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Evaluation Parameters in Ethanol-Induced Gastric Ulcer Models: Macroscopic to Molecular Endpoints

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ABSTRACT

Ethanol-induced gastric ulcer models in rats are widely used in preclinical research to understand gastric mucosal injury and to evaluate gastroprotective interventions. The validity of this experimental model relies on comprehensive evaluation parameters that capture injury progression from visible tissue damage to intracellular signalling events. Macroscopic assessment remains the primary screening approach and includes measurement of ulcer index, lesion number, haemorrhagic streaks, and gastric wall oedema. These gross observations are complemented by histopathological examination, which provides detailed insight into epithelial erosion, inflammatory cell infiltration, mucosal necrosis, and submucosal disruption. At the biochemical level, ethanol exposure induces marked oxidative stress, characterised by increased lipid peroxidation and depletion of endogenous antioxidants, including superoxide dismutase, catalase, and reduced glutathione. Alterations in gastric mucus content, prostaglandin levels, and gastric acidity further reflect impaired mucosal defence mechanisms. Advances in experimental techniques have enabled molecular evaluation of ethanol-induced ulcers, including analysis of pro-inflammatory cytokines, apoptotic regulators, tight junction proteins, and redox-sensitive signalling pathways. These molecular endpoints enhance the mechanistic understanding of ulcerogenesis and facilitate precise assessment of therapeutic efficacy. Collectively, integration of macroscopic, histological, biochemical, and molecular parameters provides a robust framework for evaluating ethanol-induced gastric ulcer models and strengthens their translational relevance in anti-ulcer drug discovery.

Keywords: *Ethanol-induced gastric ulcer, Rat model, Ulcer index, Oxidative stress, Histopathology*

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