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# LRA by ELISA/ACT®

## CLINICAL PEARLS UPDATE#29

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### *Silent maldigestion*

June 11, 2004

Dear Colleague,

**Asymptomatic or silent maldigestion** is, according to gastroenterologist Don Donaldson, endemic and epidemic in industrial society. Autoimmunity often plays an important role. Restoring immune repair tolerance can be helpful. **LRA by ELISA/ACT® tests and plans** are designed to determine the help by restoring immune repair resilience and tolerance in silent maldigestion. As the controlled study in dogs illustrates, immune reactivity as well as shift from tolerance to hypersensitivity often underlies silent maldigestion.

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**

**Westermarck E, Wiberg M. Exocrine pancreatic insufficiency in dogs. *Vet Clin North Am Small Anim Pract* 2003;33(5):1165-1179**

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Pancreatic acinar atrophy (PAA) is by far the most common cause for the maldigestion signs of canine exocrine pancreatic insufficiency (EPI). The ability to diagnose PAA in the subclinical phase before the development of total acinar atrophy and manifestation of clinical signs has offered new possibilities to study the pathogenesis of the disease. Marked T-lymphocyte infiltration during the progression of acinar atrophy and the genetic susceptibility of the disease have been taken as a primary evidence of the autoimmune nature of the disease. The term autoimmune-mediated atrophic lymphocytic pancreatitis is preferred to describe pathologic findings. A single abnormally, low serum canine trypsin-like immunoreactivity (cTLI) concentration (< 2.5 mg/L), in dogs with typical maldigestion signs has been shown to be highly diagnostic for clinical EPI and is found in dogs with end-stage PAA. Repeatedly subnormal cTLI values (2.5-5.0 micrograms/L) in dogs with no clinical signs of EPI are valuable markers of subclinical EPI and highly suggestive for partial PAA. The primary treatment of EPI is supplementing each meal with pancreatic enzymes. The long-term treatment response for the nonenteric-coated enzyme supplements has been found to be good in half of these dogs, but the response varied considerably.

**Stevens TR, Winrow VR, Blake DR, Rampton DS. Circulating antibodies to heat-shock protein 60 in Crohn's disease and ulcerative colitis. *Clin Exp Immunol* 1992;90(2):271-274.**

**Gastrointestinal Unit, London Hospital Medical College, UK.**

Heat-shock proteins (HSPs) are highly conserved immunogenic intracellular molecules that are induced by inflammatory mediators and may induce autoimmune phenomena in vivo. We have recently demonstrated the increased expression of HSP-60 in the colonocytes of patients with ulcerative colitis. To study further the role of HSP-60 in inflammatory bowel disease, we have now measured antibodies to recombinant mycobacterial HSP-65 (a member of the HSP-60 family) in patients with Crohn's disease, ulcerative colitis, healthy volunteers and, as disease controls, patients with confirmed bacterial diarrhoea. In comparison with healthy controls (n = 20; median level of 89 ELISA units; range 24-292), serum IgA HSP-60 antibodies were elevated in Crohn's disease (n = 21; 157; 57-364; P < 0.05) and active ulcerative colitis (n = 16; 188; 58-373; P < 0.01) but not bacterial diarrhoea (n = 10; 106; 51-285). Increased IgA HSP-60 antibody levels in patients with inflammatory bowel disease may occur as the result of HSP release from injured gut epithelium; alternatively, increased intestinal permeability could facilitate mucosal access of luminal antigens and the generation of cross-reactive anti-bacterial HSP antibodies.