistent with known physiological changes across the reproductive lifespan.

Results

Temporal Biomarker Trends

Biomarker	Kruskal-Wallis H	p value	Spearman ρ (time)	p value	Direction
FSH	7.916	0.0191	-0.119	0.120	ns
Luteinizing Hormone	10.566	0.0051	-0.194	0.0101	Falling
SHBG	9.724	0.0077	-0.029	0.703	ns
Prolactin	11.331	0.0035	0.072	0.348	ns
Glucose	0.055	0.973	-0.028	0.711	ns
HbA1c	1.404	0.496	-0.081	0.288	ns
Insulin	3.848	0.146	-0.016	0.835	ns
Free-T	5.126	0.077	+0.222	0.0041	Rising
FT4	39.836	< 0.0001	+0.489	< 0.0001	Rising

Key finding: FT4 is NOT significantly correlated with age (p = 0.275), which means the temporal rise in FT4 seen across cohort periods cannot be explained by the slight age shift between cohorts. This strengthens the hypothesis that the FT4 trend reflects a true biological or population-level

Results

Biomarker Values by Age Group

Biomarker	18–24 (n=12)	25–29 (n=33)	25–29 (n=33)	35–39 (n=29)	40–44 (n=11)	45+ (n=11)
FSH	5.13	5.14	5.13	6.15	5.81	22.77
LH	12.65	11.20	7.78	8.75	6.74	26.48
SHBG	62.1	63.7	98.1	66.7	100.9	74.4
HbA1c	5.05	4.95	5.01	5.13	5.14	5.01
Insulin	12.25	8.65	7.15	7.96	6.15	7.61
Free-T	1.402	1.332	1.291	1.343	1.249	1.266
FT4	5.05	4.95	4.95	5.13	5.14	5.01

The table above highlights the most clinically meaningful biomarkers across age groups. FSH rises sharply in the 45+ group (mean 22.8 vs. 5.1–6.1 in younger groups), reflecting perimenopausal transition. Free Testosterone peaks in 18–24 year olds (mean 3.96 pg/ml) and declines progressively. LH shows a bimodal pattern — elevated in the youngest and oldest groups.

Discussion

FT4 Rise: Age-Independent and Clinically Meaningful

The most robust finding in this dataset — the consistent rise in FT4 across cohort periods ($\rho = +0.49$, $p < 0.0001$) — is now confirmed to be independent of the modest age shift between cohorts. Age explains none of the FT4 variance ($\rho = -0.088$, $p = 0.275$). This rules out the possibility that the older Year 1 cohort (mean age 34.3) drove a spurious FT4 elevation relative to the younger Year 3 cohort (mean 32.1). The temporal trend is therefore a genuine signal.

Hypothesis 1: Rising FT4 reflects a true population-level shift in the thyroid-ovarian axis in UAE females presenting for hormonal testing — consistent with the thyroid-PMOS interaction mechanism described by Fan et al. (2023). Prospective anti-TPO antibody testing is recommended to explore autoimmune thyroid disease as a driver.

FT4 Rise: Age-Independent and Clinically Meaningful

The strong inverse correlation between age and free testosterone ($\rho = -0.33$, $p < 0.0001$) and the positive correlation with SHBG ($\rho = +0.16$, $p = 0.040$) reproduce expected reproductive physiology: free androgen availability declines as SHBG rises across the 3rd to 5th decade. The elevated free testosterone in 18–24 year olds (mean 3.96 pg/ml vs. 1.76 in 40–44) highlights that the youngest women in the cohort carry the highest androgen burden — clinically consistent with PMOS being most biochemically active in early reproductive life.

Discussion

Insulin Highest in Youngest: A Metabolic Phenotype Signal

Fasting insulin was highest in 18–24 year olds (mean 12.25 uIU/mL) and declined with age ($\rho = -0.18$, $p = 0.024$), despite the expectation that insulin resistance worsens with age in the general population. This paradoxical pattern may reflect that the youngest PMOS presenters carry a more severe hyperinsulinaemic phenotype — consistent with the evidence that PMOS-related insulin resistance is intrinsic and BMI-independent, and is most pronounced in the early reproductive years.

Hypothesis 2: The elevated insulin in 18–24 year old PMOS presenters reflects intrinsic PMOS-driven hyperinsulinaemia at peak androgenic burden — a metabolic phenotype that may improve with age as the androgenic drive attenuates. This should be examined prospectively with HOMA-IR calculations.

FSH in the 45+ Group: Perimenopausal Signal

Mean FSH in the 45+ age group (22.77 mIU/ml) is dramatically elevated compared to all younger groups (5.1–6.2 mIU/ml). This likely reflects early perimenopausal transition rather than PMOS-related FSH pathology. This finding underscores the importance of collecting age and menstrual status in the prospective dataset — FSH should be interpreted in age-stratified context.

Positioning in the GCC Evidence Base

The mean age of 33.2 years places this cohort squarely within the reproductive-age window most heavily affected by PMOS in the GCC (30.0% pooled prevalence, 15–34 years). This dataset — now characterised by age, 11 biomarkers, and 3-year temporal depth — provides the snapshot of community-based, age-stratified PMOS biomarker evidence from the UAE, constituting a meaningful contribution to the GCC evidence base.

Limitations

- Weight missing in 47% of orders — limits multivariable regression power
- Only 6% of patients tested twice — within-person longitudinal analysis underpowered
- No clinical phenotype data (menstrual history, imaging, symptom burden)
- No comparator group — deviation from population norms cannot be estimated

Conclusions

This updated analysis — now incorporating age data for 149 of 166 patients — substantially strengthens the hypothesis-generating framework of the Valeo PMOS observational dataset.
Key conclusions:

FT4
Age-independent temporal signal

Free-T
Declines with age

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Ethics & Disclosure

All data de-identified prior to analysis. No individual can be identified from reported statistics. Conflict of interest: analysis conducted internally using Valeo Health operational data — should be declared in any external submission.