ABSTRACT

This chapter describes how an individual progresses towards aging, several age-related cognitive declines are becoming an ever-increasing problem. Ageing causes changes to brain size, vasculature, and cognition. Protective factors that reduce cardiovascular risk, namely regular exercise, a healthy diet, and low to moderate alcohol intake, seem to aid the ageing brain as does increase cognitive effort in the form of education or occupational attainment. A healthy life both physically and mentally may be the best defense against the changes of an ageing brain. This chapter aims to characterize changes in brain structure with aging, and to investigate relationships between brain aging and cognitive decline. Along with these it will make and attempt to identify possible management, treatment and preventive measures for managing cognitive impairment in brain ageing and promoting cognitive reserve for healthy brain ageing.

INTRODUCTION

With the world facing an aging demographic, strategies to preserve cognitive and physiological functions and prevent age-related diseases are of government priority in many countries (Kirkwood, 2008); early cognitive decline is a stage where intervention is most likely to be effective. The biological basis of cognitive ability and how it changes with age is therefore of enormous interest (Deary, Penke, & Johnson, 2010). In particular, the biological basis of cognitive reserve, of which childhood mental ability is a useful index (Whalley et al., 2004), is important in any life course approach to cognitive aging. Over the last few decades, a large body of research has been conducted to identify those changes in cognition...
that represent the normally aging brain by contrast to those that may be evidence of brain pathology. The areas of cognition that are often examined in this context include memory, language, visuospatial ability, speed of information processing, attention and executive functioning.

The effects of ageing on the brain and cognition are widespread and have multiple etiologies. Ageing has its effects on the molecules, cells, vasculature, gross morphology, and cognition. Using National Institute of Mental Health criteria in an elderly population it was reported that a high (38.4%) prevalence of age-associated memory impairment (AAMI). In a study of the International Psychogeriatric Association proposed criteria for ‘ageing-associated cognitive decline’ (AACD). The prevalence was slightly related to age and education. The rate was lowest in the oldest age of 75 - 78 years (20.5%) and highest in the age of 71 -74 years (30%). Subjects with less than 4 years of education had the lowest (14.3%) and subjects with more than 6 years of education had the highest rate (29.4%) for AACD (Hanninen et al., 1996). Brain ageing has been reported due to several reasons. In this cohort, irrespective of the clinical symptoms, the frequency of Alzheimer’s disease-related pathology is between 19 and 67%, of Lewy body pathology is between 6 and 39%, of vascular pathologies is between 28 and 70%, of TDP-43 proteinopathy is between 13 and 46%, of hippocampal sclerosis is between 3 and 13% and, finally, of mixed pathologies is between 10 and 74%. Some studies also mention tauopathies. White-matter pathologies are not discussed specifically in all studies, although these lesions may be present in more than 80% of the aging brains (Rahimi & Kovacs, 2014).

Age-associated cognitive decline is becoming more and more prevalent. There are many causes of dementia, but neurodegenerative diseases (NDDs) are thought to be one of the most prevalent in the aging population. Indeed, during the last century neuropathological examinations have demonstrated that the brains of the majority of the individuals with cognitive decline show Alzheimer’s disease (AD)-related pathologies, including neurofibrillary tangles and senile plaques. This observation led to the concept that Alzheimer’s Disease is the most frequent neurodegenerative diseases and cause of cognitive decline in older people. Community-based neuropathology studies have shown that complex constellations of underlying pathologies may lead to cognitive decline, and that the number of possible combinations increases in the aging brain.

Structural alterations observed mostly in cross-sectional imaging studies may, however, be related to events much earlier in life, and not all attributable just to advancing age (Penke et al., 2010). For example, the size and function of the brain in old age is likely related to its size and functional ability in early life, and even to in utero factors (Shenkin et al., 2009). Resistance to vascular disease and its effects on the heart, brain and other organs may also be ‘preset’ in utero. Genetic, and early life social, environmental, educational and nutritional factors may, therefore, all interact and lead to events in adulthood that influence the state of the brain and its vulnerability to additional insults in old age (Deary et al., 2009; Deary & Johnson, 2010).

Mental abilities change throughout life, first as a result of brain maturation and later with aging of brain cells and their billions of complex interconnections. As people age, their movements and reflexes slow and their hearing and vision weaken. Ageing has its effects on the molecules, cells, vasculature, gross morphology, and cognition. With ageing, the brains shrink in volume, particularly in the frontal cortex. Vasculature ageing and rising of blood pressure increases the possibility of stroke and as a result ischaemia increases and white matter may develop lesions. Memory decline also occurs with ageing and brain activation becomes more bilateral for memory tasks. Genetics, neurotransmitters, hormones, and experience play important role in brain ageing. But, it is not all negative, higher levels of education or occupational attainment may act as a protective factor. Also protective are a healthy diet, low to moder-
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