

## Mini Review

# New Insights on the Relation Between Human Microbiota and Food Allergy

**Davide Frumento\***

Department of Health Sciences, DISSAL, University of Genoa, Genoa, Italy

\***Corresponding author:** Davide Frumento, Department of Health Sciences, DISSAL, University of Genoa, Genoa, Italy. Tel: +393334310322; Email: [davide.frumento@edu.unige.it](mailto:davide.frumento@edu.unige.it)

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### Abstract

Food allergy onset occurs in early life, in fact an Australian non-selected 1-year-old children cohort showed a prevalence of sesame, peanut and egg allergy of 8.9, 3.0 and 0.8 % respectively. Other experiments showed that clinical reactivity to such foods arises around 4 months of age. Several researchers reported dysbiosis in food allergic subjects. Some studies about human microbiome genetics evidenced altered gut microbiota in individuals suffering from both cow's milk and egg allergies. It is generally accepted that dysbiosis arises prodromal to the food allergy onset. In fact, after analyzing gut microbiota in children, belonging to a Canadian cohort, within 12 months of age, it was found proof of food sensitization after 12 months of age. The aim of this opinion paper is to enforce existing evidences and, above all, to introduce the concept that, in many cases, human features and perceptions are mostly shaped by microbiota, as outlined in my previous papers.

### Introduction

Food allergy is a non-physiological reaction to food which is usually mediated by immune processes. Among them, IgE-mediated ones are increasing in prevalence (5% of adults and 8% of children) [1-3], but the underlying mechanisms are still matter of investigation; food allergy is partially genetically-based and partly depending on environmental factors that affect the quality of the intestinal microbiota. Food allergy is generated by degranulation of both basophils and mast cells via hapten cross-linking of IgE bound to the cell surface. If gut microbiota is not intact, IgE levels are increased and basophils are mobilized; as an outcome, allergy-related symptoms occur [4].

Food allergy onset occurs in early life, in fact an Australian non-selected 1-year-old children cohort showed a prevalence of sesame, peanut and egg allergy of 8.9, 3.0 and 0.8 % respectively [5]. Besides, in the USA, prevalence ranges from 1.5% to 10%. Interestingly, a recent epidemiological population-based research reported that, from 2007 to 2010, prevalence of parents-reported food allergy in infants is 6.53%. The most commonly reported pediatric allergies were to milk, peanut and shellfish. Another USA study showed a lightly higher value of food allergy prevalence in children (8%) [1]. Other experiments showed that clinical reactivity to such foods arises around 4 months of age [6,7]. These findings suggest that environmental risk factors could take

part to this phenomenon during the postnatal period or even in utero. Such factors comprehend breastfeeding, birth mode, human contact and exposure to any kind of pet [8]. This becomes even more interesting considering that, although gastrointestinal tract has generally been accounted as sterile until environmental bacteria colonization takes place at birth, recent researches showed evidences of microorganisms presence in amniotic fluid, fetal membranes, umbilical cords, placentas and meconium [9]. The above stated considerations corroborate the Hygiene Hypothesis, which states that an inappropriate and insufficient microbial exposure in early life causes allergies. It was demonstrated that each one of these parameters individually affects gut microbiota composition [10-14]. The aim of this opinion paper is to enforce existing evidences and, above all, to introduce the concept that, in many cases, human features and perceptions are mostly shaped by microbiota, as outlined in my previous papers [15-17], in which was reported that microbiota composition markedly influences both taste sense, HCV infection process and liver regeneration.

### Evidences in Humans

Several researchers reported dysbiosis in food allergic subjects. Some studies about human microbiome genetics evidenced altered gut microbiota in individuals suffering from both cow's milk and egg allergies [18-20]. Moreover, it was demonstrated that Scandinavian children with egg or cow's milk allergy hosted

higher *Staphylococcus aureus* and Coliforms loads with the respect to the non-allergic ones; these findings were coupled with the lower Bifidobacteria/Lactobacilli ratio if compared to non-atopic kids [18]. Furthermore, investigators highlighted markedly higher *Atopobium* group\Clostridium *coccoides* cluster if compared to a control group [20].

## Dysbiosis in Humans

It is generally accepted that dysbiosis arises prodromal to the food allergy onset. In fact, after analyzing gut microbiota in children, belonging to a Canadian cohort, within 12 months of age, it was found proof of food sensitization after 12 months of age. Moreover, every quartile rise in bacterial richness at 3 months of age was linked with a 55% lowering in probability for food sensitization by 12 months. Food sensitive children, at the genus level, had an excess of gut Enterobacteriaceae, while Bacteroidaceae were underrepresented [21]; with this in mind, it can be said that early infancy could represent the crucial period for the intervention, corroborating the idea that gut microbiome and food allergy are related, in an age-based manner. Furthermore, it was found that milk allergic children's gut microbiota composition up to 6 months from birth was linked with subsequent clearance of such allergy by age 8 years. This resolution was imputed to an enrichment of both Firmicutes and *Clostridia* genera [21]. Analogue results were obtained in atopic dermatitis patients, in which microbial diversity was not associated with allergic phenomenon [23]. Egg allergy patients were found to have an excess of Streptococcaceae, Leuconostocaceae and Lachnospiraceae within the gut when compared to controls [24]. According to [25] there is no variation in microbial diversity, but allergic children were found to have higher levels of *Clostridia* and *Anaerobacter* genera and decreased ones of *Bacteroides* and *Clostridium XVIII* in infants with the respect to healthy individuals.

Murine models' results are in nice agreement with age-sensitive relationship between gut microbiota and the host. Microbiota introduction germ-free mice dramatically lowered IgE levels and inhibited the arise of food allergy [26]. Due to close dependence between diet and microbiota composition [26], as well as the different clinical outcomes related to allergen types, comparison of bacterial genus combinations between allergens need more attention.

## Conclusion

After the evaluation of the above findings and coupling them with the evidences collected in [15-17], it can be safely said that microbiota plays a pivotal role in several essential pathophysiological processes within human organisms. All evidences converge to the demonstration that bacterial diversity is essential for a physiological and healthy functioning of immune system, taste sense, as well as liver regeneration. Considering that

microbiota formation take place during both pre- and post-birth periods, it is safe to assume that also food allergy etiology has to be chronologically placed in correspondence of such time zones. So, if an efficient prevention therapy will be developed, it should be focused not only on the child, but even on the giving-birth parent. It is thus clear that microbiota contribute both to shape our perception of reality and to defend ourselves from pathogens. Since some evidences similar to the microbiota contribution to taste sensing [27] are standing about olfactory sense [28], it can be hypothesized that the human bacterial set is a necessary component of human subjectivity.

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