

Food allergy and intolerance

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Abstract: Allergic reactions to foods represent severe actual problems for mankind having increased global character. Adverse food reactions are divided to food allergy, an immunological response to food, and food intolerance, a non-immune reaction with allergy-like symptoms. It is estimated that 6–8 % of children and 1–2 % of adults suffer from food allergy. The prevalence of food intolerance in adults is no more than 5–6 %, however in infants and young children, it is varied from 0.3 % to 20 %. Allergy is caused by different food allergens (milk proteins, cereals, nuts, fruits and vegetables), while there is frequent cross-allergy among them. Food intolerance is adverse reaction resulting from enzyme deficiencies, pharmacological reactions, and response to toxic or irritant components of food. Focusing on dairy products and cereals, the impact of fermentation in reducing food intolerance or allergenicity is reviewed in this paper.

Keywords: food allergy, food intolerance, allergens, lactic acid bacteria, fermentation

Introduction

The concept that certain foods can produce adverse reaction in sensitive individuals has been gradually revealed for a long period. Food allergy and food intolerance have been known since antiquity Hippocrates (460–370 BC) reported that cow's milk could cause gastric upset and urticaria. Galen (131–210 BC) described a case of intolerance to goat's milk (David, 2005; Kayserová, 2004). For instance, the observations on the cause of food allergens as fishes and eggs or clams in the development of asthma and dermatitis were expanded, respectively in the 17th and 18th centuries. Other food allergy milestones were carried out in 1656 when, P. Borel introduced a skin test with egg white in France and probably in 1919 when C. Richet summarized all up to date knowledge in his paper on Food Anaphylaxis (cit. Hefle, 2001). Demonstrations of food antigens that were rapidly absorbed from the gastrointestinal tract and transported to the various organs of the human body were provided by Prausnitz and Kustner in 1921. In 1959, Burnet proposed a theory describing the recognition of foreign antigens by lymphocytes and the subsequent induction of immune response (cit. Hayakawa et al., 1999). Further investigations of food allergy finished in 1972 and 1984 understood the intestinal and common mucosal immune systems by Tomasi and Bienenstock (cit. Hefle, 2001).

In the past, food allergy and intolerance were considered as minor health problems. Scientific research and interest are focused on food adverse reactions only in last decade of 20th century (Smith, 1997).

The terms food allergy and food intolerance are often mixed up, but there are some differences

between them (Mills and Breiteneder, 2005). Food allergy is only certain part of adverse food reactions. These reactions can be toxic or non-toxic. Bacterial toxins or high content of biogenic amines may activate a toxin reaction. Non-toxic reactions are caused by immune or non-immune mechanisms (food allergy or intolerance) (Ispano et al., 1998; Halken, 1997). Immunologic reactions to food are mediated by the immune system, while all other reactions fall into the non-immunologic category (Davis, 2009). Cereals and milk cause most food allergies and intolerances in our country.

The production of fermented foods is one of the oldest food processing technologies known to man. Lactic acid bacteria are used in food fermentation for longer preservation and improving textures, flavours and tastes. Moreover, proteolysis during fermentation can lead to reduction of immunoreactivity of food proteins. Hence, it could be expected that fermentative transformations with suitable lactic acid bacteria could produce hypoallergenic products (El-Ghaish et al., 2011).

Food allergy

Food allergy is defined as an adverse immunological (hypersensitivity) response to food (food proteins) and as such it is not a single disease, nor is it caused by one pathophysiologic disturbance (Sicherer, 2002; Sicherer and Sampson, 2006; Sicherer and Sampson, 2010; Macdougall and Etuwewe, 2005; Madsen, 1997; Halken, 1997; Crevel et al., 2007). This is manifested only in hypersensitive individuals (Rimárová, 2008), who have so-called predisposition (Hefle, 1996) and where symptoms appear rapidly following exposure to macromolecules (Mills and Breiteneder, 2005). Food allergic reaction can be

divided into IgE mediated and non IgE mediated (Ispano et al., 1998; Halken, 1997; Ortolani and Pastorello, 2006; Davis, 2009).

The number of patients suffering from food allergy has increased during recent decades, and allergic diseases have become a major clinical and public problem (Sicherer and Sampson, 2010; Macdougall and Etuwewe, 2005; Emmett, 1996).

There are several factors, which are responsible for development of food allergy: especially genetic allergy predisposition, early “foreign” food protein exposure (time, dose, and frequency), allergen uptake and handling (Halken, 1997).

The prevalence of specific food allergies is dependent on regional dietary habits and methods of food preparation (Sampson, 2004; Davis, 2009).

