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Lipid transfer protein allergy: A review of current controversies

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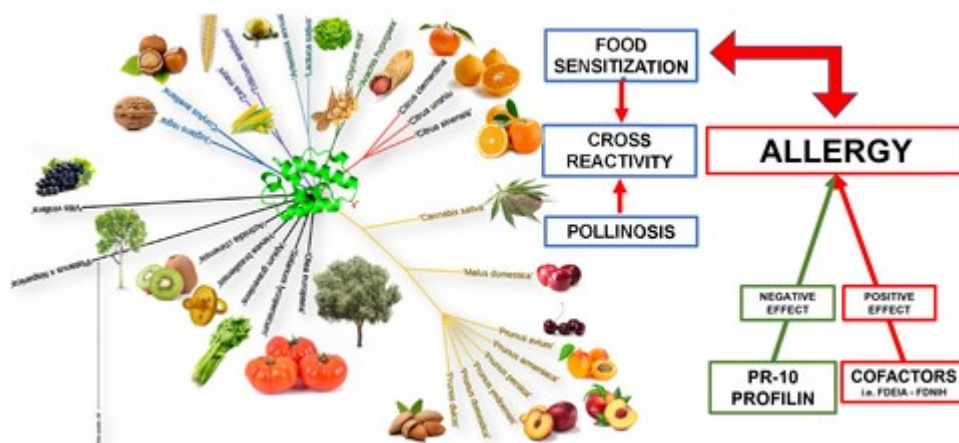
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Abstract

Sensitization to lipid transfer protein (LTP), the most frequent cause of food allergy in southern Europe, still shows several controversial, but also intriguing, aspects. Some of these include the degree of cross-reactivity between LTPs from botanically distant sources, the definition of risk factors, the role of some cofactors, clinical outcomes, geographical differences and the identification of the primary sensitizer in different areas. This review article tries to analyse and comment on these aspects point by point suggesting some explanatory hypotheses with the final scope to stimulate critical thoughts and elicit the scientific discussion about this issue in the readership.

Abstract

Sensitization to non specific lipid transfer protein (nsLTP) is the most frequent cause of food allergy in Southern Europe but is increasing in frequency across Europe. Intriguing aspects are the degree of cross-reactivity between nsLTPs from botanically distant sources, the importance of risk factors or cofactors, clinical outcomes, geographical differences and which is the primary sensitizer in different areas. This review article analyzes and comments on these aspects.



Key Messages

LTP is a plant panallergen and is the major cause of food allergy in southern Europe.

LTP allergy outcome is strongly dependent on aggravating or protective cofactors.

The sensitization process follows a rather precise hierarchical order.

1 INTRODUCTION

Non-specific lipid transfer proteins (LTPs) represent the most frequent cause of primary food allergy in adults living in Mediterranean countries and are the main cause of food-induced anaphylaxis in these areas as well.¹⁻³

Due to their widespread distribution in the plant kingdom and to their phylogenetically conserved structure, consisting of four alpha-helices stabilized by four highly conserved disulphide bridges and connected by flexible loops, it is not surprising that immunological cross-reactivity to botanically unrelated plant foods or pollens may occur in LTP-hypersensitive patients. However, this phenomenon is not constant, and offending allergen sources may show much variability from one geographical area to another. A pair of excellent and comprehensive review articles about LTP endorsed by the European Academy of Allergy and Clinical Immunology and co-authored by the world-leading experts in this field have been very recently published.^{3,4} Thus, we decided to focus the present paper on the several controversial aspects and unanswered questions that surround this particular type of food allergy, including its peculiar geographical prevalence, the cross-reactivity between homologous allergens, and the risk factors associated with sensitization to LTP. We have tried to analyse and comment point by point these aspects to stimulate critical thoughts, new ideas and fruitful discussions in the readership (see Table 1).

