Commentary: ‘Unhealthy diet,’ nutrient status, and ADHD symptoms: a confounding role for environmental nitrous oxide exposure – reflections on Rijlaarsdam et al. (2016)

Keith Fluegge

Journal of Child Psychology and Psychiatry
DOI: 10.1111/jcpp.12649

Abstract

Rijlaarsdam et al. (2016) recently published their findings utilizing a longitudinal design showing that prenatal ‘unhealthy diet’ was positively associated with IGF2 DNA methylation at birth across both youth cohorts. However, only in the EOP youth was prenatal ‘unhealthy diet’ positively associated with ADHD symptoms presumably through IGF2 DNA hypermethylation. Rijlaarsdam et al.’s (2016) choice to assess high fat and sugar diet with the Food Frequency Questionnaire (FFQ) may offer some indication as to prenatal nutrient status, as the foods identified by the FFQ in their study are relatively low in free choline. It has been shown that gestational choline deficiency in rats leads to hypermethylation of IGF2. Consistent with the literature describing an association between air pollution and cognitive neurodevelopmental impairment, the author of this commentary has previously proposed through empirical investigation that chronic environmental exposure to the trace levels of the pervasive air pollutant, nitrous oxide (N2O), may facilitate core features of neurodevelopmental disorders, like ADHD. Impaired acetylcholine synthesis in rats exposed to N2O has been shown, with a 53% reduction in [1-2H2,2-2H2] choline. Low-dose N2O exposure is also thought to stimulate central release of opioid peptides, like dynorphin, which play a role in significantly increasing food intake behavior and/or modulating sucrose intake. Taken altogether, these studies present a strong confounder to the interpretation made by Rijlaarsdam et al. (2016) that prenatal ‘unhealthy diet’ may play a role in the onset of ADHD symptoms in youth with EOP conduct problems through induction of IGF2 DNA hypermethylation. While the ‘unhealthy diet’ may represent possible maternal nutrient deficiencies during gestation, it is also possible that exposure to air pollutants, particularly N2O, may not only directly reduce fetal cholinergic status thereby enhancing IGF2 DNA hypermethylation but may also significantly modulate maternal food intake behaviors (i.e. sucrose).