The risk of developing allergies is significantly influenced by genetic disposition. The changes in lifestyle and environmental factors result in increases of adverse food reactions. Complex factors include socio-economic impacts, impacts of external and internal environment, exposure to new allergens, stress, use of antibiotics, infectious diseases, climate change, and others (Rimárová, 2008; Samartín et al., 2001).

Food allergy affects 2–4 % of the population (Fuchs, 2008). The highest prevalence observed in children between 1.5 to 3 years (25 % of all food reactions). According to various studies suffer from food allergy 6–8 % of children and 1–2 % of adults (Altman and Chiaramonte, 1997). Only about 20 % of all food allergies persist into adulthood, others resolve spontaneously within three years of age (Svačina, 2008).

This is probably due to delayed onset of the mechanisms of oral tolerance to food proteins (Fuchs, 2008). Some studies indicated only 11 % resolved egg and 19 % resolved milk allergy by age 4 years (Sicherer and Sampson, 2010; Savage et al., 2007; Skripak et al., 2007).

Symptoms range from mild and tolerable (slight abdominal pain) to anaphylaxis which can result in physical collapse and death. The symptoms are divided into three main groups: immediate (within 1h of ingestion), delay or late (more than 1h after ingestion), and remote (Gray and Chan, 2003).

In Table 1, there is a summary of symptoms caused by food, which contains allergens. These symptoms involving the skin, gastrointestinal tract, respiratory tract, the motoric system, cardiovascular system, genitourinary tract and central nervous system, or can be shown by overall reaction (life threatening anaphylactic reaction – in rare instances).

Symptoms of food allergy vary depending on several factors, such as the age of the subject, the allergen involved and the amount of food eaten, physical exercise, stress, coexisting medical problems, among others (Samartín et al., 2001; Gray and Chan, 2003).

The food allergens can be defined as chemical, physical and biological substances occurring in environmental, which in sensitive individuals produce allergic reactions. The ability of allergen provoke this reaction depends on the type of allergen, the amount, duration of operation, points of entry and the degree of hypersensitivity of a particular organism (Rimárová, 2008). The allergic reaction can be caused by different amount of protein, from perhaps a tenth of a milligram up to grams, and sometimes tens of grams (Crevel et al., 2007). The major food allergens are water- or salt-soluble proteins or glycoproteins with molecular weights of 10–60 kD that are stable to heat, acid, and proteases (Sicherer and Sampson, 2006; Sicherer, 2002; Hayakawa et al., 1999; Smith, 1997; Sampson, 1999; Davis, 2009). Allergens, which are stable against denaturation and degradation during food processing, are mostly responsible for causing food allergies (Davis, 2009).

Tab. 1. The most common clinical manifestations of food allergies (Svačina, 2008; Sicherer and Sampson, 2006; Macdougall and Etuwewe, 2005; Fuchs, 2008; Halken, 1997; Ring et al., 2001; Carter, 2003; Muraro et al., 2014).

Location	Manifestations
Gastrointestinal	abdominal cramps, flatulence, blood in the stools, nausea, abdominal distension, colic, pain, vomiting, meteorism, diarrhea, constipation, malabsorption
Skin	atopic dermatitis, contact dermatitis, eczema, skin rashes, itching or flushing, tingling, swelling of the lips, palate, tongue or throat erythema, urticaria, angioedema,
Respiratory	recurrent wheezing, nasal congestion, itchiness or sneezing, asthma, laryngeal edema, stridor, cough, rhinoconjunctivitis cold, shortness of breath, dyspnea
mouth, neck, ears	stomatitis, otitis, pharyngitis
nervous system	irritability, restlessness, fatigue, migraine
blood count	anemia, eosinophilia, thrombocytopenia
other signs	enuresis, nephrotic syndrome, arthritis

More than 200 proteinaceous allergens have been identified and characterized, and over 100 different foods or food components may cause adverse reactions (Hayakawa et al., 1999; Astwood and Fuchs, 1996).

The eating habits and socio-cultural background are responsible for differences in foods most commonly involved in allergy. Variations of occurrence are between age groups as well as countries (Madsen, 1997; Ring et al., 2001).

The main allergens, which according to recommendation of European Union subject to mandatory marking on the food are:

- cereals containing gluten (i.e. wheat, rye, barley, oats, spelt, kamut or their hybridised strains) and products thereof,
- crustaceans and products thereof,
- eggs and products thereof,
- fish and products thereof,
- peanuts and products thereof,
- soybeans and products thereof,
- milk and products thereof (including lactose),
- nuts i. e. Almond (*Amygdalus communis* L.), Hazelnut (*Corylus avellana*), Walnut (*Juglans regia*), Cashew (*Anacardium occidentale*), Pecan nut (*Carya illinoensis* (Wangenh.) K. Koch), Brazil nut (*Bertholletia excelsa*), Pistachio nut (*Pistacia vera*), Macadamia nut and Queensland nut (*Macadamia ternifolia*) and products thereof,
- celery and products thereof,
- mustard and products thereof,
- sesame seeds and products thereof,
- sulphur dioxide and sulphites at concentrations of more than 10 mg/kg or 10 mg/litre expressed as SO₂ (DIRECTIVE 2003/89/EC).

