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Review

Detecting the Early Signs of Cognitive Decline

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ABSTRACT

Alzheimer's disease (AD) is a neurodegenerative brain disorder that is a primary cause of cognitive decline and dementia, and its incidence and prevalence are rising in both urban and rural areas. Despite extensive study over the previous thirty years, no effective drug is currently available. Amyloid deposition and neurofibrillary tangles are neuropathological markers of Alzheimer's disease, although their presence does not fully explain the disease. The pathophysiology of the illness A peptide, the proteinaceous precursor of brain amyloid plaques, has just been discovered. As an antimicrobial factor, it has been proposed. Virus and/or bacterial infections have been demonstrated to cause cancer in recent studies.

KEYWORDS: Down syndrome, trisomy 21, chromosome abnormality.

INTRODUCTION

Longitudinal population studies have found that genetic, phenotypic, epidemiological, and clinical factors are linked to an increased risk of cognitive decline or dementia. For example, our findings revealed that some genetic markers, as evidenced by AD genome wide association studies, may reduce host antimicrobial defence responses and influence clinical dementia development. The purpose of this commentary is to quickly demonstrate novel applicative approaches for determining individual dementia risk and the prospect of modifying cognitive decline/dementia risk through individualised preventative measures. Adults with Down's syndrome are at an increased risk of cognitive decline and senility, therefore the methodology outlined here may have practical importance.

CONCLUSION

Main regulatory molecules involved in the cell cycle, transcription and translation, the cytoskeleton, cell-cell adhesion, and receptor-coupled signal transduction have been shown to be phosphorylated by isoforms of the CK1 family. Given their function in essential cellular signalling pathways, it's not shocking that CK1 isoform dysregulation has been related to the occurrence of inflammatory and proliferative diseases, as well as neurodegenerative disorders. We are primarily concerned with cell division in this study. CK1 isoforms are involved in cell division during both mitosis and meiosis. CK1 regulates the transition from interphase to metaphase in mitosis in mammals. CK1 phosphorylates Rec8 subunits of the cohesin complex in budding yeast and fission yeast, regulating chromosome segregation during meiosis. However, the role of CK1 in mammalian oocyte meiosis is still uncertain, and further study is required [1, 2, 3].

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