TABLE 1. Main unanswered questions regarding LTP allergies

Controversial issue	Probable explanation answer
Primary sensitization	Peach (Pru p 3), cannabis (Can n 3), pollen LTP (Art v 3, Pla a 3)
The route of LTP sensitization	Gastrointestinal, skin or airways
Protective cofactors (co-sensitization to other vegetable panallergen)	Occupation of FcεRI on IgE mast cells reactive to multiple allergens
Aggravating cofactors (food-dependent NSAID- or exercise-induced reactions)	Faster or more efficient diffusion of ingested allergens
Causes of LTP endemicity in some regions	Haplotype, epigenetic factors
Hierarchy of progression from botanically related to non-taxonomically related species sensitization	Somatic hypermutation
Progression from sensitization to symptomatic allergy	Behavioural cofactors

1.1 History of LTP as allergens

Lipid transfer proteins (LTPs) made their appearance as food allergens during the early “1990s” when some embryonic studies carried out in Spain and Italy appeared in the medical literature.^{5, 6} Such studies were already able to detect some of the most specific features of LTP allergy: (a) high frequency of Rosaceae (particularly peach) allergy; (b) preferential localization of the allergen in the skin of fruits; (c) molecular weight of the allergen protein of less than 10 kDa; (d) high prevalence of systemic symptoms following the ingestion of the offending foods; (e) frequent absence of associated pollen sensitization; and (f) positive skin prick tests with commercial food extracts suggesting elevated stability of the allergen.

Subsequent studies confirmed such observations⁷⁻¹⁰ leading to the definitive characterization of this food allergen. At the beginning of the new century, it became clear that LTPs were not specific to Rosaceae fruits, but could be detected also in various pollen sources¹¹ and botanically unrelated plant-derived foods, and was heat- and pepsin-resistant.^{12, 13} The list of foods found to contain LTP has increased over the years (summarized in references ³ and, ¹⁴) up to the point that this protein is now considered one of the most typical examples of panallergen (Figure 1).



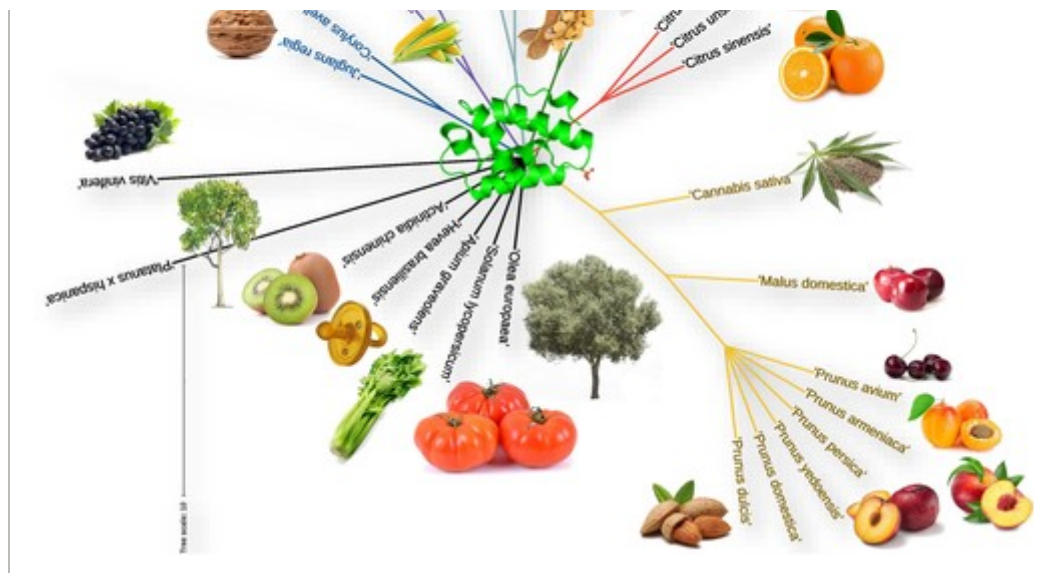


FIGURE 1

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Taxonomic tree includes some botanical sources in which LTPs have been identified, both 7 and 9 kDa MW. From the figure, it is possible to observe the botanical relationship between the different sources, often associated with a greater or lesser capacity for IgE recognition by the individual patient (i.e., patients reactive to LTP from Rosaceae—yellow—generally tolerate, e.g., those present in citrus fruits—indicated in red—as they are taxonomically distant and consequently structurally different)¹⁵

