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Cultured Primary Neurons Inflammatory Responsesto Fibrinogen and the Role of Nuclear Factor-kappa B

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DESCRIPTION

Traumatic Brain Injury (TBI), an inflammatory disorder, is linked to neurodegeneration and a breached blood-brain barrier (BBB). One of the effects of inflammation is an increase in the protein fibrinogen (Fg), which is largely produced in the liver. The BBB is altered by inflammation, which increases the chance that Fg may interact with neurons by extravasating into the brain's parenchyma. In the past, we've shown that connections between the neuronal intercellular adhesion molecule 1 and the Fg and cell prion protein increased oxidative damage, pro-inflammatory cytokines, apoptosis, and cell death. However, the transcription route implicated in this process was not identified. In primary cultured mouse cortical neurons.

The expression of NF-B protein and the Fg-induced genes CCL2 and IL-6 both increased in response to a particular interaction between Fg and neurons. These findings suggest that extravasated Fg directly interacts with neurons during TBI-induced neurodegeneration, activating the transcription factor NF-B and increasing the expression of pro-inflammatory cytokines. This interaction may be a mechanism for neuro-inflammatory diseases that result in vascular cognitive impairment.

Traumatic Brain Injury (TBI) is one of the primary causes of morbidity and mortality in the US. It is a neurodegenerative inflammatory illness. Unintentional falls and car accidents are the most frequent injuries that necessitate hospitalization for TBI. A single fall could have a negative, fatal, or even life-changing impact on these patients. An additional injury, such as neurodegeneration and cognitive decline, may result from a TBI's immediate physical harm and inflammation, which may last for weeks, months, or even years.

Some TBI patients may get post-concussion syndrome over time, which is also referred to as cognitive impairment, pain, sleep difficulties, and physical handicap. Blood-Brain Barrier (BBB) disruption is known to begin early in TBI, although it may persist for many years after the initial damage.

This research proved that Fg stimulated neurons in a dose-dependent way. Additionally, our results showed that Fg has a specific effect on NF-B activation as evidenced by the reduction of Fg-induced NF-B activation by blocking its recognised receptors (ICAM-1 and PrPC). This study showed for the first time that inhibition of ICAM-1 and PrPC neuronal receptors decreased Fg-induced NF-B activation and the subsequent generation of pro-inflammatory cytokines. As a result, this study advises against using Fg receptor blockers or NF-B-specific. Studying the role of Fg-induced NF-B activation during TBI may open up new therapeutic avenues or provide light on the mechanisms of action of drugs with the potential to treat neuro-inflammatory illnesses by preventing neurodegeneration and the resulting memory loss.

In particular, traumatic Brain Injury (TBI), which is connected to persistent inflammation, the buildup of Fg in the brain parenchyma, and accelerated neurodegeneration that causes memory loss, could be treated with ICAM-1 and PrPC. These findings point to potential explanations for the observed effects, which originate in the vascular system (vascular permeability, Fg extravasation), cause neuro-inflammation (represented by the expression of CCL-2 and IL-6), and result in memory loss that has previously been linked to traumatic brain injury (TBI) and is referred to as vascular cognitive impairment and dementia.

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