

Chemokines in the Central Nervous System and Alzheimer's Disease

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Introduction

Alzheimer's disease is a progressive neurologic disorder that cause the brain to shrink (atrophy) and brain cells to die. It is the most common cause of dementia. A continuous decline in thinking, Behavioral and social skills that affects a person's ability to task independently. The person with Alzheimer's disease will develop strict memory impairment and lose the ability to carry out everyday tasks. Medications may temporarily improve or slow progression of symptoms. These treatments can sometimes help people with Alzheimer's disease maximize function and maintain independence for a time. Different programs and services can help support people with Alzheimer's disease and their caregivers. There is no treatment that cures Alzheimer's disease or alters the disease process in the brain. In advanced stages of the disease, complications from severe loss of brain function – such as dehydration, malnutrition or infection and usually results in death.

Chemokines

The chemokines and receptors found in the central nervous system (CNS), their changes in alzheimer's disease (AD) brains, the direct effect of some chemokines on neuronal signaling pathways, a hypothesis regarding the pathophysiological role of chemokines within the CNS particularly in AD, and the way this delicate balance are often achieved or disrupted under physiological or pathological conditions and eventually , the implications of this hypothesis for future therapeutic intervention. Chemokines and their receptors detectable in brain tissues or neuronal and glial cultures under pathophysiological conditions by immunohistochemistry, by in place or by reverse transcriptase-polymerase chain reaction (RT-PCR) are summarized. Immunoreactivity for a number of chemokine receptors has also been detected in CNS cells. In AD brain, upregulation of CXCR2 expression was observed on dystrophic neurites of senile plaques. CC chemokine receptors CCR3 and CCR5 are found on microglia of both normal and AD brains and their expression is increased on some reactive microglia in AD. The present data support that chemokine expression in vivo is more restricted to particular cell types and is under fine regulation despite the very fact under in vitro conditions many cell types have the capacity to express chemokines. The detection of the many chemokines and their receptors on various resident cells of CNS suggest their physiological role within the CNS. Given the first role of chemokines as inflammatory mediators within the body's defence system , the upregulation of the many chemokines and their receptors in AD brain implies that localized inflammation is important in the disease pathogenesis. There could also be detrimental effects on neurons if chemokine expression is continuously triggered during a nonregulated fashion. Animal models are available to test these hypotheses. For potential therapeutic intervention through the chemokine system in AD, the double-edged role of chemokines suggests that pharmacological modulation of the system instead of complete blockade may help the body regain a state of homeostasis.