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We read with great interest article titled "Do Maternal Micronutrient Deficiencies Program the Body Composition and Behavior of the Offspring? Probable Underlying Mechanism."<sup>1</sup> We wish to congratulate the authors for the extensive review. While authors have focused on selective micronutrients indicating that prenatal micronutrients restriction leads to altered body composition, epigenetic phenomenon, change in lipid/fat metabolism, and anxiety like behavior in mother and offspring in rodent model. However, some additional information may also be highlighted in their review.

Micronutrient access can also be associated with adverse effects on developing fetus. Rahimi et al found that though moderate dose folic acid supplementation reduced DNA methylation variance at certain imprinted genes in embryonic and placental tissues, high dose supplementation exacerbated the negative effects of assisted reproductive technology (ART) at imprinted loci. A high dose of folic acid during pregnancy in mice showed deleterious outcomes such as DNA hypomethylation, epigenetic abnormalities, and delay in embryonic development.<sup>2</sup>

Further, micronutrient selenium (Se) plays a critical role in fetal development that needs to be highlighted. The researchers, using C57BL/6 mice model, demonstrated that maternal Se deficiency leads to reduced fetal weight and increased plasma tetraiodothyronine and triiodothyronine concentrations. This was associated with reduced expression of seleno-dependent deiodinases, DIO2, and DIO3. Se deficiency reduced fetal glucose concentrations, leading to reduced fetal weight<sup>3</sup> evidence supported by another study on human conception and pregnancy.<sup>4</sup> Current research, however, does not recommend use of Se during pregnancy due to insufficient evidence, lack of measurable markers to assess the effect of Se supplementation on the human metabolism, and Se's narrow therapeutic index.<sup>5</sup>

Conflict of Interest None declared.

## References

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