Commensal microbiota and food antigens are important factors in the regulation of IgE production in mice

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IgE is one of key modulators in the pathogenesis of allergy by inducing FcR-mediated activation of mast cells and basophils. Production of IgE is regulated by commensal microbiota, as it is normally undetectable in conventional mice, but highly elevated in germ-free mice shortly after weaning into solid food. The mechanism behind IgE production in germ-free mice is poorly understood, but could be a consequence of dysregulated immune responses to Ags in the food. To address this idea, germ-free mouse pups were weaned into an antigen-free elemental diet comprised of essential amino acids, vitamins and minerals. Strikingly, neither these mice nor their offsprings, designated as antigen-free mice, possessed detectable levels of serum IgE. Moreover, feeding adult germ-free mice with antigen-free diet reduced the serum IgE level, indicating continued presence of food is required to sustain high amount of IgE production. As expected, antigen-free mice weaned into normal solid food diet produced high levels of IgE. To determine the age-dependency of commensal microbiota in IgE production, germ-free mice were conventionalized at various ages, starting from 4 wks. Colonization of commensal microbiota at 4 wks of age prevented high levels of serum IgE, but not in older mice. These results suggest that food Ags by default induce a strong IgE response that is suppressed by the commensal microbiota.