



Causes of Infrequent Prevalence of Eosinophilic Esophagitis in Hispanics May Involve Social and Cultural Factors: Probable Role of Digestive Microbiota

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To the Editor,

We read with great interest the article by Yu Ch et al. who found in a cohort study that the prevalence of eosinophilic esophagitis (EoE) is significantly lower in Hispanic population compared to Caucasians. They also found that analysis of gene expression had similar results in subjects with EoE from both ethnic groups. The authors concluded that environmental, racial, and cultural factors should be studied to explain these marked differences [1]. A low prevalence of EoE has also been observed in our Latin American countries in the few studies published so far [2, 3].

In the light of current knowledge, we think that the prevalence of EoE in a given population is determined by the interaction of three factors with variable specific weight: (1) genetics, (2) environmental allergens, and (3) immunological susceptibility. Firstly, genetic factors have recently been identified [4]. The estimated sibling recurrence rate in EoE is approximately 80 percent, indicating a potentially significant role of genetics in this disease. In another study, the gene that encodes eotaxin-3 was the most upregulated gene in EoE [5].

Secondly, there exist some aeroallergens and food-based allergens that could have a central role. Notwithstanding, why foods and pollens that had been tolerated by humans for thousands of years now trigger EoE? Maybe sensitization of individuals (the third crucial factor) could explain this issue. This is also extended to other allergic diseases mediated through Th2 inflammatory response (implicated in the

pathophysiology of EoE) such as asthma, atopic dermatitis, and allergic rhinitis. It has been shown that these allergic diseases have increased their incidence in developed countries in the last two or three decades [6].

The increase in sensitization could be the result of the modification of lifestyles as a consequence of the improvement of the socioeconomic levels in developed countries in the last decades. For example, it has been observed that the prevalence of EoE is inversely proportional to the prevalence of infection by *Helicobacter pylori* [7], which is still highly prevalent in Latin populations [8] and is related to low socioeconomic levels. Whether or not *Helicobacter pylori* could protect against EoE is a non-cleared issue. We think that the presence of the infection could be the reflection of modifications of the digestive microbiota, which is determined from birth and can suffer variations due to cesarean delivery, antibiotics use, type of diet, breast-feeding, repeated gastrointestinal infections, and rigorous aseptic codes. Along with these findings, the hygiene hypothesis postulates that the elimination of non-lethal bacterial infections mediated by the inflammatory reaction Th1 could increase the expression of inflammatory Th2 response [9]. Dysbiosis of the intestinal microbiota during the neonatal stage is linked to the development of atopy and asthma in childhood [10]. This linkage has not been studied for EoE. On the other hand, the microbiota of the stomach and esophagus have been poorly studied. In the future, these hot topics could be the subject of research to better understand the pathogenesis of EoE and the difference in prevalence among diverse populations throughout the world.

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References

1. Yu CH, Sterling D, Albayati I, et al. The prevalence of biopsy-proven eosinophilic esophagitis in hispanics undergoing

- endoscopy is infrequent compared to caucasians: a cross-sectional study. *Dig Dis Sci*. 2017;62:3511–3516.
2. De la Cruz-Patiño E, Ruíz Juárez I, Meixueiro Daza A, Grube Pagola P, Roesch-Dietlen F, Remes-Troche JM. Eosinophilic esophagitis prevalence in an adult population undergoing upper endoscopy in southeastern Mexico. *Dis Esophagus*. 2015;28:524–529.
 3. García-Compeán D, González González JA, Marrufo García CA, et al. Prevalence of eosinophilic esophagitis in patients with refractory gastroesophageal reflux disease symptoms: a prospective study. *Dig Liver Dis*. 2011;43:204–208.
 4. Blanchard C, Wang N, Rothenberg ME. Eosinophilic esophagitis: pathogenesis, genetics, and therapy. *J Allergy Clin Immunol*. 2006;118:1054–1059.
 5. Shoda T, Morita H, Nomura I, et al. Comparison of gene expression profiles in eosinophilic esophagitis (EoE) between Japan and Western countries. *Allergol Int*. 2015;64:260–265.
 6. Masoli M, Fabian D, Holt S, Beasley R; Global Initiative for Asthma (GINA) Program. The global burden of asthma: executive summary of the GINA dissemination committee report. *Allergy*. 2004;59:469–478.
 7. Dellon ES, Peery AF, Shaheen NJ, et al. Inverse association of esophageal eosinophilia with *Helicobacter pylori* based on analysis of a US pathology database. *Gastroenterology*. 2011;141:1586–1592.
 8. Porras C, Nodora J, Sexton R, et al. Epidemiology of *Helicobacter pylori* infection in six Latin American countries (SWOG Trial S0701). *Cancer Causes Control*. 2013;24:209–215.
 9. Okada H, Kuhn C, Feillet H, Bach JF. The ‘hygiene hypothesis’ for autoimmune and allergic diseases: an update. *Clin Exp Immunol*. 2010;160:1–9.
 10. Hill DA, Artis D. Intestinal bacteria and the regulation of immune cell homeostasis. *Ann Rev Immunol*. 2010;28:623–667.