

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

January 21 2009

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 PCR, 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.

Source: World Journal of Gastroenterology

Citation: What is the risk factor for gastric cancer in a Costa Rican? (2009, January 21) retrieved 19 April 2024 from

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