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Apigenin Ameliorated Alcohol-induced by Peripheral Neuropathy in Experimental Rats: a Modulatory Effect Against Oxidative, Inflammatory and Apoptotic Pathways

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SUMMARY. **Chronic alcohol consumption caused various clinicopathological changes resulting in peripheral polyneuropathy. Apigenin is bioactive structures and biological activities, including anti-inflammatory, antioxidant, and neuroprotective potential. The aim was to assess the potential of apigenin against ethanol-induced by alcoholic neuropathy. Sprague-Dawley rats (180–220 g) received ethanol (10 gm/kg, 35% v/v, b.i.d.) for 10 weeks to induce alcoholic neuropathy, followed by concomitant administration of either apigenin (5, 10, or 20 mg/kg) or vehicle or alpha-tocopherol (100 mg/kg). Various behavioral, biochemical, and molecular changes were evaluated. Administration of apigenin at a dose of 10 and 20 mg/kg effectively ($p < 0.05$) attenuated alcohol-induced decreased thermal and mechanical hyperalgesia, mechano-tactile allodynia, nerve conduction velocities and increased sciatic nerve oxidative stress and myeloperoxidase levels. Apigenin also significantly ($p < 0.05$) attenuated ethanol-induced elevated inflammatory biomarkers (TNF-α, IL-1β, IL-6, IL-4, IL-12, and INF-γ) and apoptosis levels (Bax, Bax: Bcl2, and Caspase-3 mRNA expressions). Apigenin exerts its potential against ethanol-induced alcoholic neuropathy** *via* **its antioxidant (decreased myeloperoxidase, malondialdehyde, nitric oxide levels, increased glutathione, and superoxide dismutase levels), anti-inflammatory (TNF-α, IL-1β, IL-6, IL-4, IL-12, and INF-γ) and antiapoptotic (Bax, Bax: Bcl2, and Caspase-3) potential.**

RESUMEN. El consumo crónico de alcohol provocó diversos cambios clínico-patológicos que derivaron en polineuropatía periférica. La apigenina es estructuras bioactivas y actividades biológicas, incluido el potencial antiinflamatorio, antioxidante y neuroprotector. El objetivo fue evaluar el potencial de la apigenina frente a la neuropatía alcohólica inducida por etanol. Ratas Sprague-Dawley (180-220 g) recibieron etanol (10 g/kg, 35 % v/v, dos veces al día) durante 10 semanas para inducir neuropatía alcohólica, seguido de la administración concomitante de apigenina (5, 10 o 20 mg/día). kg) o vehículo o alfa-tocoferol (100 mg/kg). Se evaluaron varios cambios conductuales, bioquímicos y moleculares. La administración de apigenina a dosis de 10 y 20 mg/kg de manera eficaz ($p < 0,05$) atenuó la disminución de la hiperalgesia térmica y mecánica inducida por el alcohol, la alodinia mecano-táctil, las velocidades de conducción nerviosa y el aumento del estrés oxidativo del nervio ciático y los niveles de mieloperoxidasa. La apigenina también atenuó significativamente ($p < 0,05$) los biomarcadores inflamatorios elevados inducidos por etanol (TNF-α, IL-1β, IL-6, IL-4, IL-12 e INF-γ) y los niveles de apoptosis (Bax, Bax: Bcl2 , y expresiones de ARNm de caspasa-3). La apigenina ejerce su potencial contra la neuropatía alcohólica inducida por etanol a través de su acción antioxidante (disminución de los niveles de mieloperoxidasa, malondialdehído y óxido nítrico, aumento de los niveles de glutatión y superóxido dismutasa), antiinflamatoria (TNF-α, IL-1β, IL-6, IL- 4, IL-12 e INF-γ) y potencial antiapoptótico (Bax, Bax: Bcl2 y Caspasa-3).

KEY WORDS: alcoholic neuropathy, apigenin, apoptosis, inflammation, nerve conduction velocity.

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