These so-called main allergens are responsible for almost 90 % of allergic reactions and intolerance (Rimárová, 2008; Macdoughal and Etuwewe, 2005; Hayakawa et al., 1999). Reactions to fruits (apples, peaches, apricots, cherries, kiwi and citrus fruits) and vegetables (celery) are common (approximately 5 %) but usually not severe (Sicherer, 2002; Sicherer and Sampson, 2006; Svačina, 2008) and the allergens are often sensitive to cooking (Hayakawa et al., 1999). Also, some food additives – colours, preservatives, flavorings, colorings, antioxidants, can be implicated in food allergies and intolerances (Smith, 1997; Ring et al., 2001).

It is generally assumed that sensitization to the classical food allergens such as milk, egg, peanut and fish occurs via the gastrointestinal tract, although other types of food allergy occur as a consequence of prior sensitization to inhaled allergens such as pollen (Mills and Breiteneder, 2005; Sampson, 2004; Breiteneder and Ebner, 2000).

Cereals are the major plant food, which cause adverse food allergies. Cereals contain a range of

allergens (Mills and Breiteneder, 2005). IgE mediated reactions to wheat have been demonstrated as early as the beginning of the 20th century (Scibilia et al., 2006). The prevalence of cereal allergy has increased among the children as well as among the adults. More than 0.5 % of children and 3 % of adults suffer from an allergy to wheat (Battais et al., 2005; Zuidmeer et al., 2008). Some cereal allergens: water soluble albumins and globulins (lipid transfer proteins, inhibitors of trypsin and α -amylase), and water insoluble gliadins and glutenins, known as prolamins were described in scientific studies (Battais et al., 2005; James et al., 1997; Walsh et al., 1985).

Prolamins are responsible for food-dependent exercise-induced anaphylaxis and atopic dermatitis, inhibitors of proteases and α -amylases have also been described as both inhalant and food allergens (Mills and Breiteneder, 2005). Glycosylated subunits of tetrameric α -amylase inhibitors from wheat (CM16), and its homologs from barley (CMb), and rye *Sec c1* have the highest allergenic activity. Inhibitors of enzymes have been described as major allergens in rice and buckwheat (Mills and Breiteneder, 2005).

From the animal origin foods, milk is the food, which is often responsible for formation of allergies.

Cow's milk is one of the most common causes of adverse reactions in foods and it contains about 20 proteins, which are considered to be an allergens. Allergy is frequently induced by casein and whey proteins. Casein is fractionated into α -, β -, and κ -casein. Whey proteins include: α -lactalbumin (α -la), β -lactoglobulin (β -lg), bovine serum albumin (BSA) and immunoglobulin (Igs) (El-Agamy, 2007; Cocco et al., 2003; Jarvinen et al., 2002). The most common allergens are β -lactoglobulin, α -casein and serum albumine (Besler et al., 2001; Mills and Breiteneder, 2005). Allergy to cow milk can be observed in about 2.5 % of children below 3 years of age (El-Ghaish et al., 2011).

Food intolerance

Food intolerance is abnormal non-immune reaction with allergy-like symptoms after ingesting of food (Kayserová, 2004; Madsen, 1997; Halken, 1997; Ortolani and Pastorello, 2006). It results from enzyme deficiencies, pharmacological reactions, and response to toxic or irritant components of food (Gray and Chan, 2003).

Estimates of the prevalence of food intolerance vary widely from 2 % to over 20 % of the population (Nelson and Ogden, 2008). The prevalence of food intolerance in adults are no more than 5–6 %, in infants and young children is varying from 0,3 % to 20 % (Gray and Chan, 2003).

Symptoms of food intolerance include skin rashes, urticaria, angioedema and eczema, nasal congestion, sinusitis, pharyngeal irritations, asthma and an unproductive cough, mouth ulcers, abdominal cramp, nausea, gas, intermittent diarrhea, constipation, irritable bowel syndrome, and may include anaphylaxis (Ortolani and Pastorello, 2006; Ozdemir et al., 2009; Cardinale et al., 2009; Gray and Chan, 2003).

Intolerance to lactose

The lactose intolerance is a result of lactase deficiency and is a form of carbohydrate malabsorption. Lactose is hydrolyzed by lactase in the intestinal mucosa. The by-products of lactose hydrolysis are the monosaccharides, glucose and galactose (Wilson, 2005).