1.2 Clinical presentation of LTP allergy

One surprising aspect of LTP sensitization is the great variability of its clinical expression, encompassing contact urticaria, oral allergy syndrome, food-dependent exercise-induced anaphylaxis, urticaria/angioedema and anaphylaxis. However, sometimes sensitization is only detected by chance during the diagnostic workup carried out for different reasons, such as pollen allergy. The higher the level of specific IgE, the higher the probability of experiencing a systemic reaction upon the ingestion of offending foods, but this is not a constant rule: sometimes patients with very high IgE levels may not have any adverse reactions after ingestion of foods containing LTP.¹⁶⁻¹⁸ However, two follow-up studies showed that sensitized but tolerant patients may develop a clinical allergy to different foods (particularly belonging to the Rosaceae family, tree nuts and peanut) over time.^{19, 20}

As a difference from most other plant food allergies, contact urticaria seems quite common among LTP-hypersensitive patients. Peach-induced contact urticaria seems particularly common in Spain and Italy,²¹⁻²³ probably due to the high amount of allergen contained in peach fuzz.²⁴ However, contact urticaria from other LTP sources including asparagus,²⁵ melon²⁶ and cannabis leaves^{27, 28} has also been reported. These observations are in keeping with the prevalent surface distribution of the protein in plant foods.⁹

Another peculiar feature of LTP sensitization is the frequent need for a cofactor for the

allergic reaction to occur. In most cases, the cofactor is exercise; in fact, allergy to LTP is the most frequent cause of food-dependent, exercise-induced anaphylaxis (FDEIA) in the countries where this kind of food allergy is common,²⁹⁻³² although cases from other geographical areas have also been reported.³³ Another cofactor that may facilitate the clinical expression of LTP sensitization is non-steroidal anti-inflammatory drugs (NSAIDs). The association between LTP hypersensitivity and NSAID intolerance has been reported by several researchers.³⁴⁻³⁷ In effect, NSAIDs appear to enhance the IgE-mediated activation of effector cells, which may amplify the histamine release induced by the allergenic protein, as demonstrated by Pascal M et al.³⁸ It has recently been shown that food-dependent NSAID-induced hypersensitivity reactions (FDNIH) are associated with a considerable percentage of patients with a clinical history of NSAID hypersensitivity, LTP and gliadin being the main risk factors. In this regard, FDNIH should be always suspected and food allergy tests included in the diagnostic workup of patients with a clinical history of NSAID hypersensitivity.³⁹

1.3 Geographical differences in the prevalence of LTP sensitization/allergy

Sensitization and allergy to LTPs unquestionably prevail in adults and children living in southern European countries, either Mediterranean or Atlantic ones, such as Portugal.^{1, 40-42} Nonetheless, even within these areas, there are marked differences in the prevalence of sensitization. For instance, in Italy, the prevalence markedly increases from the north of the country, characterized by a continental climate, to the centre-south of the country, characterized by the typical Mediterranean climate.^{1, 43} In particular, significantly lower prevalence (10.8% and 11.5%, respectively) was recorded in the north-east and north-west of Italy than in the south of Italy (27.2%), central (21.3%) and isles (22.3%).⁴³ Spanish studies also found differences in the prevalence of LTP sensitization/allergy among different areas of that country.^{44, 45} Due to unclear reasons, the prevalence of LTP sensitization/allergy suddenly drops north of the Alps⁴⁶ and remains low throughout the rest of Europe, although sparse reports are gradually appearing in the medical literature.

