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Key words: cardiovascular prevention, *Helicobacter pylori*, ischemic heart disease.

Dear Sir,

A recent issue of Panminerva Medica, dedicated to cardiovascular prevention and rehabilitation, included several reviews dedicated to classical risk factors for cardiovascular diseases.[1-3]

We would like to highlight another potential risk factor largely investigated in the last decades: *Helicobacter pylori* (*H. pylori*) infection.[4]

H. pylori infection is a chronic one, acquired during childhood as a rule and involved in the pathogenesis of several gastroduodenal diseases including peptic ulcer, precancerous lesions, gastric adeno-carcinoma and mucosa-associated lymphoid tissue lymphoma. Moreover, a potential role of *H. pylori* has been investigated in several extra-gastric manifestations,[5] including ischemic cardiovascular diseases (IHD). Regarding this topic, a number of studies have reported conflicting results. In fact, numerous discordances due to the lack of homogeneity of investigations emerged. There was a high degree of heterogeneity in the definition of IHD, the control groups were biased, hence allowing for large variations in the adjustment for potential confounding factors. From a clinical point of view, the main heterogeneity stemmed from differences among cardiovascular diseases included in the studies: acute myocardial infarction, chronic IHD, angina, abnormalities in ECG, or total mortality due to any cardiovascular cause.[4,5] A crucial question deals with possible pathogenic mechanisms through with the bacterium could be involved in IHD. *H. pylori* genomic material has been detected in the plaque only in sporadic occasions. Therefore, the hypothesis of an indirect pathway has been raised. Several mechanisms of damage have been proposed. Indeed, the long-term inflammation generated by *H. pylori* might raise cytokine levels in the bloodstream, and consequently, activate fibroblast and smooth muscle cell proliferation. Furthermore, it is possible that the lipidic profile or fibrinogen levels, as well as circulating markers of inflammation related to artery atherosclerosis, could be influenced by the bacterium.[4] It has been also proposed that *H. pylori* binds von Willebrand factor and interacts with glycoprotein Ib to induce platelet aggregation mediated by cytokines, such as interleukin (IL)-1, IL-6 and TNF (tumor necrosis factor)-α.[5] However, the exact mechanism remains unclear.

Because IHD is the outcome of a multiplicity of steps and risk factors, many of which with only a limited individual effect, complete understanding of causation is difficult. Nevertheless, the study of infectious agents represents a promising direction for future studies beyond the traditional risk factors.

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Conflicts of interest. The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the letter.

Funding. The authors report no involvement in the research by the sponsor that could have influenced the outcome of this work.

Authors' contributions. Author MD has conceptualized the manuscript and revised it critically, author AF has drafted the manuscript. All authors read and approved the final version of the manuscript.