The lactase deficiency has been described as primary, secondary, or congenital ones. Primary lactase deficiency is the normal gradual reduction in lactase production seen as an individual matures from infancy into adulthood and is expressed variably across populations (Wilson, 2005). Approximately 25 % of the human population maintains a high level of lactase activity and therefore a large capacity to digest lactose throughout life (Suarez et al., 2003). Secondary lactase deficiency occurs because of gastroenteritis, bowel surgery, cystic fibrosis, or immune disorders. Congenital lactase deficiency is a rare hereditary disorder in which lactase activity is absent (Wilson, 2005).

The prevalence of lactose intolerance is lowest in people of Northern European descent (15 %) and highest in many Asian populations (near 100 %). The prevalence lactase deficiency in individuals of African descent is approximately 70–80 %. Similar level is reported for Latinos and those of Eastern and South American ancestry (Paige, 2005).

The symptoms of lactose intolerance include, for example flatulence, loose stools, abdominal pain, diarrhea, vomiting, skin irritation (Suarez et al., 2003; Wilson, 2005; Paige, 2005). Not all individuals with a reduce level of the enzyme lactase exhibit symptoms with the ingestion of dietary lactose. The presence or absence of symptoms varies with amount and type of food consumed, intestinal transit time, and the level of residual intestinal lactase (Paige, 2005).

Intolerance to gluten

Gluten is a protein, which is rich in amino acids proline and glutamine. These amino acids are collectively known as prolamins. Gluten is found mainly in foods (wheat, rye and barley) but may also be found in everyday products such as drugs or vitamins (Rimárová, 2008; El-Ghaish et al., 2011).

Consumption of gluten can lead to development of celiac disease (gluten enteropathy) in subjects with genetic predisposition (Rimárová, 2008; Hybenová et al., 2013). It causes inflammation of the small intestine, leads to numerous abdominal as well as non-gastrointestinal symptoms and interferes with absorption of nutrients from food (Counts and Sierpina, 2006; El-Ghaish et al., 2011).

Celiac disease has a four sub-phenotype:

- classic celiac disease – dominated by symptoms and sequelae of GI malabsorption,
- celiac disease with atypical symptoms – few or no GI symptoms, extraintestinal symptoms predominate
- silent celiac disease – patients are asymptomatic but have a positive serologic test
- latent celiac disease – persons are asymptomatic but are at increased risk for later development of symptoms and/or histologic changes (Counts and Sierpina, 2006).

Celiac disease (CD) affects 1 % of the children and adults in the United States and Europe with similar prevalence rates in many other countries worldwide (Hadithi and Peña, 2010).

Symptoms of celiac disease occur when dietary proteins in wheat, barley, and rye are ingested by susceptible patients, activating an abnormal mucosal immune response that damages the small intestine by inducing chronic inflammation. The most common gastrointestinal (GI) symptoms of celiac disease include diarrhea, weight loss, vomiting, abdominal pain (with or without distention), anorexia, and constipation. The most common non-GI symptoms include iron-deficiency anemia (up to 5 % of celiac patients are anemic), failure to grow, short stature, delayed puberty, infertility, recurrent fetal loss, osteoporosis, vitamin deficiencies, fatigue, protein-calorie malnutrition, recurrent aphthous stomatitis, elevated transaminase levels, and dental enamel hypoplasia. The presence of obesity does not preclude a diagnosis of celiac disease. Several neuropsychiatric conditions have been reported to accompany celiac disease, including depression, anxiety, ataxia, seizures, peripheral neuropathies, and migraines (Counts and Sierpina, 2006; Van Heel and West, 2006; Berti et al., 2006; Freeman et al., 2002).

Reduction of allergenicity by lactic acid bacteria

Fermentation by lactic acid bacteria (LAB) is one of the oldest and most economic methods of manufacture and storage of food. It represents a natural way of increasing nutritional and sensory value of foods and reducing of antinutritional factors. In addition, fermentation leads to reduction of allergenicity of foods.

It is well-known that LAB release more or less proteolytic enzymes. Because allergens are proteins, LAB may degrade them during fermentation. The proteolytic system of LAB is composed of proteinases, peptidase and peptide transport systems. It is essential for their growth (Kleber et al., 2006; Pescuma et al., 2011).

