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Neurodegenerative Process and Ageing of CNS

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Abstract

Neurodegeneration is the advancing deficit of structure or function of neurons, which might also include death of neurons. Neurodegenerative diseases such as Alzheimer's, Parkinson's, amyotrophic lateral sclerosis and Huntington's happen as a consequence of neurodegenerative processes. As research advances, many resemblances are related about diseases to one another on a sub-cellular level. There are many parallels between different neurodegenerative disorders including atypical protein assemblies as well as induced cell death.

Keywords: Neurodegenerative; Sclerosis; Trauma; Ischemia

Neurodegenerative Process

Chen et al. established that amyloid proteins in the microbiota possibly be a cause in the initiating neurodegenerative diseases [1]. Brain injury could be a result of various pathological illnesses. Acute brain injuries cause in the oxidative stress of brain cells leading to trauma and ischemia [2-4]. Also the accumulation of labile zinc in the brain notably promotes oxidative brain injury [5-9]. Similarly, neurodegenerative diseases such as Alzheimer's (AD), Huntington's (HD), Parkinson's (PD) and Amyotrophic Lateral Sclerosis (ALS) are result of advancement of structural deficit or functional damage of neurons. These neurodegenerative disorders lead to induced cell death as well as atypical protein assemblies [10,11]. Zinc deficiency in the brain intensifies chronic neurodegeneration [12], signifying to the key role of zinc dyshomeostasis enacts in the brain diseases.

Initial symptom of neurodegenerative disorders is impairments in olfaction leading to Parkinson's disease (PD) and Alzheimer's (AD) [13-17]. Studies conducted have shown that olfactory deficits are witnessed in people living with psychiatric disorders including schizophrenia [18], and depression [19].

Various researches have pointed association of progression of Alzheimer's disease with elevated blood Cortisol levels [20,21]. Cortisol apparently influences the functioning of alpha, beta and gamma enzymes and Secretases which function for the production and modulation of amyloid-β peptide [22]. Eventually as with high cortisol levels that leads to neurodegeneration [23].

Alzheimer's is depicted by critical intellectual impairment, learning, language, memory, capacity, comprehension, reasoning and judgment. Alzheimer's can be seen usually in elderly people over the age of 65, although in some rare cases it can be seen in individuals of age group above 40 [24]. In the Initial years of the disease, individuals often grow lose some short-term memory, forgetful and have trouble in communication, as well as some difficulty making judgments and reasoning [25]. Some individuals also incident a slight change in behavior or mood. Higher degree of care is required in comparison with other chronic diseases for patients suffering with Alzheimer's or other forms of dementia [26].

Conclusion

The conclusions are predominantly significant because the hippocampus is particularly prone to degenerative and dysfunctional processes through aging or in Alzheimer's disease, and plays a pivotal role in memory and learning processes. The hippocampus involvement can also be seen in the antidepressant responses and mood regulation [27].

Recent evidences have brought to light that, it is crucial to identify that deficits in olfaction are often happen along with sleep disturbances, changes in behaviour, insomnia and fluctuations in the sleep cycle [28]. In initial phase of Parkinson's disease Irregularities in Rapid Eye Movement (REM) sleep stage are observed [29,30]. REM sleep is characterized by rapid eye movements, skeletal muscle paralysis, vivid dreaming, cortical activation and muscle twitches which is intermediated by a dispersed system within the brainstem, limbic regions and hypothalamus, including the crucial amygdala in the regulation of REM sleep [31]. REM sleep Rapid Behaviour Disorder (RBD) cause disturbance in the neural control of REM sleep which is correlated to dream-enacted behaviour coupled with skeletal muscle atonia during REM sleep [32]. 37% of individuals suffering with RBD progressed with parkinsonian disorder at average period of 3.7 years after RBD onsets. Other conclusions suggest that patients with RBD may develop dementia or PD in 66.7% of as reported by patients [30-36].

References

1. Chen SG, Stribinskis V, Rane MJ, Demuth DR, Gozal E, et al. (2016) Exposure to the functional bacterial amyloid protein curli enhances alpha-synuclein aggregation in aged Fischer 344 rats and Caenorhabditis elegans. Sci Rep 6: 34477.
2. Bramlett HM, Dietrich WD (2004) Pathophysiology of cerebral ischemia and brain trauma: similarities and differences. J Cereb Blood Flow Metab 24: 133-150.
3. Lewen A, Matz P, Chan PH (2000) Free radical pathways in CNS injury. J Neurotrauma 17: 871-890.
4. Koike M, Shibata M, Tadakoshi M, Gotoh K, Komatsu M, et al. (2008) Inhibition of autophagy prevents hippocampal pyramidal neuron death after hypoxic-ischemic injury. Am J Pathol 172: 454-469.
5. Cho E, Hwang JJ, Han SH, Chung SJ, Koh JY, et al. (2010) Endogenous zinc mediates apoptotic programmed cell death in the developing brain. Neurotox Res 17: 156-166.
6. Hwang JJ, Lee SJ, Kim TY, Cho JH, Koh JY (2008) Zinc and 4-hydroxy-2-

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nonenal mediate lysosomal membrane permeabilization induced by H_2O_2 in cultured hippocampal neurons. J Neurosci 28: 3114-3122.

