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Reactive Nasal Inflammation: Current Pathophysiological and Therapeutic Approach

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1. Editorial

The pathophysiology of processes that underlie the onset and progression of reactive nasal inflammatory conditions is very complex. These include a heterogeneous group of disorders, ranging from seasonal allergic rhinitis to nonallergic, persisting, refractory forms of chronic rhinosinusitis (CRS). About 400 million people worldwide are affected by allergic rhinitis and another 200 million are thought to be affected by nonallergic forms of nasal inflammation including CRS [1,2]. The overall prevalence of these conditions has been on a steady rise for almost 25 years concomitant with gross environmental changes in developed and developing countries [3]. While the inflammatory responses underlying allergic rhinitis are triggered by exposure to molecules with intrinsic allergenic properties, which promote type 2 T helper cell- (Th2-) biased, IgE-dependent immune responses, triggers of nonallergic rhinitis or CRS are nonspecific and largely unknown [5,6]. Regardless, a number of common factors variably contribute to favoring and worsening the inflammatory response in these reactive nasal conditions [7,8]. These include the innate and adaptive immune system, the epithelial barrier function, a neuroinflammatory component (i.e., neurogenic inflammation), tissue remodeling processes and the nasal microbiota [9,10].

The relationship between nasal dysbiosis and reactive, allergic or nonallergic, nasal inflammation involves a complex network of processes regulating mucosal permeability and TJ function, neurogenic signals, innate immunity cells and receptors, vascular and mucosal remodeling factors, effector T cells and related cytokines and the production of specific IgE or IgA antibodies.

The literature to date has not clarified the timing and reciprocity of these connections and whether, for instance, intrinsic alterations in the mechanisms governing barrier function would typically precede or follow immune activation and inflammation. Moreover, the precise mechanisms that lead to distinct clinical phenotypes and endotypes and the inherent specific inflammatory processes are still largely unknown.

Regardless, it can be concluded that the barrier function of the nasal mucosa, or mucosal firewall, represents the key element linking nasal dysbiosis to the cellular and molecular processes that lead to and sustain inflammation.

An increased mucosal permeability may in turn favor bacterial translocation to the submucosa, where

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antigen presentation and recognition take place, as well as the interaction of bacterial components with innate immune and nociceptive receptors.

In light of these considerations, given the complex interactions between the microbial microenvironment, the nasal epithelium, the innate and adaptive immune system and the nasal nervous system, it would be quite reductionist to classify nasal inflammatory processes based on the prevailing inflammatory cytotypes, for example, neutrophils, eosinophils, or mast cells, but should include at a minimum a definition of the immune phenotype or endotype to allow for a more targeted and effective line of intervention in the clinical management of patients with these conditions [11,12].

In this light, even the resection of largely hyperplastic or extended, frankly polypoid lesions of the chronically inflamed nasal and sinus mucosa by minimally invasive, functional endoscopic surgery would not be seen as just the last resort whereby all other treatments have failed, but as the integral part of an organic strategy including conventional and biotechnological anti-inflammatory agents, antibiotics and probiotics

The rationale for probiotics administration in allergic rhinitis, CRS and related nasal reactive disorders comes from studies documenting the antagonistic interactions of symbiotic and pathogenic species within the nasal mucosa or other niches [13-15] and the ability of certain symbiotic species to regulate the fine balance between host immunity and tolerance. The possible mechanisms mediating disruption of the basic homeostatic functions of the human nasal mucosa concomitant to alterations in the local microbiota, which have been documented in nasal inflammatory conditions. Much of our knowledge comes from studies of gut bacterial communities, which provide a solid basis to understand the complex interactions between the host mucosa and the microbial milieu. Future studies will hopefully reveal how unique changes in the nasal microbiota, including viral and fungal components, result in distinct clinical phenotypes and how its manipulation may contribute to their current and prospective treatments.

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