Most studies revealed that allergy to cow milk is caused mainly by casein and β -lactoglobulin (El-Ghaish et al., 2011). The reduction of milk protein antigenicity depends on the species of LAB and on condition of fermentation. Bu et al. (2010) chose three LAB strains for fermentation of milk (*Lb. bulgaricus*, *S. thermophilus* and *Lb. helveticus*) and determined the protein allergenicity after 12 h fermentation. They found out that whey protein allergenicity decreased significantly, by 53–87 % for α -lactalbumin and 86–95 % for β -lactoglobulin as compared with unfermented milk. It was demonstrated that combination of two LAB strains (*Lb. helveticus* and *S. thermophilus*) leads to the reduction of antigenicity of both whey proteins (α -lactoalbumin and β -lactoglobulin) during fermentation (Bu et al., 2010).

Pescuma et al. (2011) found out decreasing of β -lactoglobulin antigenicity during fermentation by *Lb. delbrueckii* subsp. *bulgaricus* CRL 656 and they showed for the first time that a *Lactobacillus* proteinase was able to degrade allergenic response of human sera towards this protein.

Kleber et al. (2006) observed the potential of some lactic acid bacteria in combination with *S. thermophilus* subsp. *salivarius* for the reduction of β -lactoglobulin antigenicity in sweet whey and skim milk. Reduction of more than 70 % in sweet whey and more than 90 % in skim milk was detected.

In addition to all probiotic effects associated with the consumption of yoghurt, one may expect modification of allergenic properties of milk due to the process of fermentation. Hydrolysis of β -lactoglobulin and α -lactalbumin (allergenic whey protein of milk) by lactic bacteria may decrease (99 % of antigenicity) their allergenicity (Besler et al., 2001; Bertrand-Harb et al., 2003).

Bu et al. (2010) found that at the beginning of the fermentation, the antigenicity decreased gradually but at longer fermentation time it slightly increased.

It was demonstrated that LAB have a capacity to hydrolyze the wheat gliadin fraction improving their digestibility. De Angelis et al. (2006) showed that probiotic commercial preparation VSL#3 was able to hydrolyze gliadin polypeptides during dough fermentation. They also found out that pool of lactic acid bacteria (*Lb. alimentarius* 15M, *Lb. brevis* 14G, *Lb. sanfranciscensis* 7A and *Lb. hilgardii* 51B)

was able to hydrolyse 109 of 129 ethanol-soluble polypeptides during fermentations of rye flour (De Angelis et al., 2006).

Gobbetti et al. (2007) demonstrated the hydrolysis of gliadin during long-time fermentation of dough, which was made from wheat (30 %) and non-toxic oat, millet and buckwheat flours started with the selected *Lb. alimentarius* 15M, *Lb. brevis* 14G, *Lb. sanfranciscensis* 7A and *Lb. hilgardii* 51B.

Rizzello et al. (2006) showed the capacity of the same pool of LAB to hydrolyzed wheat and rye allergens. Lactic acid fermentation caused a certain hydrolysis of albumins/globulins, and especially of gliadins.

Beyond proteolysis activity of LAB during fermentation, they may aid in the host protection against allergenic sensitization by degradation of potentially allergenic epitopes in the intestinal lumen.

Several pathologies of the gastrointestinal tract, particularly food allergy, are due to an exaggerated and imbalanced response of the gut mucosal immune system (Weid et al., 2002).

At the beginning of last century, it was observed that the consumption of fermented foods could be beneficial to health. Further, it was proposed that the health-promoting properties of such foods were imparted by fermentative microbes. It is now understood that lactic acid bacteria present in fermented foods are primarily responsible for imparting health benefits (Cross et al., 2001).

Lactic acid bacteria present in the human gut play a beneficial or probiotic role including the improvement of the local immune system. The intestinal barrier consists of physiologic and immunologic factors that restrict mucosal colonization by pathogens, prevent foreign antigens and pathogens (including food allergens) from penetrating the mucosa and regulate the antigen-specific immune responses (Rautava et al., 2005; Weid et al., 2002). Local damage may cause increasing macromolecular absorption resulting in increased systemic food allergen load, particularly in patient suffering from gastrointestinal pathology (Houben et al., 1997). Indeed, the balance of bifidobacteria versus clostridia in the neonatal flora appears to determine the allergic status in infants (Weid et al., 2002; Kalliomaki et al., 2001). In addition to bifidobacteria, several epidemiological studies clearly support the beneficial effects of lactobacilli against food allergy (Weid et al., 2002).

Scientists have attempted to select strains of LAB with immuno-stimulatory properties to use against gut diseases or to improve gastrointestinal mucosal immunity (Ortolani and Pastorello, 2006).

Also, Majamaa and Isolauri (1997) hypothesized that oral introduction of probiotics may prove to be

a useful tool for the treatment of food allergy by alleviating intestinal inflammation.

