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# Helicobacter pylori promotes angiogenesis in gastric cancer cells depending on cyclooxygenase-2-mediated vascular endothelial growth factor via p38MAPK/ATF-2 pathway

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Angiogenesis, the growth of new blood vessels, is closely related with the incidence and development of gastric cancer, but the pathogenesis of angiogenesis is still unknown in *Helicobacter pylori* (*H. pylori*) -induced gastric cancer. Previously, we reported that *H. pylori* could increase the expression of COX-2 via p38MAPK in vitro. In this study, we established a mice model of *H. pylori* infection to define the exact role of *H. pylori* infection in gastric carcinogenesis. Microvessel density (MVD) and Vascular endothelial growth factor (VEGF) mRNA expression in gastric mucosa were significantly higher in *H. pylori* infected mice than that in untreated mice after 72 weeks. Further analysis revealed that *H. pylori* infection induced VEGF through COX-2 gene by the activation of p38MAPK. Thus, inhibition of either COX-2 or p38MAPK suppressed *H. pylori* infection induced VEGF at mRNA and protein level. In conclusion, our study has provided the first direct evidence that *Helicobacter pylori* induces C57BL/6 mice gastric adenocarcinoma and enhances VEGF expression via p38MAPK /COX-2 pathway.

## Biography

Qi Li has completed his Ph.D and MD.at Shanghai University of Traditional Chinese Medicine and postdoctoral studies from Cornell University. He is the director of Laboratorial Center and the chief of Research Branch at Putuo Hospital in Shanghai. He has published more than 50 papers in reputed journals, including 5 papers in SCI, and serving as an editorial board member of Tumor, Shanghai Traditional Chinese Medicine, and so on.