Results: Stress moderated the effect of age on the basal cytokine composite, such that age was associated with increased inflammation only among those with higher stress [$B = 0.02$, $p < 0.01$]. No significant interaction effects were found in models predicting CRP or stimulated cytokines.

Conclusion: Results support the hypothesis that stress exacerbates the negative effect of aging on basal cytokine levels. Stress did not moderate the association of age with CRP or stimulated cytokines.

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Symposium 5: Stress Biology in Pregnancy and Early Development

Chair: Karin de Punder

Presentation 1: Maternal behavioral health during pregnancy and newborn telomere length

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Background: The importance of health behaviors for age-related disease risk is well established. The period of embryonic and fetal life represents among the most sensitive developmental windows at which time the effects of maternal behavioral factors can impact the initial (newborn) setting of telomere length (TL). In the present study, we aimed to investigate the aggregate effect of maternal diet, sleep and physical activity during pregnancy on newborn TL.

Methods: Diet quality (Mediterranean Diet Score), sleep quality (Pittsburgh Sleep Quality Index) and physical activity (Measurement of Physical Activity and Sport Questionnaire) were assessed in a sample of N = 125 healthy pregnant women enrolled in a longitudinal prospective cohort study. A Behavioral Health Sum Score was computed as the sum of z-scores. Newborn TL was measured in cord blood mononuclear cells (CBMCs) collected at birth using a quantitative real-time polymerase chain reaction method.

Results: Regression analysis showed that healthier maternal behavior (i.e. higher Behavioral Health Sum Score) during pregnancy was associated with longer CBMC TL ($B = 0.03$, 95%CI: 0.006 to 0.05, $p = .01$), also after adjusting for maternal age, SES, pre-pregnancy BMI, child sex and gestational age at birth.

Conclusion: Our results emphasize the importance of promoting maternal behavioral health during pregnancy.

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Presentation 2: Estrogen receptor genes and the longitudinal assessment of perinatal anxiety symptoms

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Single nucleotide polymorphisms (SNP) in estrogen receptor genes may lead to inter-individual differences in the sensitivity of the estrogen receptors (ER) alpha, beta and G-protein coupled estrogen receptor (GPER), ultimately modulating the susceptibility of women to mood symptoms during periods of estrogen fluctuation. This study examines the association between SNPs in estrogen receptor genes and anxiety in 159 women from 34-36 weeks of gestation up until to 8-12 weeks postpartum. Anxiety was assessed at five time-points during peripartum by using a standardized anxiety inventory. Five SNPs were genotyped from dried blood spots: rs2234693 and rs9340799 (ER-alpha), rs1256049 and rs4986938 (ER-beta), and rs3808350 (GPER). Linear mixed models revealed that rs2234693 and rs9340799 encoding for ER-alpha were significantly associated with anxiety scores ($p = 0.01$ and $p = 0.001$, respectively, F-test). This work highlights that SNPs encoding for ER-alpha may contribute to the genetic susceptibility to maternal perinatal anxiety. For emotional processes, SNP-sensitivity differences in ER-alpha seem to play a more important role than ER-beta and GPER. This may be explained by its more dominant expression in the hypothalamus and amygdala.

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Presentation 3: The moderating role of the child's anxiety vulnerability on parent-child cortisol patterns

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Background: Altered cortisol levels are an important biological correlate of childhood anxiety disorders and can be modulated by family environment. Parent-related factors moderate the link between parent and child cortisol levels. It remains unknown if child-related variables also play a moderating role. In children, sensitivity to anxiety, intolerance of uncertainty, and repetitive thoughts are associated with a vulnerability to developing anxiety. This study examined if a child's anxiety vulnerability moderated the link between cortisol levels in parent-child dyads.

Methods: Through hair samples, we quantified hair cortisol concentrations (HCC) in 50 parent-child dyads. A composite score for child and parent anxiety vulnerability was created (Anxiety Sensitivity Index, Intolerance of Uncertainty Scale, and the Perseverative Thinking Questionnaire). A moderation analysis was performed using parent and child HCC, with the child's anxiety vulnerability score as a moderator (controlling for the parent's anxiety vulnerability).

Results: With high child anxiety vulnerability, child and parent HCC was positively associated ($p = 0.03$).

Conclusion: It is important to consider child-related characteristics when investigating dyadic effects.

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