It was demonstrated that probiotic bacteria such as *Lactobacillus GG* may promote endogenous barrier mechanisms in patients with atopic dermatitis and food allergy, and by alleviating intestinal inflammation may act as a useful tool in the treatment of food allergy (Hayakawa et al., 1999; Chalk and Chalk, 2003).

On the contrary, Muraro et al. (2014) stated that the evidence that probiotic supplements have preventative or therapeutic activity for food allergy is lacking, and further research is needed to make recommendations in this area.

The mechanisms by which allergies might be reduced by consuming fermented foods is uncertain. Several theories exist, including the ability of LAB to enzymatically hydrolyze allergenic food molecules or to stabilize the gut mucosa sufficiently to reduce systemic uptake of food-borne allergens (Cross et al., 2001; Sutas et al., 1996). It is also possible that fermented foods containing LAB could serve to limit the establishment of an allergic phenotype during neonatal development (Matricardi et al., 1999).

One mechanism by which specific strains of lactobacilli may aid in host protection against allergic sensitization is the degradation of potentially allergenic epitopes in the intestinal lumen. However, only proteolysis of allergenic epitopes is not sufficient to explain the clear anti-allergic effects of certain strains of LAB that have been demonstrated (Weid et al., 2002). The permeation of antigens across the gut lining may be the primary factor in food hypersensitivities which manifest as allergic disease (Chalk and Chalk, 2003).

Most of the current probiotic foods are mainly dairy based; there is a growing interest in the development of non-dairy probiotic products due to problems such as lactose intolerance in many people and the unfavourable cholesterol content of fermented dairy products. Cereals contain water-soluble fiber (such as β -glucan and arabinoxylan), oligosaccharides (such as galacto- and fructooligosaccharides) and resistant starch, and thus have been suggested to fulfill the prebiotic concept (Rivera-Espinoza and Gallardo-Navarro, 2010).

Conclusion

Recent changes in eating habits and in the environment are thought to be connected to the recent rapid increase in food and other allergies. Food allergies are common, result in both acute and chronic disease, might be increasing in prevalence, affect quality of life, and can be severe and poten-

tially fatal. Effective management of food allergy is dependent on complete avoidance of the food allergen, patient education, and emergency treatment of anaphylaxis. Elimination diets can be expensive, are socially disruptive, and run the risk of being nutritionally inadequate.

Theory that fermented foods can benefit health has been extrapolated to the possibility that fermentative LAB could present a dietary means of anti-allergy treatment (and possibly prophylaxis), through a mechanism of immunoregulation.

Probiotic bacteria don't have only immunomodulatory effect, but they are able to reduce food allergens during the fermentation of food with allergenic potential. There is evidence that they are able to reduce the gluten content of cereals, by proteolysis they reduce albumin and globulin content in milk and prolamins in cereals.

Now, there is a challenge before us to enrich the market with ferment and probiotic foods, because our manufacture's offer is limited to dairy foods. Because milk is one of the major allergens, we have to focus on other substrates that are suitable carriers of probiotic bacteria.

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References

- Altman DR, Chiamonte LT (1997) *Environmental Toxicology and Pharmacology* 4: 95–99.
- Astwood JD, Fuchs RL (1996) *Trends in food science and technology* 7: 219–226.
- Battais F, Courcoux P, Popineau Y, Kanny G, Moneret-Vautrin DA, Denery-Papini S (2005).
- Berti I, Della Vedova R, Paduano R, Devetta M, Caradonna M, Villanacci V, Not T, Martelossi S, Tamburlini G (2006) *Digestive and Liver Disease* 38: 461–467.
- Bertrand-Harb C, Ivanova IV, Dalgarrondo M, Haertllé T (2003) *International Dairy Journal* 13: 39–45.
- Besler M, Steinhart H, Paschke A (2001) *Journal of Chromatography B*, 756: 207–228.
- Breiteneder H, Ebner C (2000) *Journal of Allergy and Clinical Immunology* 106: 27–36.
- Bu G, Luo Y, Zhang Y, Chen F (2010) *Journal of the Science of Food and Agriculture*. 90: 2015–2020.
- Cardinale F, Mangini F, Berardi M, Sterpeta Loffredo M, Chinellato I, Dellino A, Cristofori F, Di Domenico F, Mastrototaro MF, Cappiello A, Centoducati T, Carella F, Armenio L (2009) *Minerva Pediatrica* 60: 1401–1409.
- Carter CM (2003) In: Caballero B (Ed) *Encyclopedia of food science and nutrition*, Vol 4 (2642–2648). Elsevier, London.
- Chalk ChS, Chalk AJ (2003) *Journal of Chiropractic Medicine* 2: 131–133.
- Cocco RR, Järvinen KM, Sampson HA, Beyer K (2003) *Journal of Allergy and Clinical Immunology* 112: 433–437.

