(3), suggesting that environmental factors may play a role in this PCOS-specific IR. Yet, the molecular mechanisms regulating IR remain unclear (4). Previous work suggested that Transforming Growth Factor Beta (TGFβ) superfamily ligands may be involved in the metabolic morbidity associated with PCOS (5). In this study, we investigated the effects of TGFβ1 (1, 5, 10, 30ng/ml), and the Anti-Müllerian hormone (AMH; 5, 10, 30ng/ml), a novel TGFβ superfamily ligand elevated in women with PCOS, as causal factors of IR in cultured myotubes from women with PCOS (n=10) and healthy controls (n=10). AMH negatively affected glucose uptake and insulin signalling increasing p-IRS1 (ser312) in a dose-dependent manner in myotubes from both women with and without PCOS. AMH did not appear to activate the canonical TGFβ/RBM signalling pathway. Conversely, TGFβ1 had an opposite effect in both PCOS and control myotubes cultures, decreasing phosphorylation of IRS1 (ser312) and enhancing glucose uptake via Smad2/3 signalling. In conclusion, these results suggest that AMH may play a role in skeletal muscle IR observed in PCOS, however, further research is required to elucidate its mechanisms of action and broader impact in this syndrome. References: (1) Stepto et al. Hum Reprod 2013 Mar;28(3):777-784. (2) Cassar et al. Hum Reprod 2016 Nov;31(11):2619-2631. (3) Corbould et al., Am J Physiol-Endoc 2005 May;88(5):E1047-54. (4) Stepto et al. J Clin Endocrinol Metab, 2019 Nov 1;104(11):5372-5381. (5) Raja-Khan et al. Reprod Sci 2014 Jan;21(1):20-31.

Cardiovascular Endocrinology
FROM BEDSIDE TO BENCH AND BACK AGAIN: LIPID METABOLISM & VASCULAR DISEASE

Retinol Binding Protein 4 Predicts Functional Vascular Disease in Early Postmenopausal Women
ELENI ARMEMI, MD, PhD,1 Meletios P. Nigdelis, MD,2, Areti Augoulea, MD, PhD,3 Asimina Chondrou, MD,4 Dimitrios Rizos, PhD,4 George Kaparos, PhD,4 Andreas Alexandrou, MD, PhD,4 Dimitrios G. Goulis, MD,PhD,5 Georgios Georgiopoulos, MD, PhD,5 Kimon Stamatakopoulos, MD, PhD,6 Irene Lambrinoudaki, both women with and without PCOS. AMH did not appear to activate the canonical TGFβ/RBM signalling pathway. Conversely, TGFβ1 had an opposite effect in both PCOS and control myotubes cultures, decreasing phosphorylation of IRS1 (ser312) and enhancing glucose uptake via Smad2/3 signalling. In conclusion, these results suggest that AMH may play a role in skeletal muscle IR observed in PCOS, however, further research is required to elucidate its mechanisms of action and broader impact in this syndrome. References: (1) Stepto et al. Hum Reprod 2013 Mar;28(3):777-784. (2) Cassar et al. Hum Reprod 2016 Nov;31(11):2619-2631. (3) Corbould et al., Am J Physiol-Endoc 2005 May;88(5):E1047-54. (4) Stepto et al. J Clin Endocrinol Metab, 2019 Nov 1;104(11):5372-5381. (5) Raja-Khan et al. Reprod Sci 2014 Jan;21(1):20-31.

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Diabetes Mellitus and Glucose Metabolism
LIPIDS, OBESITY AND METABOLIC DISEASE

The Gut Microbiome Regulates Host Glucose Homeostasis via Peripheral Serotonin
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SAT-657
The gut microbiome is an established regulator of aspects of host metabolism, such as glucose handling. Despite the known impacts of the gut microbiota on host glucose homeostasis, the underlying mechanisms are unknown. The gut microbiome is also a potent mediator of gut-derived serotonin synthesis, and this peripheral source of serotonin

OR17-07
Introduction: The impact of gender on the development of cardiovascular disease has long been recognized. The potential effect of sex-specific cardiovascular risk factors on molecular mediators of oxidative stress has received limited attention and the results remain conflicting. Hypothesis: To assess the link between retinol binding protein 4 (RBP4) and menopause-specific cardiovascular risk factors, on indices of early subclinical atherosclerosis, in a sample of apparently healthy young, postmenopausal women.

Methods: This cross-sectional study included a total of 123 healthy postmenopausal women, recruited from a University Menopause Clinic. Participating women were, not on hormone therapy, antihypertensive or hypolipidemic treatment and had a menopausal age of up to 10 years. Fasting venous blood samples were obtained for hormonal and biochemical assessment, including levels of RBP4. Sonographical studies were performed on the same day and included carotid-femoral pulse wave velocity (PWV) and calculation of the carotid artery stiffness index (SI).

