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# LRA by ELISA/ACT<sup>®</sup>

## CLINICAL PEARLS UPDATE#20

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### *Vitiligo*

February 10, 2004

Dear Colleague:

**Vitiligo** is depigmentation from melanocyte loss in the skin. Autoimmunity often plays an important role. Restoring immune repair tolerance can be helpful. **LRA by ELISA/ACT<sup>®</sup>** tests and plans are designed to determine the help that restoring immune repair resilience and tolerance can provide in vitiligo.

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***

Kemp EH, Waterman EA, Weetman AP. **Autoimmune aspects of vitiligo.** *Autoimmunity* 2001;34(1):65-77.

Vitiligo is a depigmenting disorder characterised by the loss of melanocytes from the cutaneous epidermis. Although the exact cause of the condition remains to be established, an autoimmune aetiology has been suggested and several observations support this theory. These will be the topic of discussion in this review. In brief, the disease is frequently associated with other disorders which have an autoimmune origin such as autoimmune thyroiditis and insulin-dependent diabetes mellitus. Furthermore, circulating antibodies and T lymphocytes which react against melanocyte antigens are present in the sera of a significant proportion of vitiligo patients compared with healthy individuals. Immunosuppressive therapies which are reasonably effective in treating the condition, well-studied animal models of the disease as well as the association of vitiligo with MHC antigens, all add credence to the hypothesis that immune mechanisms play a role in the development of vitiligo.

Zerubavel-Weiss R, Markovits D, Cohen IR. **Autoimmune thyroiditis (EAT) in genetically resistant mice mediated by a T cell line.** *J Autoimmun* 1992;5(5):617-627.

Experimental autoimmune thyroiditis (EAT) can be induced in genetically susceptible strains of mice by immunization to mouse thyroglobulin (Tg). EAT also can be produced by administration of anti-mouse Tg T cell lines and clones. Previously we were able to raise virulent anti-Tg T cell lines from mice genetically susceptible to EAT. These virulent lines, upon attenuation, were able to vaccinate the susceptible mice against EAT. We now report the isolation of a virulent T cell line from C57BL/6 mice genetically resistant to EAT. The T cell line and its clones recognize a Tg epitope cross-reactive between mouse and bovine Tg. Unexpectedly, the virulent anti-Tg line attenuated in various ways failed to vaccinate C57BL/6 mice against EAT mediated by the line itself. These results shed some light on the regulation of autoimmunity.

Das PK, van den Wijngaard RM, Wankowicz-Kalinska A, Le Poole IC. **A symbiotic concept of autoimmunity and tumour immunity: Lessons from vitiligo.** *Trends Immunol* 2001;22(3):130-136.

Vitiligo is a skin disease in which melanocytes (MCs) are eradicated from lesional epidermis, resulting in disfiguring loss of pigment. MCs are destroyed by MC-reactive T cells, as well as other non-immune and immune components. Similarities exist between the autoimmunity observed in vitiligo and the tumour immunity observed in melanoma immuno-surveillance. An analysis of these mechanisms might lead to the development of new therapies for both vitiligo and melanoma.

