

## What is the risk factor for gastric cancer in a Costa Rican?

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A research group from Costa Rican evaluated risk factors for gastric cancer in Costa Rican regions with contrasting gastric cancer incidence rates (GCIR). They found that although a pro-inflammatory cytokine genetic profile showed an increased risk for developing gastric cancer (GC), the characteristics of Helicobacter pylori (H. pylori) infection, in particular the status of cagA and vacA genotype distribution seemed to play a major role in GCIR variability in Costa Rica.

Costa Rica, one of the countries with the highest age-adjusted incidence and mortality rates for gastric cancer (GC), has regions with contrasting gastric cancer incidence rate (GCIR). Helicobacter pylori (*H. pylori*) is a Gram-negative microaerobic bacterium that persistently colonizes the human gastric mucosa. There is an increased GC risk in subjects infected with *H. pylori* strains, especially those co-expressing the cagA, vacA s1 and babA2 genes. Cytokine gene polymorphisms of the host, IL-1beta, IL-1RN and IL-10, in response to *H. pylori* infection, have been also associated with an increased risk for developing gastric cancer.

A research team led by Dr. Sergio A Con from Costa Rica evaluated the potential impact of *H. pylori* and/or host genetic factors on GCIR variability in Costa Rica. Their study will be published on January 14, 2009 in the *World Journal of Gastroenterology*.

In their study, 191 *H. pylori*-positive patients were classified into groups A (high GCIR, n = 101) and B (low GCIR, n = 90). Human DNA obtained from biopsy specimens was used in the determination of



polymorphisms of the genes coding for interleukin (IL)-1beta and IL-10 by PCRRFLP, and IL-1RN by PCR. *H. pylori* DNA extractions obtained from clinical isolates of 83 patients were used for PCR-based genotyping of *H. pylori* cagA, vacA and babA2.

They found that cytokine polymorphisms showed no association with GCIR variability. However, gastric atrophy, intestinal metaplasia and strains with different vacA genotypes in the same stomach (mixed strain infection) were more frequently found in group A than in group B, and cagA and vacA s1b were significantly associated with high GCIR (P = 0.026 and 0.041, respectively).

Their result indicated that although a pro-inflammatory cytokine genetic profile showed an increased risk for developing GC, the characteristics of *H. pylori* infection, in particular the status of cagA and vacA genotype distribution seemed to play a major role in GCIR variability in Costa Rica.

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