PERSPECTIVE

Role of cystatin C in alzheimer's disease

Shawn Kruger

Kruger S. Role of cystatin C in alzheimer's disease.J Clin Psych Neurosci.2022; 5(6):70-1.

ABSTRACT

A function for CysC in these situations is supported by changes in *cystatin* C (CysC) expression and secretion levels in the brain associated with a variety of neurological illnesses and in animal models of neurodegeneration. Alzheimer's disease risk is elevated by a variation in the CysC *gene* (CST3) (AD). The pathogenesis of AD is characterised by neurodegeneration, neurofibrillary tangles made primarily of hyperphosphorylated tau, and the deposition of oligomeric and fibrillar forms of Amyloid (A) in the walls of the cerebral artery and neuropil. *CysC's* co-localization with A in amyloid-laden vascular walls and in senile plaque cores of amyloid in the brains of patients with AD, Down's syndrome, Hereditary Cerebral Haemorrhage With Amyloidosis, Dutch Type (HCHWA-D), and cerebral infarction initially raised questions about its potential role in the disease. *CysC's* co-localization with A in amyloid-laden vascular walls and in senile plaque cores of amyloid in the brains of patients with AD, Down's syndrome, Hereditary Cerebral Haemorrhage With Amyloidosis, Dutch Type (HCHWA-D), and cerebral infarction initially raised questions about its potential role in the disease. In the brains of elderly people without dementia, CysC also co-localizes with an amyloid deposit. There is a wealth of evidence indicating that CysC functions as a protective factor in AD. Studies conducted in vitro have demonstrated that CysC binds to A and prevents A from oligomerizing and forming fibrils. Results obtained in vivo from the brains and plasma of transgenic mice that deposit A supported CysC's connection with the soluble, non-pathological form of A and its inhibitory effect on the development of A plaques. The connection of CysC with A was also discovered in the brain and Cerebrospinal Fluid (CSF) of AD patients and non-demented controls. Furthermore, in vitro studies have shown that CysC protects neuronal cells from a variety of toxins that can cause cell death, such as oligomeric and fibrillar A. According to these studies, decreased CysC levels associated with AD may enhance neuronal susceptibility and diminish their capacity to stop neurodegeneration. This review elaborates on CysC's neuroprotective functions in AD and the therapeutic utility of this protein in clinical settings.

Key Words: *Neuroprotective Functions*

INTRODUCTION

Basic protein CysC, commonly referred to as "trace," was first discovered in human CSF before being discovered in all other mammalian body fluids and tissues. Neurons, astrocytes, and microglial cells in the brains of many species all express CysC, which is highly prevalent in brain tissue. CysC has a wide range of biological functions, including bone resorption, tumour metastasis, control of inflammatory responses, cell proliferation and growth, and astrocytic differentiation in the developing mouse brain. CysC involvement has been demonstrated in a number of diseases, including cancer and neurological disorders. Numerous studies have shown that changes in CysC concentrations in serum are related to a number of diseases and disorders, including rheumatoid arthritis, cancer, chronic kidney disease, urinary tract infections, hypertension, cardiovascular disease, thyroid function, and ageing. CysC concentration in particular tissues and bodily fluids can act as a marker for a number of illnesses, the development of those illnesses, and the impact of treatment.

Additionally, CysC is thought to have a role in the nervous system's processes of neuronal degeneration and repair. Initially, was discovered to be an inhibitor of cysteine proteases such as cathepsins needed for housekeeping during protein turnover. Uncontrolled proteolysis may result from an imbalance between active proteases and their endogenous inhibitors, which has been linked to a number of neurological disorders. Review of the role of proteases and their inhibitors in the processes of nervous system repair and neuronal degeneration.

The choroid plexus is the primary source of i in the CSF. CysC CSF levels were discovered to be five times greater in normal brain tissue than plasma levels, pointing to a potential physiological function for CysC in the brain. Neurodegenerative diseases have been shown to alter the levels of CysC in the CSF. For instance, Amyotrophic lateral

Editorial office, Journal of Clinical Psychiatry and Neuroscience, United Kingdom

Correspondence: Shawn Kruger, Editorial Office, Journal of Clinical Psychiatry and Neuroscience, United Kingdom; E-mail: clinicalpsychiatry@neurologyjournals.org

Received: 06-November-2022, Manuscript No. PULJCPN-23-6131; Editor assigned: 08- November- -2022, Pre QC No. PULJCPN-23-6131 (PQ); Reviewed: 20-November- 2022, QC No. PULJCPN-23-6131 (Q); Revised: 22- November-2022, Manuscript No. PULJCPN-23-6131 (R); Published: 27- November- -2022, DOI: 10.37532/puljcpn.2022.5(6).70-1.

Sclerosis (ALS), a fatal neuromuscular disease characterised by progressive motor neuron degeneration, has demonstrated a high diagnostic potential for CysC as a biomarker. Compared to healthy controls, CysC levels in the CSF of ALS patients were markedly lower. Additionally, initial CSF CysC levels were predictive of patient survival, and the direction of the longitudinal shift in CSF CysC levels was connected with the pace of ALS disease development, indicating that CysC may serve as a stand-in marker of disease progression and survival. As one of only two proteins known to localise to Bunina bodies and cause the tiny intraneuronal inclusions found in deteriorating motor neurons that are a particular neuropathologic hallmark of ALS, CysC is also connected to ALS histopathologically. In a similar vein, it has been demonstrated that CysC levels in the CSF of AD patients are lower than those of non-demented people. Additionally, certain groups of neuronal cells in the brains of AD patients displayed an increased expression of CysC.