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Manage your PC activities with Paragon.. Paragon Hard Disk Manager 17 is the most useful tool for. Protect files, folders, and devices in an easy-to-use. the increase in the IL-1 β production after the stimulation with LPS, which is important to study, but the antibodies that we used can recognize also endogenous IL-1 β . Antibodies have been chosen on the basis of their specificity to this cytokine and not on their inhibitory function. In addition to the cytokine production, NO production was also significantly increased upon LPS stimulation. To investigate the effect of rTNF on the NO production, the latter cytokine was added to the astrocyte cultures and the NO production was measured after 24 h. The NO production was increased in the presence of rTNF. This increased production was NO-dose dependent, suggesting that the effect is mediated by the receptor. NO can contribute to the pathogenesis of major depressive disorder, influencing survival and endothelial differentiation \[@CR39\]. NO production in the hippocampus is implicated in the pathophysiology of epilepsy \[@CR40\] and is increased in patients with bipolar disorder \[@CR41\]. On the other hand, exogenous NO has antidepressant-like effects in mice and rats \[@CR42\]-\[@CR44\]. No previous studies have investigated NO production in astrocytes isolated from chronic stressed animals. Astrocytes are involved in the elimination of inflammation by secreting cytokines and growth factors. Pro-inflammatory cytokines can activate the brain microglia and decrease their production of anti-inflammatory cytokines \[@CR45\]. Microglia is often considered the first line of defense in the central nervous system. After the activation of microglia, they produce NO and release pro-inflammatory cytokines, such as TNF α , IL-1 β and IL-6 and this can promote the activation and proliferation of astrocytes in neuroinflammatory diseases \[@CR25\]. In our study, the coculture of LPS-stimulated microglia with astrocytes produced an increase in IL-1 β and TNF α production. This could activate the production of NO and pro-inflammatory cytokines by astrocytes, increasing their proliferation and glial scar formation. TNF α is considered a critical factor in neuronal injury during ischemic disease

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