In Belgium, LTP sensitization is not infrequent in atopic subjects but is mostly clinically irrelevant⁴⁷ with some exceptions.⁴⁸ In Austria, the few patients showing severe peach allergy are sensitized to LTP,^{49, 50} although one could hypothesize that an important role in the spreading of LTP reactivity might be driven by the extensive growing of apricots rather than peaches. Notably, Pru p 3 from peach and Pru ar 3 from apricot (*Prunus armeniaca*) show 92.21% of sequence identity (83 identical positions and 6 similar positions out of 91 amino acids). Cases of apple, blueberry, and wine allergy in LTP-hypersensitive allergic patients have been reported from Germany.^{33, 51, 52} Eggplant allergy has been reported from LTP-hypersensitive allergic patients in Poland,⁵³ and Cor a 8 from hazelnut allergy from the Netherlands.⁵⁴ In the UK, LTP sensitization was associated with clinical reactivity to an array of plant-derived foods, different from those involved in adverse reactions in Italy,^{55, 56} although the molecular sensitization profile was almost identical.⁵⁵

Outside Europe, relevant LTP hypersensitivity/allergy has been reported from some regions of China.^{57, 58} One case has been reported from Australia,⁵⁹ while no case has been detected so far from the American continent and Africa. In Japan, also, LTP allergy is very rare, probably due to the habit of eating a peeled peach so that the sensitization occurs more frequently to peamaclein, a cysteine-rich protein belonging to the family of the gibberellin-regulated protein (GRP). They are cross-reactive molecules shown in cypress pollen (Cypmaclein), peach (Pru p 7—peamaclein), apricot (Pru m 7), orange (Cit s 7) and pomegranate (Pun g 7), and present both in the skin and in the pulp of the fruit, associated in many cases with systemic symptoms after ingestion.⁶⁰

Looking at these data, one observation is that, interestingly, outside the “LTP endemic area” where the peach uniformly and largely dominates the field, the foods involved in LTP allergy show much more unpredictability and are often biologically distant from the Prunoideae family.

1.4 The relevance of pollen LTPs and the question of the primary sensitization to the protein

Given the geographical differences reported above, it is not surprising that a primary pollen LTP sensitization has been hypothesized. Several pollen sources, including plane (Pla a 3), mugwort (Art v 3) and olive (Ole e 7), have been proposed as primary sensitizers over the years.^{57, 61-66} However, commercial extracts of these pollens are regularly used for skin prick testing in “endemic LTP areas,” and score negatively in most LTP-hypersensitive patients.⁶⁷ Further, a very recent study addressing this issue based on specific IgE level measurements and inhibition studies concluded that the possibility of a primary pollen LTP sensitization seems very unlikely, in endemic LTP areas.⁶⁸ A possible exception seems to be China, where the link between LTP sensitization and mugwort pollen allergy appears to be stronger.⁵⁷ An interesting experimental model of the nasal allergen provocation test with Pru p 3 and Art v 3 demonstrated that pollen LTP can induce rhinitis in sensitized patients, also suggesting that a primary sensitization to Pru p 3 can drive to respiratory symptoms through cross-reactivity.⁶⁹

One interesting point is the apparent inverse relationship between the prevalence of birch pollen allergy and the prevalence of LTP allergy,^{43, 70} along with the “protective effect” against severe food-allergic reactions exerted by the co-sensitization to the major birch pollen allergen, Bet v 1, and/or the pollen plant allergen, profilin, in Italian patients.^{61, 71}

A probable cause for the protective effect of co-sensitization to LTP and other vegetable panallergens, such as profilin and/or PR-10 allergens, could be that in monoreactive individuals, the IgE receptors on the mastocytes are solely occupied by LTP-specific IgE, thus facilitating a more efficient allergen-induced crosslinking of the IgE receptor and subsequent mastocyte activation.⁷²

If this is the case, where does LTP sensitization start from? Which risk factor for LTP sensitization shows such a marked difference between the different areas of the world?

It has been postulated that LTP sensitization occurs in most cases via the gastrointestinal route.⁷³ However, if this were the case one could expect that LTP allergy occurs world-wide in genetically predisposed individuals, like fish or shrimp allergy, but things are markedly different.

