Omics and Allergic Diseases: Fact or Fiction

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OMICS technologies are focused on the massive analysis of systems biology levels. This means analysis of the different steps in the biological process and metabolism, which, in the end, are responsible for the clinical phenotype.[1,2] This process starts with the genome, which encompasses the complete set of DNA, including the coding and noncoding genes. Coding genes produce all transcribed RNAs, which are expressed and transcribed into proteins. These, due to their biological activities, determine the metabolites. This mechanism is complex and full of interconnections among levels; thus, information from all levels is necessary to fully understand systems biology.[2] Although there are more, genomics, transcriptomics, proteomics, and metabolomics are the main technologies that represent each broad level.

INTRODUCTION

A single omics technology can characterise a biological organism at its level, but it is multi-omics integration that will help obtain the overall biological meaning. Clinical decisions should continue based on clinical history but combined with biomarkers coming from the characterisation of molecular phenotypes, such as a set of genes, transcripts, proteins, and/or metabolites, as well as cellular characteristics, to reach personalised medicine.[3]

In the context of allergy, we have an umbrella of different diseases, including allergic rhinitis, asthma, drug allergy or hypersensitivity drug reactions, food allergy, and atopic dermatitis. These have a significant impact on the global population.[2] Most of them have a common feature to be elicited by effector cells (including mast cells, eosinophils, basophils) and the release of pro-inflammatory cytokines and metabolic mediators upon exposure to an allergen.[2] However, important questions, such as the molecular mechanisms underlying different endotypes to make

a precise diagnosis, or how the patient evolves from a mild allergic phenotype into a severe one, or biomarkers for monitoring the response to allergen-immunotherapy, are still unsolved; here, omics technologies can be applied.

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In a recent publication, the results of the DIFAMEM clinical trial, funded through the Joint Programming Initiative – A Healthy Diet for a Healthy Life (JPI HDHL) European Research Area Network (ERA-NET), a European programme supporting transnational research on nutrition and health, were reported. The study adopted a multi-omics approach to investigate food allergy (FA).[4] Lipid transfer protein (LTP) allergy is a prevalent FA in adults and is very heterogenous, as symptoms can range from mild manifestations such as oral allergy syndrome, to life-threatening reactions, including anaphylaxis. This means that allergen immunotherapy is not beneficial for some patients with LTP allergy. FA has been associated with microbial dysbiosis; the use of prebiotics has emerged as a promising option to restore the microbiome.[5,6] Thus, the aim of DIFAMEM was to evaluate the use of prebiotics to treat LTP allergy.

The prebiotics employed were pectins, which are natural polysaccharides usually found in the peel and pulp of fruits. Based on their esterification degree, pectins can be classified as low- or high- methoxyl pectins (LMP or HMP, respectively). Although both pectins have shown potential to be immune regulators, LMP is more fermentable by the microbiota than HMP.[5,6] In DIFAMEM, 37 patients with a clinical history of FA, who were sensitised to the LTP allergen from peaches, Pru p 3, were recruited.[4] Patients were randomly allocated into three groups to receive placebo or one of two pectin varieties with different degrees of esterification: citrus-derived pectin (CP), a LMP pectin derived from citrus fruits; and AP, an HMP pectin derived from apples.[4] Serum and faecal samples were obtained before and after 2 months

of intervention.[4] Serum samples were used to perform targeted proteomics, whereas faeces were used for metagenomics and targeted metabolomics. Finally, the generated data were integrated using bioinformatic tools.[4] Additionally, to assess the clinical efficacy of the pectin treatment, patients were on a double-blind placebo-controlled food challenge (DBPCFC) to Pru p 3, before and after the pectin intervention.[4]

Clinical characteristics of the enrolled patients with LTP allergy indicated that the majority of the participants were female and presented with rhinitis. Participants were positive to inhalant allergens, being mainly sensitised to olive and plane tree pollens. Regarding other LTP sensitisations, peanuts, walnuts, and hazelnuts were the main plant foods that participants were sensitised to. Results from the DBPCFC confirmed that none of the participants reached the maximum dose of the challenge, and only mild and moderate reactions were observed.[4] These findings suggested that the groups were very homogenous before starting the treatment with pectin.

After the 2-month intervention, no significant differences were found for specific IgE (sIgE) or skin-prick test, except for an increase in the wheal area for the CP after the treatment. Regarding the amount of Pru p 3 tolerated after the treatment, the authors found a significant increase in tolerated allergen in around 50% of the patients from both pectin groups, but not in the placebo group.[4] Interestingly, following pectin treatment, four patients in the CP group and five patients in the AP group tolerated the maximum cumulative dose of Pru p 3, equivalent to the amount of allergen present in one medium-sized peach.

Regarding the systemic proteins, the authors found that several of the significantly altered proteins highly related to the Th2 response. Common to both pectins, they found a decrease in IL-13 and IL-24 compared to placebo. However, specific changes were found for each pectin. For the AP treatment, IL-4 and TSLP were found to be reduced compared to placebo, suggesting a higher impact on the Th2 response. In the CP group, IL-33 and IL-17C were observed to be decreased compared to placebo, suggesting the downregulation of the Th17 response.[4] Although these findings suggest a systemic decrease of the allergic response, they point out that each pectin follows a different path.

Regarding the faecal metagenomics analysis, contrary to what was expected, the α-diversity of the gut microbiota of both pectin-treated groups was reduced after the intervention. Bacterial diversity reduction was associated with an increase in the abundance of pectin-degrading taxa. Some of these alterations were common to both pectins, whereas others were specific to each treatment. Among these, the authors highlighted the increase in the *Bacteroides* genus in the case of the CP, and *Bifidobacterium* in the case of AP.[4]

The metagenome prediction with Kyoto Encyclopedia of Genes and Genomes (KEGG) pathways showed five pathways that were common to both pectins. Among them, the production of microbial-derived metabolites, such as primary and secondary bile acids biosynthesis, was highlighted.[4] Driven by these findings, bile acids and short-chain fatty acids (SCFA) were analysed in faecal samples.

The faecal metabolome was also observed to be modified by both pectins. Surprisingly, no significant differences were observed in the canonical SCFA (i.e., acetic, propionic, and butyric acids). Instead, reduced concentrations of branched-SCFA in both pectins, namely isobutyric, isovaleric, and 2-methylbutyric acids, were found in the pectin-treated groups. These are the bacterial products of the fermentation of the branched-chain amino acids.[4] Additionally, the authors confirmed alterations in the levels of primary and secondary bile acids modified by both pectins when compared to placebo.[4]

Due to the potential interaction between the faecal metabolites and proteins and bacterial taxa, correlation analyses were carried out. Branched-SCFA were significant and positively correlated with Th2-type inflammatory cytokines such as IL13, IL4, and IL33, and with the Th17-response by the cytokine IL17C.[4] These associations were not observed for the canonical SCFA. These findings highlighted their yet unexplored importance.

Multi-omics integration revealed a possible stronger immune modulation by AP treatment, as the analysis showed one model that clustered the AP group separately from the others.[4]

Discussing the title of this presentation, are the omics findings a fact or fiction? The interest in omics findings in allergy has been captured in different and important reviews.[2,3,7] A position paper published last year points out that in the near future, the analysis of some omics markers could be of help to detect patients at asthma risk, defining asthma endotypes, or the severity of the patients.[2]

CONCLUSION

In summary, omics are rapidly expanding our knowledge of allergic diseases. We are still in the process of extracting critical information that can be effectively used in clinical practice. As shown by the DIFAMEM clinical trial, discovered molecular targets should be explored in follow-up studies to better understand their role in the allergic process.

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