**Single nucleotide polymorphism in the promoter of the steroidogenic factor 1 (SF-1) gene after gestational exposure to Bisphenol A.**

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Polycystic ovary syndrome (PCOS) is a major cause of dysovulation and consequent subfertility in women. Cross-sectional data reports that levels of BPA, a widespread endocrine disrupting chemical (EDC), are higher in women with PCOS than in reproductively healthy women. PCOS may have a prenatal origin supporting the concept of fetal origin of adult disease.

We have previously shown that prenatal exposure to a high dose of BPA was associated with hypermethylation of the SF-1 promoter in the female rat placenta using methylation microarray. SF-1 plays a key role in the development and function of the ovaries.

We aimed at validating SF-1 promoter hypermethylation in female placenta on a large number of samples and at correlating those epigenetic changes with changes in SF-1 expression.

Pregnant rats were exposed orally to BPA (10mg/kg/d) from gestational day 6 (GD 6) to 18. Placentas obtained by cesarean section were sexed using classical PCR for SRY expression. DNA methylation of the SF-1 promoter was studied in female placenta using Methylation-Specific PCR after bisulfite treatment. Changes in expression of SF-1 mRNA were examined by RT-PCR.

So far, we have not identified any region with differential methylation of the SF-1 promoter in female placenta exposed to BPA compared to control. However, the sequencing of this region highlights a single nucleotide polymorphism (SNP) at CpG 22 in the SF-1 promoter (substitution of a cytosine to adenosine). There was no effect of exposure to BPA on SF-1 mRNA levels in female placenta.

In conclusion, prenatal exposure to a high dose of BPA leads to a SNP of the SF-1 promoter in female placenta. We hypothesize that such a SNP, if existing in the ovaries could mediate the effects of BPA exposure on reproduction. Moreover, our results suggest that placenta could provide early markers of effects of exposure to BPA and thereby should be investigated in humans as a biomarker of exposure to EDC.