

Significance of Nerve Growth Factor in Angiogenesis Ageing

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DESCRIPTION

The stomach is constantly subjected to a wide range of intrinsic and exogenous stressors. These factors can include luminal irritants, *Helicobacter pylori* infection, and ingested therapeutics such as non-steroidal anti-inflammatory drugs. These insults to the gastric tissue can cause gastric, or peptic, ulcers that may develop into chronic or recurring ulcers. More than 5 million people in the United States alone suffer from gastrointestinal ulcers, with approximately 500,000 new cases reported each year.

Fortunately, the general population's incidence of gastric ulcers is decreasing; however, gastric ulcer hospitalization and mortality remain high in the elderly. This is due, in part, to the aged stomach's increased susceptibility to injury and delayed healing.

The elderly stomach has been shown to have decreased gastric acid secretion, motility, and proliferation, which likely results in an increased susceptibility to chronic ulceration, which can be adversely affected by chronic insults such as *H. pylori* infection or the administration of non-steroidal anti-inflammatory drugs. The number of people aged 65 and up in the United States is expected to more than double by 2050, reaching 84 million. Despite the prevalence of gastric ulcers, the ulcer healing process remains unknown. Furthermore, because elderly patients exhibit gastric pathology, a better understanding of repair within the aged stomach is critical. As a result, producing methods to lower ulcer incidence or accelerate healing is an important goal for gastric research. Previous research has described the intricate process of gastric wound repair, which includes immune cell infiltration, cell proliferation, re-epithelialization, angiogenesis, and tissue remodeling. Although growth factors are known to play an important role in gastric epithelial regeneration, the mechanism is not well understood. The new study reported that

it improved understanding of gastric regeneration by elucidating the role of Nerve Growth Factor (NGF) expressed in gastric endothelial cells during the stomach repair process. In an *in vitro* culture of gastric endothelial cells isolated from aged rats versus gastric endothelial cells isolated from young rats, the researchers observed decreased expression of NGF, which correlated with decreased angiogenesis. It demonstrated increased *in vitro* angiogenesis in ageing gastric endothelial cells via NGF gene therapy.

The researchers also used inhibitors to demonstrate that this response was mediated by the phosphatidylinositol 3 kinase/protein kinase B and mammalian target of rapamycin signaling pathways. This study also demonstrated that silencing serum response factor inhibited NGF-induced angiogenesis in ageing gastric endothelial cells *in vitro*. *In vivo* studies using acetic acid to induce focal gastric injury in young and old rats revealed delayed epithelial wound repair as a result of decreased angiogenesis in the elderly. Exogenous NGF increased angiogenesis, accelerated repair, and improved mucosal regeneration in aged rats' gastric tissue.

The analysis of human gastric biopsy specimens corresponded to experimental results showing that people over the age of 70 had lower NGF expression in gastric endothelial cells than people under the age of 40. When *in vitro* and *in vivo* experiments are combined, they show that decreased expression of NGF in gastric mucosal endothelial cells in ageing gastric tissue results in impaired reparative processes. Exogenous NGF expression effectively reversed angiogenesis and healing impairment. The researchers discovered a novel role for NGF in ulcer healing, with particular relevance in ageing gastric mucosa. Furthermore, suggested a potential therapeutic target for the treatment of gastrointestinal mucosal injuries.

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