Reply to: Characterisation of crevicular fluid microbiota in primary Sjögren's disease

Sirs,

We thank Dr. Vissink et al. for their interest in our work. There is a significant difference in the literature about the prevalence of periodontal disease (PD) among patients with primary Sjögren's disease (SjD). The prevalence reported in several clinical studies ranges from 30% to 83.9% (2-4), in contrast to the prevalence of 7.5% recorded by the Norwegian registry (1). In our study, we observed that 97.7% of patients had PD on at least one tooth. We totally agree that the patients' origin (dental offices, rheumatology clinics, or national registries), as well as the disease definition, different periodontal examination protocols and units of analysis have an impact on the prevalence. Furthermore, an uneven distribution of PD has been found among the different racial/ ethnic groups. For instance, among Americans, Hispanic Americans had the highest prevalence of periodontitis (63.5%), followed by African Americans (59.1%), and Caucasian Americans (40.8%). In addition. the lowest and highest socioeconomic categories had a twofold difference in prevalence (5). Specifically, in a study in the Mexican general population with a low socioeconomic level, the average number of sextants with no PD at clinical examination was reported to be 1.8 per person (6). Therefore, our prevalence may have been influenced by the aforementioned characteristics; yet, this does not make our findings invalid.

Moreover, there is disagreement and discussion in Sjögren's literature regarding the link between PD and primary SiD. Regarding this, there was no discernible difference in periodontitis between primary SiD patients and controls according to the meta-analyses by De Goés et al. (which included nine studies) (7) and Maarse et al. (which included ten studies) (8). Wu et al. pointed out that the inclusion of various kinds of controls in the Maarse trial and the failure to convert standard errors into standard deviation in the Goés study could have skewed these results. As a result, the authors conducted out a new meta-analysis and made the necessary corrections. When comparing SjD patients to healthy controls, they found a detrimental effect on the gingival index (GI) and plaque index (PI), but not on the clinical attachment level (CAL) or probing pocket depth (PPD). Thus, the

authors were unable to draw a conclusion supporting or refuting a link between SiD and PD (9). Later, a new meta-analysis now by Yang et al. which included five trials, revealed an OR of 2.1 for the association between SiD and PD. Then, when the same meta-analysis included 16 papers with complete data, the authors found that while bleeding on probing and GI scores were greater in SjD patients, PPD, CAL, and PI scores were not. The authors concluded that SjD patients were more likely to be diagnosed with PD, although there was high heterogeneity among the studies (10). Similarly, information from national registries varies as well. For instance, the Taiwan's National Health Insurance Research Database, which includes 7709 primary SjD patients and 7090 controls, reported an RR for periodontitis of 1.44 in patients with SjD regardless of disease duration, age, or gender (3). Conversely, the nationwide study conducted in Norway, which included 760 primary SjD patients and 22178 controls, found no statistically significant difference in periodontitis between SiD patients and a control group (1). As a result, we concur with an earlier review of this topic (4), which noted that the literature provides some degree of evidence for an increased risk of periodontitis in SjD, but the opinion is not unanimous. This debate will continue until well-designed studies that account for confounding variables are developed.

G.A. MARTÍNEZ-NAVA¹², PhD C. HERNÁNDEZ-HERNÁNDEZ3, MD V. RUIZ-GONZÁLEZ3, MD L. LLORENTE⁴, MD G. HERNÁNDEZ-MOLINA⁴, MS

¹Laboratorio de Gerociencias, Instituto Nacional de Rehabilitación Luis Guillermo Ibarra Ibarra, Mexico City; ²Centro de Investigación sobre el Envejecimiento, Mexico City; ³Servicio Dental, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City; ⁴Departamento de Inmunología y Reumatología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City,

Please address correspondence to: Gabriela Hernández-Molina, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Department of Immunology and Rheumatology, Vasco de Quiroga 15, Col. Belisario Domínguez Sección XVI, CP 14080, Mexico City, Mexico. email: gabyhm@yahoo.com Competing interests: none declared. © Copyright CLINICAL AND EXPERIMENTAL RHEUMATOLOGY 2024.

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