Major results: Univariate analysis showed that RBP4 values correlated positively with age, total cholesterol, triglycerides, LDL-cholesterol, testosterone-to-estrogen ratio; negatively with circulating estrogen and almost significantly with homocysteine levels. Levels of homocysteine were inversely associated with RBP4 (homeostasis: RBP4 <10.5ng/ml vs ≥10.5ng/ml: 11.2±2.81μmol/L vs 12.5±3.44μmol/L, p-value=0.049 ANCOVA, adjusted for age, BMI, HOMA-IR). Multivariate analysis showed that PWV values were predicted by RBP4 (b-coefficient=-0.435, p-value=0.006), age, pulse pressure, homocysteine. S.I. beta was predicted independently by RBP4 levels (b-coefficient=0.324, p-value=0.039). Both models were adjusted for menopausal age, LDL-cholesterol, FEI, smoking, HOMA-IR.

Conclusion: RBP4 levels are linked with measures of local carotid and aortic arterial stiffness, in this sample of healthy postmenopausal women. This association seems to be mediated by higher levels of homocysteine, which may interfere with retinoic acid synthesis. Larger studies are required to further elucidate the significance of our findings.

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3. Limpach A, Dalton M, Miles R, Gadson P: Homocysteine inhibits retinoic acid synthesis: a mechanism for homocysteine-induced congenital defects. Exp Cell Res 2000;260:166-174.
is itself a regulator of glucose homeostasis. Here, we determined whether the gut microbiome influences glucose homeostasis through effects on gut-derived serotonin. Using both pharmacological inhibition and genetic deletion of gut-derived serotonin synthesis, we find [1] that the improvements in host glucose handling caused by antibiotic-induced changes in microbiota composition are dependent on the synthesis of peripheral serotonin.

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Pediatric Endocrinology
PEDIATRIC SEXUAL DIFFERENTIATION, PUBERTY, AND BONE BIOLOGY
A Quantitative-PCR Based Rapid and Cost-Effective Diagnostic Method for Turner Syndrome and Its Variants
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SUN-084
Turner’s syndrome (TS) is a common aneuploidy diagnosed by peripheral blood karyotyping in patients. Karyotyping involves manual labour, time and costs. Quantitative real-time PCR (qPCR) is a rapid molecular diagnostic test for TS that yields results of up to 48 samples within two hours at low cost with reasonable accuracy. To assess the sensitivity and specificity of qPCR in the rapid diagnosis of TS, genomic DNA was isolated and estimated from peripheral blood from 50 TS patients(45,XO-23, XO/XX mosaics - 10, Isochromosome Xq-12, XO/XY mosaics - 5), 25 normal females(46,XX) and 5 normal males(46,XY). qPCR was done using 96 well plates, fast real-time PCR. 4 primers were used - two on Xp [SHOX and ARSE] and two on Xq (VAMP7 and XIST). Autosomal gene HBB was used as housekeeping gene. The ΔΔ CT method was used for calculation of the ‘X gene dose’ with respect to the housekeeping gene and X genes from normal females. Differences of doses of the four X-chromosomal primers in different karyotypes were analysed. ROC curves were plotted to determine cut-offs to discriminate the different karyotypes of TS from normal females. qPCR could distinguish classical TS from normal females with >95% sensitivity and specificity. SHOX gene primer was the best to diagnose TS of all karyotypes combined and also classical TS(XO) from normal females. qPCR could also identify non-classical TS. The cut-offs determined from our study corroborates with past similar studies.1,2 qPCR using an appropriate panel of primers on the short and long arms of X chromosomes can be a rapid and cheaper alternative to karyotyping to diagnose TS of different karyotypes. The choice of primers should be guided by the need for a more sensitive or specific test depending on the clinical scenario. If used as a neonatal screening test, SHOX should be the best primer. For diagnostic purposes, when the pre-test probability is low, a

Neuroendocrinology and Pituitary
CASE REPORTS IN UNUSUAL PATHOLOGIES IN THE PITUITARY
A Case of Ectopic Neurohypophysis
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SUN-287
A CASE OF ECTOPIC NEUROHYPOPHYSIS
Pituitary stalk interruption syndrome (PSIS) is a congenital disorder of the pituitary gland. Symptoms at presentation may vary widely as this disease presents along a spectrum which includes; ectopic posterior pituitary, interrupted pituitary stalk or aplasia and hypoplasia of the pituitary gland. It is a heterogeneous disorder in terms of its radiologic and clinical presentation. It can present clinically as an isolated pituitary hormone deficiency (most common being growth hormone deficiency) or as multihormonal deficiencies.

CASE PRESENTATION
Patient is a 34-year-old woman with history of primary amenorrhea who was evaluated by a gynecologist and was prescribed oral contraceptive pills which lead to her having a menstrual bleed for the first time in her life. She denied any difficulty with smell. She had undergone normal psychomotor milestones and highest level of education was high school. She had normal puberty with normal pubic and axillary hair growth, normal breast development but no menarche. Of note, patient has a short stature, height is 4 feet and 11 inches, and her biological parents are of normal adult height
On evaluation, patient had normal am cortisol, prolactin and thyroid function tests. IGF-1 was significantly low for her age, FSH and LH were inappropriately low for her low estradiol level suggesting hypogonadotropic hypogonadism. Patient subsequently had an MRI of the pituitary and DEXA scan. MRI findings were suggestive of ectopic neurohypophysis. DEXA scan showed significant reduction in bone mineral density for age.