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N-STEAROYLETHANOLAMINE (NSE) MODULATES SURVIVAL, PROLIFERATION, AND MIGRATION OF LEWIS LUNG CARCINOMA CELLS (LLC) AND REGULATES PROINFLAMMATORY CYTOKINE LEVELS IN VITRO UNDER STRESS CONDITIONS

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Introduction. The cell response to stress involves modulation of cellular functions and changes in the microenvironment, accompanied by the development of inflammatory reactions. Under these conditions, cells acquire characteristics typical of malignant neoplasms, such as enhanced proliferation, increased survival, and metastatic potential, ultimately driving tumor progression. A promising avenue for addressing this challenge lies in biologically active lipids, cannabimimetics, which exhibit notable anti-inflammatory and cytoprotective properties.

Aim. To examine the biological responses of LLC cells subjected to LPS-induced stress in vitro.

Methods. Stress was induced using 5 μg/ml lipopolysaccharide (LPS). Metabolic activity was assessed by the MTT assay after 24 h, following 24 h of LPS preincubation. Proliferative activity was evaluated by direct cell counting. Cell migration was measured using the scratch assay. Proinflammatory cytokines in the conditioned medium were assessed by ELISA.

Results and Discussion. NSE at concentrations of 10^{-6} and 10^{-7} M significantly suppressed the metabolic activity, proliferation, and migration of LLC cells compared to the control, both under normal conditions and under LPS-induced stress. NSE also modulated proinflammatory cytokine production, reducing IL-6 levels and influencing TNF-α expression depending on incubation conditions: a) NSE reduced IL-6 in the conditioned medium both in LPS-treated cells and following NSE preincubation; b) NSE pretreatment increased TNF-α levels under LPS stress, whereas preincubation with 10^{-6} M NSE prevented the LPS-induced rise in TNF-α; c) In samples without stress induction, NSE reduced IL-6 content in the conditioned medium by approximately 50%.

Conclusions. The findings demonstrate that NSE exhibits antitumor and protective activity under LPS-induced stress in vitro. Specifically, NSE inhibits cellular metabolism, suppresses proliferation, and slows migration. In addition, NSE modulates the production of proinflammatory cytokines, reducing IL-6 levels and influencing TNF-α expression depending on incubation conditions. Collectively, these effects highlight the biological significance of NSE and support its potential as a basis for developing novel antitumor strategies. By targeting cellular regulatory networks, NSE may serve as an effective tool for the prevention and treatment of stress-associated malignant neoplasms.

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