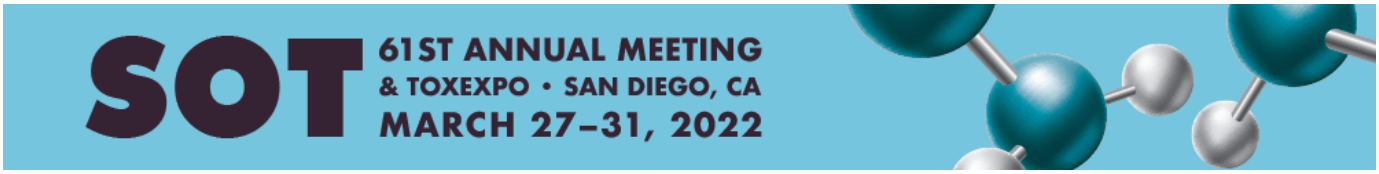
Mice exposed to food-grade TiO 2 from in utero life to adulthood show sex-specific gut microbiota and metabolic disorders

Lauris Evariste, Eric Gaultier, Christel Cartier, Johanna Noireaux, Benoit Chassaing, Eric Houdeau, Bruno Lamas



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Mice exposed to food-grade TiO_2 from *in utero* life to adulthood show sex-specific gut microbiota and metabolic disorders

Author Block: L. Evariste[1], E. Gaultier[1], C. Cartier[1], J. Noireaux[2], B. Chassaing[3], E. Houdeau[1], and B. Lamas[1]. [1]Toxalim (Research Centre in Food Toxicology), Team Enterisk, INRAE, Toulouse, France; [2]Department of biomedical and inorganic chemistry, LNE, Paris, France; and [3]Inserm U1016, Cnrs UMR 8104, Paris University, Paris, France. Sponsor: *D. Zalko*

Abstract:

The use of titanium dioxide (TiO_2) particles as a food additive (E171 in EU) in ultra-processed foods raises public health issues. Our previous study showed transplacental passage of TiO_2 nanoparticles in the human placenta, and presence of TiO_2 particles in meconium, demonstrating foetal exposure. Due to biocidal properties of TiO_2, whether E171 exposure starting early during pregnancy may alter the establishment of gut microbiota homeostasis with potential deleterious effects at adulthood has not been explored. The current study in mice aimed to assess the perinatal fate of E171 given to dams and the consequences on gut microbiota and metabolic functions of the offspring. Female mice were exposed to a control or E171-enriched diet at a human relevant level (10 mg/kg bw/day) during pregnancy and lactation until weaning of pups, then the descendants were fed with the same diet as their mother until postnatal day (PND) 150. Oral glucose tolerance and fasting insulin were assessed at PND143. At PND150, all mice were sacrificed and gut microbiota composition as well as intestinal pro- and anti-inflammatory cytokine production were measured by 16S gene sequencing and ELISA, respectively. Biodistribution of TiO_2 particles was also studied by ICP-MS in fetus at day 18 of pregnancy, and in the liver at PND150. In E171-exposed mice, higher Ti level was first detected in fetus, and Ti accumulation was reported in liver at PND150 compared to controls regardless of the sex. Changes in gut microbiota composition occurred in E171-exposed male only, showing increased β-diversity and of the Firmicutes/Bacteroidetes ratio. Increased production of the pro-inflammatory cytokine IL1β in the colon as well as glucose intolerance and higher fasting insulin levels were also reported in E171-exposed male relative to controls. In contrast, decreased secretion of pro- (IL1β, TNFα, IFNγ, IL17) and anti-(IL10) inflammatory cytokines occurred in the colon of E171-exposed females, without other changes. These results showed that long term exposure to E171 from *in utero* life alters intestinal and metabolic homeostasis in a sex-dependent manner, characterized by altered glucose metabolism and gut dysbiosis in male mice only.

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Sponsor Affiliation : National Research Institute for Agriculture, Food and Environment (INRAe)
Sponsor Email : daniel.zalko@inrae.fr

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Society of Toxicology
11190 Sunrise Valley Drive, Suite 300
Reston, VA 20191
703-438-3115 Office
sothq@toxicology.org
http://www.toxicology.org/
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