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Protective effect of pentoxifilline and their potentiation with nitric oxide modulator in complete Freund's adjuvant induced arthritic changes in rats

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Interaction of pentoxifylline with nitric oxide modulators was studied in complete Freund's adjuvant induced rheumatoid arthritis in rats. Wistar rats (200-300 gm, n=6/group) of either sex were used in the study. On day '0' rats were injected with 0.2 ml of complete Freund's adjuvant (CFA) in sub-planter region of right hind paw along with 0.1 ml of squalene to develop RA while controls received only vehicle. Pentoxifylline treatment alone and in combination with nitric oxide modulators was given from day '14' to '28'. Arthritic parameters (1) arthritis index, (2) ankle diameter, (3) paw volume and (4) their body weight were noted to evaluate progression of RA on day 0, 7, 14, 21 and 28. On day '28' rats were sacrificed and their blood was collected for TNF-α and IL-10 cytokine estimation. Data obtained was analysed using one way ANOVA followed by Newman-Keul's posthoc test and $p<0.05$ was considered for significance. It was found that CFA significantly increased arthritis-index, paw volume, ankle diameter and serum TNF-α levels while body weight and serum IL-10 levels was significantly decreased ($P<0.05$). These CFA-induced changes were significantly reversed by pentoxifylline 5 & 10 mg/kg alone and in combination with L-arginine (100 mg/kg) and L-NAME (10 mg/kg) in dose dependent manner ($p<0.005$) in all parameters). The maximum protective effects was observed in pentoxifylline 10 mg/kg + L-NAME 10 mg/kg combination treatment group ($p<0.001$). Results of this study are suggestive of the involvement & interaction of nitric oxide with pentoxifylline which may have a protective role in adjuvant induced RA.

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Fatty acids rehabilitated long-term neurodegenerative-like symptoms in olfactory bulbectomized rats

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Our previous study demonstrated that an olfactory bulbectomy in rats induced devastating short-term, multifaceted, neurodegenerative-like effects, which included cognitive impairment, hyperactivity, hyperthermia, and increased levels of homocysteine and pro-inflammatory cytokines, including interleukin-17A. In addition, the rats exhibited increase in the hyperphosphorylation of brain Tau proteins and the number of neurofibrillary tangles. In this study, we examined the long-term effects of the surgery and found that olfactory bulbectomy decreased cognitive function and increased levels of homocysteine, stress, and inflammatory cytokines. It also rendered the rats anemic, with brain iron overload. A significant reduction of the membrane fluidity index in frontal cortex synaptosomes was also observed. Treatment with a mixture of n-3 and n-6 fatty acids improved the unwanted effects. The treated rats exhibited improved learning capacity, and the increased levels of homocysteine and cytokines were significantly reduced. Their iron status also improved. The beneficial effects of fatty acids are mediated through their effects on the structure and fluidity of the neuronal membrane. Olfactory bulbectomy may serve as a useful model for neurodegenerative disorders.

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