Long-term administration of melatonin attenuates neuroinflammation in the aged mouse brain

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Abstract

Aging is often accompanied by a decline in cognitive function in conjunction with a variety of neurobiological changes, including neuroinflammation. Melatonin is a key endogenous indoleamine secreted by the pineal gland that plays a crucial role in the regulation of circadian rhythms, is a potent free radical scavenger, has anti-inflammatory activity and serves numerous other functions. However, the role of melatonin in sterile inflammation in the brain has not been fully investigated. In the present study, we investigated the neuroinflammation status in aged mouse brains. The results showed that the protein levels of integrin α M (CD11b), glial fibrillary acidic protein (GFAP), the major pro-inflammatory cytokines (interleukin-1 beta [IL-1 β], interleukin-6 [IL-6], and tumor necrosis factor alpha [TNF- α]) and phosphor-nuclear factor kappa B (pNF κ B) were significantly increased, while N-methyl-D-aspartate (NMDA) receptor subunits NR2A and NR2B, Ca^{2+}/calmodulin-dependent protein kinase II (CaMKII), and brain-derived neurotrophic factor (BDNF) were down-regulated in the hippocampus and prefrontal cortex (PFC) of 22-months-old (aged) mice compared with 2-months-old (young adult) mice. Melatonin was administered in the drinking water to a cohort of the aged mice at a dose of 10 mg/kg/day, beginning at an age of 16 months for 6 months. Our results revealed that melatonin significantly attenuated the alterations in these protein levels. The present study suggests an advantageous role for melatonin in anti-inflammation, and this may lead to the prevention of memory impairment in aging.

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