7. Kim J, Kim TY, Hwang JJ, Lee JY, Shin JH, et al. (2009) Accumulation of labile zinc in neurons and astrocytes in the spinal cords of G93A SOD-1 transgenic mice. Neurobiol Dis 34: 221-229.
8. Lai L, Zhao C, Su M, Li X, Liu X, et al. (2016) *In vivo* target bio-imaging of Alzheimer's disease by fluorescent zinc oxide nanoclusters. Biomater Sci 4: 1085-1091.
9. Lee SJ, Seo BR, Choi EJ, Koh JY (2014) The role of reciprocal activation of cAbl and Mst1 in the oxidative death of cultured astrocytes. Glia 62: 639-648.
10. Bredesen DE, Rao RV, Mehlen P (2006) Cell death in the nervous system. Nature 443: 796-802.
11. Rubinsztein DC (2006) The roles of intracellular protein-degradation pathways in neurodegeneration. Nature 443: 780-786.
12. Lee SJ, Seo BR, Koh JY (2015) Metallothionein-3 modulates the amyloid beta endocytosis of astrocytes through its effects on actin polymerization. Mol Brain 8: 84.
13. Doty RL, Deems DA, Stellar S (1988) Olfactory dysfunction in parkinsonism: a general deficit unrelated to neurologic signs, disease stage, or disease duration. Neurology 38: 1237-1244.
14. Hawkes C (2003) Olfaction in neurodegenerative disorder. Mov Disord 18: 364-372.
15. Talamo BR, Rudel R, Kosik KS, Lee VM, Neff S, et al. (1989) Pathological changes in olfactory neurons in patients with Alzheimer's disease. Nature 337: 736-739.
16. Thompson MD, Knee K, Golden CJ (1998) Olfaction in persons with Alzheimer's disease. Neuropsychol Rev 8: 11-23.
17. Masaoka Y, Pantelis C, Phillips A, Kawamura M, Mimura M, et al. (2013) Markers of brain illness may be hidden in your olfactory ability: a Japanese perspective. Neurosci Lett 549: 182-185.
18. Brewer WJ, Wood SJ, McGorry PD, Francey SM, Phillips LJ, et al. (2003) Impairment of olfactory identification ability in individuals at ultra-high risk for psychosis who later develop schizophrenia. Am J Psychiatry 160: 1790-1794.
19. Zucco GM, Bollini F (2011) Odour recognition memory and odour identification in patients with mild and severe major depressive disorders. Psychiatry Res 190: 217-220.
20. Dong H, Csernansky JG (2009) Effects of Stress and Stress Hormones on Amyloid¬β Protein and Plaque Deposition. J Alzheimers Dis 18: 459-469.
21. Csernansky JG, Dong H, Fagan AM, Wang L, Xiong C, et al. (2006) Plasma cortisol and progression of dementia in subjects with Alzheimer type dementia. Am J Psychiatry 163: 2164-2169.
22. Swaab DF, Raadsheer FC, Endert E, Hofman MA, Kamphorst W, et al. (1994) Increased cortisol levels in aging and Alzheimer's disease in postmortem cerebrospinal fluid. J Neuroendocrinol 130: 1707-1712.
23. Strooper BD, Vassar R, Golde T (2010) The secretases: enzymes with therapeutic potential in Alzheimer disease. Nat Rev Neurol 6: 99-107.
24. Hardy J, Allsop D (1991) Amyloid deposition as the central event in the aetiology of Alzheimer's disease. Trends Pharmacol Sci 12: 383-388
25. Martin Prince A, Wimo A, Guerchet M, Gemma-Claire A, Ali M, et al. (2015) World Alzheimer Report 2015. The Global Impact of Dementia an Analysis of Prevalence, Incidence, Cost and TrenDs.
26. Duthey BB (2013) Priority Medicines for Europe and the World "A Public Health Approach to Innovation".
27. Yeshurun Y, Sobel N (2010) Anodor is not worth a thousand words: from multidimensional odors to unidimensionalodor objects. Annu Rev Psychol 61: 219-241, C1-C5.
28. Postuma RB, Lang AE, Massicotte-Marquez J, Montplaisir J (2006) Potential early markers of Parkinson disease in idiopathic REM sleep behavior disorder. Neurology 66: 845-851.
29. Boeve BF, Silber MH, Ferman TJ, Lucas JA, Parisi JE (2001) Association of REM sleep behavior disorder and neurodegenerative disease may reflect an underlying synucleinopathy. Mov Disord 16: 622-630.
30. Schenck CH, Bundlie SR, Mahowald MW (1996) Delayed emergence of a parkinsonian disorder in 38% of 29 older men initially diagnosed with idiopathic rapid eye movement sleep behaviour disorder. Neurology 46: 388-393.
31. International Classification of Sleep Disorders (2005) Diagnostic and Coding Manual (Edn 2) American Academy of Sleep Medicine, Westchester.
32. Fraigne JJ, Torontali ZA, Snow MB, Peever JH (2015) REM Sleep at its Core-Circuits, Neurotransmitters, and Pathophysiology. Front Neurol 6: 123.
33. Cotter RL, Burke WJ, Thomas VS, Potter JF, Zheng J (1999) Insights into the neurodegenerative process of Alzheimer's disease: a role for mononuclear phagocyte-associated inflammation and neurotoxicity. J Leukoc Biol 65: 416-427.
34. Isidro F (2012) Defining Alzheimer as a common age-related neurodegenerative process not inevitably leading to dementia. Prog Neurobiol 97: 38-51.
35. Wakaskar RR, Bathena SPR, Tallapaka SB, Ambardekar VV, Gautam N (2014) Peripherally cross-linkingtheshellofcore-shellpolymermicellesdecreases premature release of physically loaded combretastatin A4inwholebloodand increases its mean residence time and subsequent potency against primary murine breasttumorsafterIVadministration. Pharm Res 32: 1028
36. Ambardekar VV, Wakaskar RR, Sharma B, Bowman J (2013) The efficacy of nuclease-resistant Chol-siRNA in primary breast tumors following complexation with PLL-PEG(5K). Biomaterials 34: 4839-4848.

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