

CLINICAL SPECIFICATIONS

PARIETAL CELL + ATPASE

Function:

Parietal cells, also known as, Oxyntic Cells, are stomach epithelium cells, which secrete gastric acid and intrinsic factor. ATPases are a class of enzymes that increase the rate of the decomposition of adenosine triphosphate (ATP) into adenosine diphosphate (ADP) and a free phosphate ion. This dephosphorylation reaction releases energy. ATPase harnesses this energy to drive other chemical reactions.

Antibodies Appear:

Gastric Autoimmunity^{1, 2, 3, 4} Chronic Atrophic Gastritis⁴ Pernicious Anemia⁶

Known Cross-Reactions: Kidney brush border,⁵ Helicobactor pylori lipopolysaccharide,⁷ Helicobactor pylori⁸

Clinical Significance:

Antibodies against Parietal Cell have been shown in autoimmune gastric disorders.^{2, 3, 4} Due to the role Parietal Cells play in the absorption of Vitamin B12, patients with Parietal Cell antibodies exhibit Vitamin B12 deficiency.⁵ A high prevalence of Parietal Cell antibodies and associated autoimmune gastric disease is present in Parietal Cell antibody-positive-type 1 diabetic patients.^{2, 3} Thus, type 1 diabetic patients should be screened for antibodies to Parietal Cells. Early detection of these antibodies and the subsequent iron deficiency anemia, pernicious anemia and/or atrophic gastritis, could reduce the morbidity in the type 1 diabetic population.³ In the majority of adult patients with autoimmune gastritis, parietal cells are the target of the autoimmune destruction, the pathogenesis of which utilizes ATPases.⁴

References:

- 1. Codina, et al. The α -Subunit of the Colonic H+,K+-ATPase Assembles with β 1-Na+,K+-ATPase in Kidney and Distal Colon. J Biol Chem, 1998; 273(14):7894-7899.
- 2. De Block, et al. The presence of thyrogastric antibodies in first degree relatives of type 1 diabetic patients is associated with age and proband antibody status. J Clin Endocrinol Metabolism, 2008; 84:4062-4067.
- 3. De Block, et al. High prevalence of manifestations of gastric autoimmunity in parietal cell antibody-positive type 1 (insulin-dependent) diabetic patients. J Clin Endocrinol Metab, 1999; 84:4062-4067.
- 4. Greenwood, et al. Autoimmune gastritis and parietal cell reactivity in two children with abnormal intestinal permeability. Eur J Pediatr, 2008; 167:917-925.
- 5. Ireton, et al. Human antibody against rat gastric parietal cells and kidney brush border. Clin Exp Immunol, 1971; 8:783-789.
- 6. Varis, et al. An appraisal of tests for severe atrophic gastritis in relatives of patients with pernicious anemia. Dig Dis Sci, 1979; 24(3):187-191.
- 7. Wang, et al. Host inflammatory response to infection. In: Mobley HLT, Mendz GI and Hazell SL, (eds.) *Helicobactor pylori*: physiology and genetics. Washington (DC): ASM Press; 2001.
- 8. Claeys, et al. The gastric H+,K+-ATPase is a major autoantigen in chronic *Helicobacter pylori* gastritis with body mucosa atrophy. Gastroenterology, 1998; 115:340–347.