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# Profile of glycated-hemoglobin, antioxidant vitamin and cytokine levels in pulmonary tuberculosis patients: A cross sectional study at Pulmonary Diseases Center Semarang City, Indonesia

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Review Article: Special Edition

# Purinergic signaling in schistosomal infection

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ABSTRACT

Human schistosomiasis is a chronic inflammatory disease caused by blood fluke worms belonging to the genus Schistosoma. Health metrics indicate that the disease is related to an elevated number of years lost-to-disability and years lost-to-life. Schistosomiasis is an intravascular disease that is related to a Th1 and Th2 immune response polarization, and the degree of polarization affects the outcome of the disease. The purinergic system is composed of adenosine and nucleotides acting as key messenger molecules. Moreover, nucleotide-transforming enzymes and cell-surface purinergic receptors are obligatory partners of this purinergic signaling. In mammalian cells, purinergic signaling modulates innate immune responses and inflammation among other functions; conversely purinergic signaling may also be modulated by inflammatory mediators. Moreover, schistosomes also express some enzymes of the purinergic system, and it is possible that worms modulate host purinergic signaling. Current data obtained in murine models of schistosomiasis support the notion that the host purinergic system is altered by the disease. The dysfunction of adenosine receptors, metabotropic P2Y and ionotropic $P2X_7$ receptors, and NTPDases likely contributes to disease morbidity.

## Schistosomiasis

Human schistosomiasis (or bilharzia) is a chronic inflammatory disease caused by blood fluke worms belonging to the genus Schistosoma. According to the World Health Organization (WHO), more than 200 million people worldwide suffer from chronic schistosomiasis and approximately 800 million people live in schistosomiasis-endemic areas. Schistosomiasis is therefore considered to be one of the world's most prevalent infectious diseases (http://www.who.int/schistosomiasis/en/, as of June 2016). This neglected tropical disease largely affects people living in poverty. The Disability-Adjusted Life-Year (DALY), a time-based measure used as a health metric, considers the number of years lost-to-disability

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Original Article

# Association of endothelial dysfunction and cytotoxin-associated gene A-positive *Helicobacter pylori* in patients with cardiac syndrome X

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ABSTRACT

*Background:* Existence of coronary endothelial dysfunction has been demonstrated in patients with cardiac syndrome X (CSX). In addition, *Helicobacter pylorus* (*H. pylori*) has been associated with CSX. We aimed to assess the possible association of endothelial dysfunction and cytotoxin-associated gene A-positive *H. pylori* (CagA+) infection in CSX patients.

*Methods:* Fifty-six patients with CSX (23 male/33 female; age: 51.25 ± 8.86 years) who were anti-*H. pylori* IgG-positive [*H. pylori*(+)] and 24 CSX patients (7 male/17 female; age: 52.79 ± 9.88 years) who were *H. pylori*(−) were included. Also, anti-*H. pylori* IgG-positive patients were determined by the presence of IgG antibody to CagA. Levels of endothelin-1 (ET-1), E-selectin and intercellular adhesion molecule-1 (ICAM-1) were measured.

*Results:* Endothelial dysfunction biomarkers were higher in *H. pylori*(+) than in *H. pylori*(−) patients (ET-1: 54.60 ± 25.39 vs. 42.59 ± 18.37 pg/ml, $p = 0.04$; E-selectin: 42.68 ± 14.26 vs. 31.72 ± 8.26 ng/ml, $p = 0.001$; ICAM-1: 339.68 ± 135.8 vs. 266.51 ± 125.1 ng/ml, $p = 0.02$). Among *H. pylori*(+) subjects, 28 cases were CagA(+) and 28 cases were CagA(−). There were significant differences in measured levels of E-selectin between CagA(+) and CagA(−) groups (48.00 ± 16.37 vs. 37.37 ± 9.37 ng/ml, $p = 0.004$). For ET-1 and ICAM-1 levels, the difference between CagA(+) and CagA(−) was insignificant ($p = 0.174$ and $p = 0.07$, respectively).

*Conclusion:* High levels of endothelial dysfunction biomarkers are found in CSX patients with anti-CagA(+). These findings suggest the infection with CagA(+) *H. pylori* strain may play a role as a risk factor in development of CSX through provocation of endothelial dysfunction. Therefore, a long term follow up to investigate the outcomes of these patients is proposed.

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Original Article

# Elevated adiponectin but varied response in circulating leptin levels to *falciparum* malaria in type 2 diabetics and non-diabetic controls


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ABSTRACT

*Background:* To investigate effects of *falciparum* malaria on circulating levels of leptin and adiponectin in type 2 diabetes mellitus (T2DM) and non-diabetic controls in relation to measures of adiposity.

*Methods:* Levels of leptin and adiponectin were measured in 100 type 2 diabetics and 100 age-matched controls before and during *falciparum* malaria in a 2-year prospective study. Also, waist circumference (WC), weight, height and hip circumference were measured. Body mass index (BMI) and waist-to-hip ratio (WHR) were computed.

*Results:* At baseline, diabetics had significantly ($p < 0.05$) higher WC and BMI but lower WHR, leptin and adiponectin levels. Baseline leptin correlated positively with WC ($r = 0.633$; $p < 0.001$) and BMI ($r = 0.63$; $p < 0.001$) in diabetics but only BMI (0.562; $p < 0.001$) in non-diabetic controls. Baseline leptin and adiponectin correlated positively ($r = 0.249$; $p = 0.029$) in non-diabetic respondents only. Adiponectin correlated negatively with WC ($r = -0.58$; $p = 0.006$) in diabetic males only. During malaria, mean levels of leptin and adiponectin were comparable ($p > 0.05$) between diabetics and controls. However, compared to baseline levels, significant ($p < 0.001$) elevation of adiponectin was found in both study groups. In respect of leptin, significant ($p < 0.001$) rise but decline was observed in diabetics and controls respectively. Malaria-induced leptin correlated negatively with adiponectin ($r = -0.694$; $p < 0.001$) in non-diabetic controls only.

*Conclusion:* Diabetics and controls exhibited increased adiponectin levels due to *falciparum* malaria but differed in response in terms of leptin levels.

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