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Novel insights into the mechanism and consequences of this phenomenon have emerged, but many

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Manuscript NO: 64402

Manuscript Type: REVIEW

Role of autophagy in gastric carcinogenesis

Autophagy in GC

Abstract

Gastric cancer (GC) represents a common and highly fatal malignancy and thus, a pathophysiology-based reconsideration is necessary, given the absence of efficient therapeutic regimens. In this regard, emerging data reveal a significant role of autophagy in gastric oncogenesis, progression, metastasis and chemoresistance. Although autophagy comprises a normal primordial process, ensuring cellular homeostasis under energy depletion and stress conditions, alterations at any stage of the complex regulatory system could stimulate a tumor-genic and -promoting cascade.

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Role of autophagy in gastric carcinogenesis



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*Implications of H. pylori altered autophagy in gastric carcinogenesis. The role of autophagy in H. pylori infection is complex and may serve different functions at different stages of infection. Initially autophagy may serve as a **host defense to eliminate invading bacteria.***

Author: Laura K. Greenfield, Nicola L. Jones

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Novel insights into the mechanism and consequences of this phenomenon have emerged, but many aspects remain largely unknown. Current evidence supports a scenario in which *H. pylori*-suppressed autophagy facilitates intracellular survival and persistence of the pathogen, while also generating an environment favoring carcinogenesis.

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However, upon delivery into gastric epithelial cells, CagA is usually degraded by macroautophagy/autophagy. Hence, the induction of autophagy in *H. pylori*-infected epithelial cells is an important host-protective ability against gastric carcinogenesis. However, the mechanisms by which autophagosome-lysosome fusion is regulated, are unknown.

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