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The role and mechanism of A20 in *Helicobacter pylori*-related gastritis

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Statement of the Problem: Helicobacter pylori (*H.pylori*) infection is closely with the human gastric mucosa-associated diseases. Recent several studies on miRNAs have expanded our insights on *H.pylori* pathogenesis.

Findings: In this study, we found that miR-29a-3p was upregulated in *H.pylori*-positive gastric mucosa tissues and *H.pylori*-infected gastric epithelial-derived cell lines. The upregulation of miR-29a-3p was dose dependent in BGC-823 and GES-1 cells infected with *H.pylori*.A20 (also known as tumor necrosis factor-α-induced protein 3,TNFAIP3) is involved in NF-κB signaling pathway and cell proliferation. A20 was identified as target gene of miR-29a-3p.The expression of A20 was decreased in *H.pylori*-positive gastric mucosa tissues. A20 downregulation was time- and dose-dependent in gastric epithelial-derived cell lines infected with *H.pylori*.

Conclusion & Significance: Overexpression of miR-29a-3p with miR-29a-3p mimic significantly blocked *H.pylori*-induced A20 expression, which suggests that *H.pylori* decreased A20 expression through upregulating miR-29a-3p in gastric epithelial-derived cell lines infected with *H.pylori*. Functional findings showed that overexpression of A20 enhanced phosphorylated NF-κB p65 expression, promoted nuclear translocation of NF-κB p65 and the expression of inflammatory cytokines. Conversely, inhibition of A20 led to reduce the NF-κB p65 activation and the expression of inflammatory cytokines. In addition, compared to *H.pylori*infection group, IL-6 and IL-8 were decreased after silencing A20 and then infected *H.pylori*, which suggests silencing A20 can inhibit *H.pylori* induced inflammation. Together, the results suggest a new role of A20 in *H.pylori* infection and indicate it may positively regulate NF-κB activity and promote inflammation. The down expression of A20 may protect the body from damage in face of *H.pylori* infection.

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