Notably, LTP hypersensitivity is often already present in early life⁷⁴ when the sensitization through the skin, facilitated by the disruption of the cutaneous barrier in children with atopic eczema, may precede the first ingestion of several foods. Percutaneous LTP sensitization may occur also in adults; for instance, many Italian peach pickers need to wear gloves during their work due to the development of contact urticaria as an occupational disease.²⁴ Further, peach-induced contact urticaria represents the most frequent clinical expression of LTP allergy, mainly in patients who have reported the onset of systemic symptoms immediately after exposure.²¹ Altogether, the available evidence suggests that sensitization through the skin may represent a relevant mechanism in LTP allergy. Again, this does not per se explain the geographical differences in the prevalence of this allergy. However, one should consider that Pru p 3 is mainly concentrated in peach fuzz (in fact, many peach-allergic patients tolerate nectarines without any problem), and fuzz is mostly removed from the fruit before its marketing,²⁴ thus eliminating the most efficient vehicle of Pru p 3.

Direct sensitization to peach LTP (Pru p 3) may also occur via the airways, with respiratory symptoms preceding food allergy,^{75, 76} while in other cases, sensitization may occur to the Pru p 3 allergen released into the air from the leaves in areas with extensive orchard-tree crops.⁶⁶ While this latter mechanism may explain the development of “local” elevated frequencies of LTP sensitization, it cannot explain the geographical differences in LTP allergy. In Italy, peaches are not increasingly grown from north to south but mainly in two specific regions (Emilia Romagna and Campania) and no data seem to support a higher prevalence of LTP allergy in these two regions.¹ In contrast, if sensitization occurs via inhalation of peach fuzz after fruit picking, the hypothesis remains valid. One of the possible reasons for a low LTP hypersensitivity in some Italian regions where peaches are grown might be that the early ingestion of ripened fruits induces early gastrointestinal tolerance, but currently, there is no evidence for this.

Occupational allergy to LTP has been reported in workplaces involved in the processing of different foods.^{25, 77-81} Finally, LTP sensitization may be initiated by the inhalation/contact of Cannabis LTP, Can s 3, even via passive smoke, as demonstrated even in paediatric patients who had developed LTP-triggered anaphylaxis after a second-hand exposure to cannabis smoke.⁸² Cannabis LTP sensitization might precede the development of food allergy⁸³⁻⁸⁸ and therefore represents a further form of LTP sensitization even in other geographical areas, rather than the endemic regions (e.g., Mediterranean), such as northern Europe.⁸⁵⁻⁸⁷

An important factor able to influence the clinical outcome of patients sensitized to LTP is the presence or absence of co-sensitization to PR-10 or profilins.^{70, 71} Monoreactive LTP patients show more frequently severe reactions than polyreactive individuals.⁷⁰ Notably, hypersensitivity to Fagales (i.e., to PR-10s) is frequently found in northern Italy and northern Europe, while it is rare in southern Italy and Spain. In effect, monoreactive LTP patients in northern Europe are exceptional.

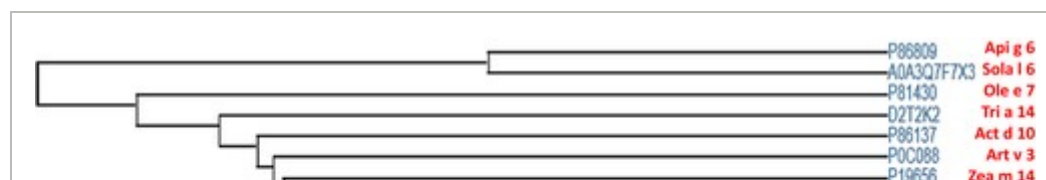
Furthermore, it is not yet known which clinical picture characterizes cases of simultaneous sensitization to LTP and GRPs and whether this might represent an aggravating factor in the clinical outcome of patients.

