



Chronic exposure to the food additive silicon dioxide (E551) at a human-relevant dose blocks induction of oral tolerance to dietary antigens

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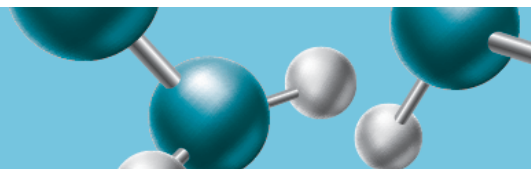
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Chronic exposure to the food additive silicon dioxide (E551) at a human-relevant dose blocks induction of oral tolerance to dietary antigens

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Abstract:

Food-grade SiO₂ (E551 in EU), composed of aggregated nanoparticles (NPs), is used as an anticaking and antifoaming agent in powdered foods, with chronic dietary exposure in humans (0.8-74 mg/kg/day). Because SiO₂-NP models block induction of oral tolerance (OT, an immune mechanism for food antigen acceptance), the current study in mice aimed at evaluating whether chronic exposure to E551 at a human-relevant dose, added to a solid food matrix or in liquid suspension, alters the establishment of OT towards a food antigen model.

Mice were daily treated for 60 days without (controls) or with E551 (10mg/kg BW/day) in water suspension (gastric gavage) or incorporated into food pellets (solid matrix). Food intake was controlled. At day 41, mice were orally exposed to the dietary antigen ovalbumin (OVA) (20mg/mouse; OVA-tolerized mice) or PBS vehicle for 3 days. All mice were subsequently immunized by subcutaneous injection of OVA (100µg/mouse) on day 48. Blood was collected 1 week after for anti-OVA IgG serum titers to evaluate OT induction in OVA-tolerized mice exposed or not to E551. In all groups, to further assess OT to food antigens, mice were *de novo* challenged by oral OVA (25µg/mouse) for 5 days before sacrifice. Isolated immune cells from mesenteric lymph nodes (MLN) were activated by PMA/ionomycin to assess pro- (IFN γ) and anti-inflammatory (IL-10, TGF β) cytokine secretion measured by ELISA. Fecal lipocalin (Lcn)-2 level was used as a global marker of gut inflammation.

In control mice, OVA tolerance protocol (oral OVA) lowered by 87% circulating anti-OVA IgG levels ($p < 0.0001$) compared to oral PBS group, showing normal OT induction to OVA. In contrast, anti-OVA IgG titers did not decrease in OVA-tolerized mice chronically exposed to E551 regardless of the vehicle, showing blockade of OT to food antigens. In OVA-tolerized mice exposed to E551 through food pellets, *de novo* oral challenge with OVA increased ($p < 0.05$) fecal Lcn-2 levels (+131%) and IFN γ (+139%) production by MLN cells, together with a drop ($p < 0.05$) of TGF β (-46%) and IL10 (-46%) compared to OVA-tolerized controls, demonstrating gut inflammation. These results showed that chronic E551 exposure at a human dietary level in solid or liquid matrix impairs OT to dietary antigens, and promotes intestinal inflammation supporting food intolerance.

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