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# LRA by ELISA/ACT®

## CLINICAL PEARLS UPDATE#32

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### *Sjogren's rhinitis sica*

July 2, 2004

Dear Colleague,

Our understanding of the fundamental roles of autoimmunity in **Sjogren's rhinitis sica** (dry eyes and mouth with hyposplenism) continues to grow. Prof. Feher and colleagues have contributed some of our more profound insights. The following studies point out that neuroimmunomodulation and lymphocyte infiltration occur early and are at the probable cause of Sjogren's syndrome. **This is consistent with the view that overload of the immune defense system in early life increases the risk of developing Sjogren's syndrome, particularly in people lacking sufficient antioxidant repair enabling nutrients. This is also consistent with the view that identifying and substituting for 'delayed allergens' can help reduce the complications of and improve the management of Sjogren's syndrome. This is what LRA by ELISA/ACT® tests and plans are designed to do, particularly for the T cells (type IV DTH) that only a functional lymphocyte response assay like LRA by ELISA/ACT can measure.**

We encourage you to share this valuable clinical update newsletter with your colleagues and staff so they can learn more about how our comprehensive approach can be applied to their practice with beneficial results. Please also let us know if any of your colleagues or staff would like to be added to our email distribution list.

We are grateful for the opportunities to be of service to you and your patients.

Sincerely,

***Russ Jaffe, MD, Ph.D., CCN, NACB***  
***Lab Director***

**Batbayar B, Nagy G, Kovesi G, Zelles T, Feher E. Morphological basis of sensory neuropathy and neuroimmunomodulation in minor salivary glands of patients with Sjogren's syndrome. *Arch Oral Biol* 2004;49(7):529-538.**

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**Objective:** A predominance of sensory neuropathy was earlier described in Sjogren's syndrome (SS), which might precede the presence of sicca symptoms. The mechanism of sensory neuropathy in SS is unknown. Therefore, the aim of this study was to determine the quantitative changes of the different neuropeptide containing nerve terminals and the immunocompetent cells in labial salivary glands of primary SS. **Design:** Immunohisto- and immunocytochemical methods were used for the detection of immunoreactive (IR) elements and the data were compared with the healthy controls. **Results:** All of the investigated IR nerve fibres were found in different quantity and localisation in both of control and SS glands. The density of them was changed variously in SS. The number of the substance P (SP), neuropeptide Y (NPY) ( [Formula: see text] ), galanin (GAL) IR nerve terminals was decreased, however, the number of vasoactive intestinal polypeptide (VIP) and tyrosine beta-hydroxylase (TH) IR nerve fibres ( [Formula: see text] ) was increased compared to the control. There were no IR immunocompetent cells in the control materials, however, a large number of them showed IR for SP (46.2%) and NPY (34.4%) in the SS. The IR was demonstrated mainly in the mast cells, plasma cells and some of the lymphocytes. **Conclusions:** These neuropeptides might have a role in the sensory neuropathy; they might activate nociceptive and sympathetic pathways. Some neuropeptides (SP, NPY) are endogenous in the immunosystem and produced in certain conditions, e.g. inflammation and chronic autoimmune disorders such as SS, so they might participate in the neuroimmunomodulation and contribute to the atrophy, apoptosis and necrosis.

**Masaki Y, Sugai S. Lymphoproliferative disorders in Sjogren's syndrome. *Autoimmun Rev* 2004;3(3):175-182.**

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Sjogren's syndrome (SS) is a chronic organ-specific autoimmune disease characterized by lymphocytic infiltration into the salivary and lacrimal glands. About half of primary SS patients develop systemic disorders. Primary SS can be divided into three stages according to the extent of organ damage and the course of the disease. In stage I, (approx. 45% of cases), patients have only sicca syndrome and do not experience any systemic involvement, even after 10 years. In stage II (approx. 50% of cases), patients experience lymphocytic organ damage, which may involve the pulmonary, renal, hepatic, hematologic, and/or dermatologic systems, among others. Finally, in stage III (approx. 5% of cases), patients develop malignant lymphomas. Lymphomas in salivary glands are thought to arise from lymphoepithelial lesions in which there are close interactions among epithelial cells, T cells, and B cells. The B cells in the lesions become activated through the interaction between CD40L and CD40. The progression from

polyclonal lymphoproliferation to monoclonal lymphoproliferation, to mucosa-associated lymphoid tissue (MALT) lymphoma, and finally to high-grade malignant lymphoma is regarded as a multi-step process. Antigenic activation of B cells, together with oncogenic events, including p53 inactivation and bcl-2 activation, may play important roles in B cell monoclonal proliferation and malignant transformation. The rheumatoid factor clone is regarded as a candidate B cell clone that undergoes transformation.