- Counts DR, Sierpina VS (2006) *The Journal of Science and Healing* 2: 43–45.
- Crevel RWR, Briggs D, Hefle SL, Knulst AC, Taylor SL (2007) *Food and Chemical Toxicology* 45: 691–701.
- Cross ML, Stevenson LM, Gill HS (2001) *International Immunopharmacology* 1: 891–901.
- David TJ (2005) In: Caballero B (Ed) *Encyclopedia of Human Nutrition* (265–277), Elsevier, London.
- Davis CM (2009) *Current Problems in Pediatric and Adolescent Health Care* 39: 236–254.
- Davit-Spraul A, Costa C, Zater M, Habes D, Berthelot J, Broué P, Feillet F, Bernard O, Labrune O, Baussan Ch (2008) *Molecular Genetics and Metabolism* 94: 443–447.
- De Angelis M, Coda R, Silano M, Minervini F, Rizzello CG, Di Cagno R, Vicentini O, De Vincenzi M, Gobbetti M (2006) *Journal of Cereal Science* 43: 301–314.
- De Angelis M, Rizzello CG, Fasano A, Clemente MG, De Simone C, Silano M, De Vincenzi M, Losito I, Gobbetti M (2006) *Biochimica et Biophysica Acta – Molecular Basis of Disease* 1762: 80–93.
- DIRECTIVE 2003/89/EC OF THE EUROPEAN PARLIAMENT AND OF THE COUNCIL of 10 November 2003, Official Journal of the European Union, <http://www.fsai.ie/uploadedFiles/Dir2003.89.pdf>.
- El-Agamy EI (2007) *Small Ruminant Research* 68: 64–72.
- El-Ghaish S, Ahmadova A, Hadji-Sfaxi I, El Mecherfi KE, Bazukyan I, Choiset Y, Rabesona H, Sitohy M, Popov YG, Kuliev AA, Mozzi F, Chobert JM, Haertlé T (2011) *Trends in Food Science and Technology* 22: 509–516.
- Emmett SE (1996) *Trends in Food Science and Technology* 46: 313–315.
- Esposito G, Vitagliano L, Santamaria R, Viola A, Zagari A, Salvatore F (2002) *FEBS Letters* 531: 152–156.
- Freeman H, Lemoyne M, Pare P (2002) *Best Practice & Research Clinical Gastroenterology* 16: 37–49.
- Fuchs M (2008) *Practicus* 6: 30–34.
- Gobbetti M, Rizzello CG, Di Cagno R, De Angelis M (2007) *Food Microbiology* 24: 187–196.
- Gray J, Chan W (2003) In: Caballero B (Ed) *Encyclopedia of food science and nutrition*, Vol 4 (2626–2630). Elsevier, London.
- Hadithi M, Peña AS (2010) *European Journal of Internal Medicine* 21: 247–253.
- Halken S (1997) *Environmental Toxicology and Pharmacology* 4: 149–156.
- Hayakawa K, Linko YY, Linko P (1999) *LWT – Food Science and Technology* 32: 1–11.
- Hefle SL (1996) *Food Technology* 22: 86–92.
- Hefle SL (2001) *Current Opinion in Allergy and Clinical Immunology* 3: 269–271.
- Houben GF, Knippels LMJ, Penninks AH (1997) *Environmental Toxicology and pharmacology* 4: 127–135.
- Hybenová E, Štofířová J, Mikulajová A (2013) *Potravinářstvo* 7: 95–100.
- Ispano M, Scibilia J, Ansaloni R, Rotondo F, Vannucci L, Ortolani C (1998) *Revue Française d'Allergologie* 38: 179–182.
- James JM, Sixbey JP, Helm RM, Bannon GA, Burks AW (1997) *Journal of Allergy and Clinical Immunology* 2: 239–244.
- Jarvinen KM, Busse PJ, Vila L, Beyer K, Sampson HA (2002) *Journal of Allergy and Clinical Immunology* 1: S287.
- Kalliomäki M, Kirjavainen P, Eerola E, Kero P, Salminen S, Isolauri E (2001) *Journal of Allergy and Clinical Immunology* 107: 129–134.
- Kayserová H (2004) *Potravinová alergia*, Via Practica, Bratislava.
- Kleber N, Weyrich U, Hinrichs J (2006) *Innovative Food Science and Emerging Technologies* 7: 233–238.
- Macdougall C, Etuwewe O (2005) *Current Paediatrics* 15: 228–232.
- Madsen Ch (1997) *Environmental Toxicology and Pharmacology* 4: 163–167.
- Majamaa H, Isolauri E (1997) *Journal of Allergy and Clinical Immunology* 99: 179–185.
- Matricardi PM, Rosimini F, Rapicetta M, Gasbarinni G, Stroffolini T (1999) *The Lancet*, 354: 430–430.
- Mills ENC, Breiteneder H (2005) *Biotechnology Advances* 23: 409–414.
- Muraro A, Roberts G, Worm M, Bilò MB, Brockow K, Fernández Rivas M, Santos AF, Zolkipli ZQ, Bellou A, Beyer K, Bindslev-Jensen C, Cardona V, Clark AT, Demoly P, Dubois AE, DunnGalvin A, Eigenmann P, Halken S, Harada L, Lack G, Jutel M, Niggemann B, Ruëff F, Timmermans F, Vlieg-Boerstra BJ, Werfel T, Dhimi S, Panesar S, Akdis CA, Sheikh A. EAACI Food Allergy and Anaphylaxis Guidelines Group. (2014) *Allergy* 69: 1008–1025.
- Nelson M, Ogden J (2008) *Social Science & Medicine* 67: 1038–1045.
- Ortolani C, Pastorello EA (2006) *Best Practice & Research Clinical Gastroenterology* 20: 467–483.
- Ozdemir O, Mete E, Catal F, Ozol D (2009) *Digestive Diseases and Sciences* 54: 8–14.
- Paige DM (2005) In: Caballero B (Ed) *Encyclopedia of Human Nutrition* (113–120), Elsevier, London.
- Pescuma M, Hébert EM, Rabesona H, Drouet M, Choiset Y, Haertlé T, Mozzi F, Font de Valdez G, Chobert JM (2011) *Food Chemistry* 127: 487–492.
- Rautava S, Kalliomäki M, Isolauri E (2005) *Journal of Allergy and Clinical Immunology* 116: 31–37.
- Rimárová K (2008) *Životné prostredie*, 42: 189–193.
- Ring J, Brockow K, Behrendt H (2001) *Journal of Chromatography B* 756: 3–10.
- Rivera-Espinoza Y, Gallardo-Navarro Y (2010) *Food Microbiology* 27: 1–11.
- Rizzello CG, De Angelis M, Coda R, Gobbetti M (2006) *European Food Research of Technology* 223: 405–411.
- Samartín S, Marcos A, Chandra RK (2001) *Nutrition Research* 21: 473–497.
- Sampson HA (1999) *Journal of Allergy and Clinical Immunology* 103: 717–728.
- Sampson HA (2004) *Journal of Allergy and Clinical Immunology* 113: 805–819.
- Savage JH, Matsui EC, Skripak JM, Wood RA (2007) *Journal of Allergy and Clinical Immunology* 120: 1413–1417.
- Scibilia J, Pastorello EA, Zisa G, Ottolenghi A, Bindslev-Jensen C, Pravettoni V, Scovena E, Robino A, Ortolani A (2006) *Journal of Allergy and Clinical Immunology* 117: 433–439.
- Sicherer SH (2002) *The Lancet* 360: 701–710.