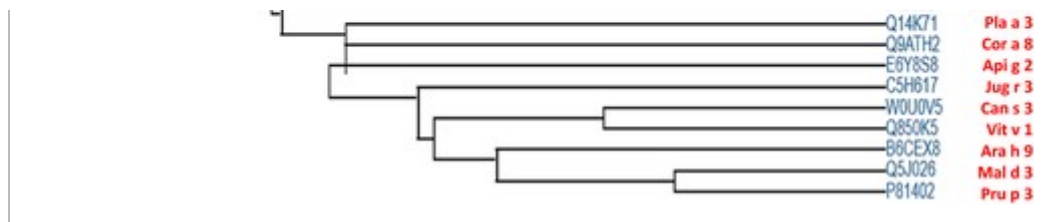
1.5 Hierarchy in LTP sensitization

The LTP-hypersensitive patient may show a spectrum of co-recognitions ranging from one single food (almost invariably the peach, at least in Mediterranean countries) to a large number of botanically unrelated plant foods. In general, this phenomenon is strictly linked to the level of Pru p 3-specific IgE. Patients showing low Pru p 3 IgE levels virtually never co-recognize LTPs from other food sources. The higher the Pru p 3 IgE levels are, the higher is the number of foods other than the peach that are co-recognized.⁸⁹ This process seems to follow a rather precise and predictable sequence starting with the peach and following with other Rosaceae fruits, through tree nuts (hazelnut and walnut), peanut and, much less frequently, cereals (maize, rice), cabbage, mustard, beer, vegetables (lettuce) and many other foods.^{44, 90} The behaviour suggests that (at least in Mediterranean countries) Pru p 3 represents the primary sensitizer containing the most relevant allergenic epitopes of LTP, while the LTPs from other sources show a gradually more limited number of epitopes and need higher levels of Pru p 3-specific IgE to be co-recognized (see Figure 1). At least in Italy, it is virtually impossible to detect an LTP-hypersensitive patient non-sensitized to the peach, and the fact that the peach dominates this kind of food allergy has been observed also in non-Mediterranean countries.⁵⁰

Recently, additional nsLTP molecules, showing a molecular weight of either 7 kDa or 9 kDa, have become available for in vitro diagnostics by multiplex tests. This has made it possible to investigate novel molecular recognition profiles.⁹¹

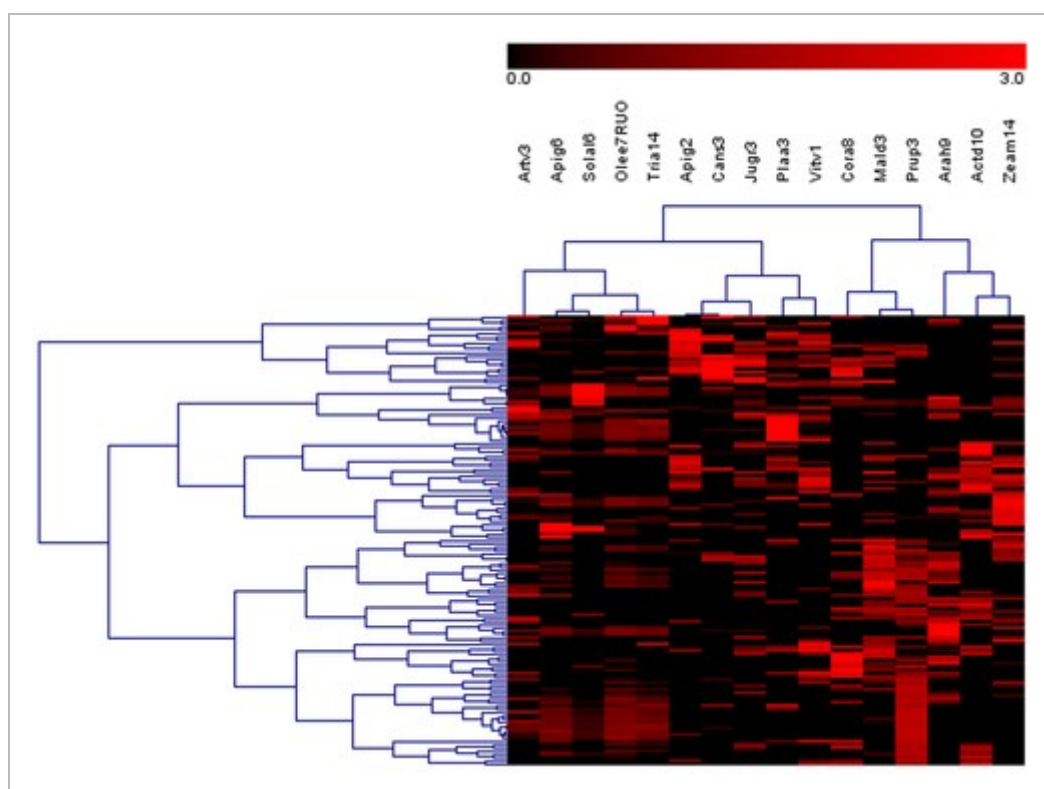
By comparing one another the structural identity of these molecules, it is possible to observe how these form clusters that largely depend on their botanical origins and phylogenetic relationships. For instance, the 2 nsLTPs of 7 kD form a cluster, as do grape (*Vitis vinifera*) nsLTP, Vit v 1, and hemp (*Cannabis sativa*) nsLTP, Can s 3, or apple (*Malus domestica*) Mal d 3 and Pru p3 (Figure 2).



