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LRA by ELISA/ACT®

CLINICAL PEARLS UPDATE#37

Asthma

August 13, 2004

Dear Colleague,

Autoimmune conditions often occur together. This is particularly true in the immune aspect of the human pneumatic system where **impaired dendritic cell function systemically, asthma, periodic antioxidant deficits with intermittent free radical bursts especially in the pulmonary walls, airway muscular status as a window on lung reactivity, and mineral status for nutritional and toxic metals form a clinically integrated way of managing these issues.** Mineral status can be checked using the d-penicillamine protocol. Repair deficits in distressed individuals may promote the concurrent autoimmunity and toxic mineral effects. The cumulative repair deficit is clinically often described as an inflammatory syndrome. Inflammatory markers increase. Examples are **hsCRP, mastocytosis, MAPK, fibrinogen, insulin and related growth factors, free cortisol, sed rate, and anti-collagen antibodies.** LRA by ELISA/ACT® tests and plans determine each individual's delayed allergic reactions or mystery hypersensitivities. Our LRA tests are **functional, ex vivo, and comprehensive assays.** Substitution for reactive items is blended into an alkaline way repair diet. Targeted supplementation aims to correct antioxidant deficits and enhance detoxification ability. Healing actions engage the mind and body in an integrated direction. Go by results. Several retest cycles will be routinely needed in complex cases. The sequential improvement in health quotient speaks clearly about how effective are LRA by ELISA/ACT interpretation plans in integrating health fundamentals.

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

Rottem M, Shoenfeld Y. Asthma as a paradigm for autoimmune disease. *Int Arch Allergy Immunol* 2003;132(3):210-214.

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Allergy and autoimmunity result from dysregulation of the immune system. Until recently, it was generally accepted that the mechanisms that govern these disease processes are quite disparate; however, new discoveries suggest possible common pathogenetic effector pathways. This review illustrates the concomitant presentation of these conditions and the potential relationship or common mechanisms in some cases, by looking at the key elements that regulate the immune response in both asthma and autoimmune conditions: mast cells, antibodies, T cells, cytokines, and genetic determinants. The parallel appearance of asthma and autoimmune conditions in the same patients may reveal that such aberrations of the immune system have a common pathophysiological mechanism. Mast cells, which play a key role in asthma, and the wealth of inflammatory mediators they express, make it likely that they have profound effects on many autoimmune processes. Activation of protein kinases by inflammatory cytokines and environmental stresses may contribute to both allergic and autoimmune diseases. The presence of autoantibodies in some allergic diseases suggests an autoimmune basis for these conditions. Because of the central role T cells play in immune reactivity, the T cell receptor loci have long been considered important candidates for a common disease susceptibility within the immune system such as asthma, atopy, and autoimmunity. Immunomodulation is the key to successful treatment of asthma and autoimmune conditions.

Johnson GL, Lapadat R. Mitogen-activated protein kinase pathways mediated by ERK, JNK, and p38 protein kinases. *Science* 2002;298(5600):1911-1912.

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Multicellular organisms have three well-characterized subfamilies of mitogen-activated protein kinases (MAPKs) that control a vast array of physiological processes. These enzymes are regulated by a characteristic phosphorelay system in which a series of three protein kinases phosphorylate and activate one another. The extracellular signal-regulated kinases (ERKs) function in the control of cell division, and inhibitors of these enzymes are being explored as anticancer agents. The c-Jun amino-terminal kinases (JNKs) are critical regulators of transcription, and JNK inhibitors may be effective in control of rheumatoid arthritis. The p38 MAPKs are activated by inflammatory cytokines and environmental stresses and may contribute to diseases like asthma and autoimmunity.