- Sicherer SH, Sampson HA (2006) *Journal of Allergy and Clinical Immunology* 117: 471–475.
- Sicherer SH, Sampson HA (2010) *Journal of Allergy and Clinical Immunology* 125: 117–125.
- Skripak JM, Matsui EC, Mudd K, Wood RA (2007) *Journal of Allergy and Clinical Immunology* 120: 1172–1177.
- Smith E (1997) *Environmental Toxicology and Pharmacology*, 4: 3–7.
- Suarez F, Shannon C, Hertzler S, Savaiano D (2003) In: Caballero B (Ed) *Encyclopedia of food science and nutrition*, Vol 4 (2634–2642). Elsevier, London.
- Sutas Y, Soppi E, Korhonen H, Syvaöja EL, Saxelin M, Rokka T et al. (1996) *Journal of Allergy and Clinical Immunology* 98: 216–224.
- Svačina Š (2008) *Klinická dietologia*, Grada Publishing, Praha.
- Van Heel DA, West J (2006) *Gut* 55: 1037–1046.
- Walsh BJ, Wrigley CW, Musk AW, Baldo BA (1985) *Journal of Allergy and Clinical Immunology* 76: 23–28.
- Weid T, Ibnou-Zekri N, Pfeifer A (2002) *Digest and Liver disease* 34: 25–28.
- Wilson J (2005) *Newborn and Infant Nursing Reviews* 5: 203–207.
- Zuidmeer L, Goldhahn K, Rona RJ, Gislason D, Madsen Ch, Summers C, Sodergren E, Dahlstrom J, Lindner T, Sigurdardottir ST, McBride D, Keil T (2008) *Journal of Allergy and Clinical Immunology* 121: 1210–1218.