**FIGURE 2**[Open in figure viewer](#) | [↓ PowerPoint](#)

Dendrogram represents in the “x”-axis the logical distance of the clusters according to the defined metric, and in the ordinate axis, the hierarchical level of aggregation between a group of nsLTPs, currently available for in vitro diagnostics. From the tree diagram, it can be seen that the two 9 kDa LTPs evaluated, Api g 6 from celery and Sol al 6 from tomato, form a cluster due to their structural peculiarities, separated from the other 7 kDa LTPs

Interestingly, in a group of 140 patients reactive/allergic to LTP, we found similar recognition clusters suggesting that the presence of specific IgE targeting a given biological source could hypothetically be the result of the recognition of another related source. Thus, one must be very cautious in defining a given molecule as the cause of sensitization in a certain patient (Figure 3).

**FIGURE 3**[Open in figure viewer](#) | [↓ PowerPoint](#)

Unsupervised bidirectional hierarchical clustering analysis generated different patterns of IgE reactivity in our population (x-axis) as a consequence of the recognition of the IgE of LTP belonging to different biological sources (y-axis). Two main IgE recognition clusters can be observed: the first, formed by 2 subclusters including Pru p 3 from

peach and Mal d 3 from apple; and the second, again with 2 subclusters comprising 9 kDa molecules and LTP from *cannabis sativa*. This observation suggests the presence of different subsets of patients in real life and possibly the existence of distinct sensitization pictures

2 CONCLUSION

Lipid transfer proteins represent a typical plant panallergen, so much so that they are recognized as the major cause of food allergy in southern Europe, even if in the last decade the sensitization/allergy to this protein is increasing all over Europe. The geographical distribution of this food allergy follows a north-south gradient but does not seem to depend on the presence of fruit orchards. Following the geographical distribution, the clinical outcome also follows a similar trend. Symptoms are particularly severe in southern European countries, albeit with large variations between regions and regions, while in northern European countries, hypersensitivity to LTP is often clinically irrelevant and sometimes detected by chance during the diagnostic workup. It is widely known that sensitization and allergy do not have the same meaning, but a simple sensitization can subsequently turn into symptomatic allergy, over the years. In the case of LTP, the allergic manifestations are influenced by aggravating and protective factors, so the distinction between the two conditions is particularly complex. Indeed, LTP allergy outcome is strongly influenced by protective factors (e.g., sensitization to other vegetable panallergens, such as PR-10 / profilin) or by aggravating cofactors (e.g., physical exercise or NSAID intake, within a few hours of taking LTP-related allergenic sources). The sensitization process follows a rather precise hierarchical route, from a single fruit (i.e., peach) to the progressive recognition of more and more LTP molecules, initially botanically related (i.e., LTP from other *Rosaceae* fruits), and subsequently also taxonomically unrelated. Finally, an open question remains the route of LTP sensitization. Although it has been postulated that LTP sensitization occurs in most cases via the gastrointestinal route, there is evidence that sensitization through the skin and the airways may represent a relevant mechanism in LTP allergy.

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CONFLICT OF INTEREST

All the authors do not have any conflict of interest to declare regarding this manuscript.

AUTHOR CONTRIBUTIONS

All the authors have contributed equally to the writing and reviewing of this article.

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