1. Although there are retrospective basal temperature data, we lack menstrual cycle and ovulation information during the pandemic. This analysis compared two independent, similar-design, convenience/community-based, single-cycle cohort studies: Menstruation Ovulation Study (MOS, n = 301, 2007-8) and MOS2 (n = 112) during the pandemic. With MOS as a control, and given evidence subclinical ovulatory disturbances (SOD; short luteal phase/anovulatory menstrual cycles of normal lengths) occur before changed cycle lengths, we hypothesized MOS2 would have increased SOD but similar cycle lengths as MOS.

Methods: In both studies, recruitment of menstruating women ages 19-35 years, not using systemic or combined hormonal contraceptives (CHC) used posters/eblasts/social media. In MOS, ovulation was assessed by 3-fold increased follicular (FP)-to-luteal (LP/premenstrual) urinary progesterone (PdG); in MOS2, by validated Quantitative Basal Temperature© (QBT) normal LP = 10+ days). We performed the same interviewer-administered (CaMos©) questionnaire for demographics, SES, and reproduction, measured anthropomorphic variables, plus collected daily Menstrual Cycle Diary© (Diary) for all. FP and LP/premenstrual PdG or salivary progesterone (Ps) samples were respectively collected. Participants in MOS2 were not different from MOS in: average age 29, menarche age 12.5, BMI 24, living situation and education (≥75% university graduates). Cohorts also differed: MOS2 women were less likely to be White (56% vs 76%), work fulltime, ever use CHC (68% vs 79%) or to be parous (8% vs 20%); they were younger at starting CHC (17.9 vs 18.6 years).

Results: MOS2 and MOS had similar cycle (30.3 vs 29.9 days, P = .306) and flow lengths (median 6.0 days; P = .055). MOS2 recorded significantly more SOD cycles (>50% anovulatory) vs MOS 3 (63% vs 10%; P < .001). MOS2 Diary analyses by Principal Components Analysis showed significantly increased anxiety/depression/frustration (negative moods) and “outside stresses” plus sleep problems and headaches vs MOS (all P < .001).

Discussion: This is the first evidence that ovulatory disturbances without cycle length changes may be associated with the multidimensional stresses women experience during the pandemic. Increased SOD may also relate to greater nulliparity, younger CHC teen use and more non-White women in MOS2, as well higher prevalence of negative moods, outside stresses and sleep problems. Salivary progesterone, cortisol and estradiol levels remain pending. Prevalent SOD cycles, if persistent/recurrent, risk increased infertility, bone loss, early heart attacks, and breast and endometrial cancers.

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Bilateral and Unilateral Malformations of Male Reproductive Tracts in Mice with Androgen Receptor Gene Deletion in the Mesenchyme
Shuai Jia, Fei Zhao, and Jillian Wilbourne

Congenital unilateral defects of male reproductive tracts have been documented in men, suggesting the potential left-right asymmetry in male reproductive tract development. However, classic textbooks of endocrinology have been teaching us that the development of the paired male...
The reproductive tract is governed by the universal action of the androgen receptor (Ar). During sexual differentiation, testis-derived androgens activate the Ar to promote the stabilization of the bilateral Wolffian ducts, the progenitor for the male reproductive tract. The Wolffian ducts then differentiate into the paired male reproductive tract organs, which include the epididymes, vas deferentes, and seminal vesicles. The Ar is expressed both in the epithelium and mesenchyme of the male reproductive tract during fetal development. Landmark tissue recombinant studies and the observation of normal morphology in the epithelium-specific Ar knockout mouse all support the notion that Ar action in the mesenchyme dictates the morphogenesis of the male reproductive tract. However, no genetic study has been performed to investigate the consequence of the loss of mesenchymal Ar on male reproductive tract development. To test the functional significance of the mesenchymal Ar, we designed a mesenchyme-specific Ar knockout mouse model (ARcKO) to ablate Ar specifically in the mesenchyme prior to the initiation of sexual differentiation. We performed immunohistochemistry of AR to confirm that Ar expression was absent in the mesenchyme while epithelial Ar remained intact in this ARcKO model. Based on the widely accepted notion that mesenchymal Ar regulates epithelial morphogenesis of the male reproductive tract, we expected to see abnormal patterning of the male reproductive tract. Indeed, the epididymis lost its characteristic coiling and became cystic. However, 40% of the 23 collected ARcKO males displayed these abnormalities on both horns; 43% and 17% displayed these abnormal phenotype solely on the left and right horn, respectively. This surprising observation might result from asynchronous activities of Cre on the left and right horns. However, when the Cre was crossed with a reporter (tdTomato), we observed comparable reporter expression on both horns at the onset of sexual differentiation of reproductive tracts. These observations suggest that the developmental programs in the morphogenesis of the left and right male reproductive tracts are asymmetrical and there could be a compensation mechanism for the loss of mesenchymal AR. We are currently in the process of comparing the left and the right horns in Ar knockout male mice to determine any biased signaling. Taken together, our study provides a unique model for not only studying congenital defects of male reproductive tracts but also for investigating the potential asymmetry in the masculinization program.

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