Chapter 2
“Personal Training”: Can Genes Guide Us?

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ABSTRACT

Dementia is one of the most devastating disease of the elderly. Alzheimer’s disease (AD) remain its most common cause. This chapter presents current data about AD risk factors, biomarkers, risk genes, available treatments. It also focuses on current and future perspectives about the use of personalized, non pharmaceutical computer-based intervention. The aim of this work is to propose that the knowledge of carrying or not AD risk can be a guide for a personalized combination of brain training in preclinical stages of the disease in order to postpone or even cancel disease onset.

INTRODUCTION

While Earth population grows older, diseases such as dementia become more common. Early diagnosis, better prognosis and more effective treatment, unfortunately, still remain an issue in case of Dementia. Researches try to investigate the causes of this devastating disease by discovering new biomarkers, such as cerebrospinal fluid proteins, by genotyping human DNA or by studying brain physiology, structure and function, using methods such as EEG, MEG, MRI, PET, fMRI. In literature, exist various suggestions about how to prevent dementia onset, raising a lot of questions: i.e. Is this really possible? Can be the same instructions suitable for everybody? What is really the main factor that will define the best combination of protective interventions in preclinical stages of the disease or even in healthy individuals? In this chapter, a brief presentation of Alzheimer’s disease is made, which is the most cause of dementia. Risk factors, clinical criteria, available treatments, risk genes are also presented. The aim of this presentation is the preclinical stages, known as mild cognitive impairment, and the possible non pharmaceutical interventions that can be applied according to one’s genotype, using new technologies and special designed software for elderly.

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BACKGROUND

Dementia: Alzheimer’s Disease

Dementia is a devastating disease of the elderly that is rather common nowadays due to the prolongation of the life span. It is characterized by cognitive decline, functional disability and behavioral disorders causing to the loss of one’s independency. The prevalence of the disease, in western world, for the ages >60 years old it is >5% and doubles every 5 year >65 years old, ending >25% for the ages over the 90 years old (Qiu, Kivipelto, & von Strauss, 2009).

Epidemiology

The most common cause of dementia is Alzheimer’s disease (AD) (Fratiglioni et al., 2000; Plassman et al., 2007). Alzheimer’s disease is one of the most common morbidities in elderly and its prevalence is expected to quadrupled in 2047 (Brookmeyer, Gray, & Kawas, 1998). The cause of this neurodegenerative syndrome is still unclear. It is certain, however, that both genetic and environmental factors take part in Alzheimer’s disease pathogenesis (Jiang, Yu, Tian, & Tan, 2013).

The diagnosis is based upon clinical criteria such as NINCDS-ADRDA (G. M. McKhann et al., 2011; G. McKhann et al., 1984) and DSM V (American Psychiatric Association, 2013). Both of the above criteria do not include currently biologic markers (Mattsson et al., 2009; Mulder et al., 2010) or predisposing genes.

Risk Factors

Multiple risk factors have been related to AD (see Table 1). Some of them can be modified, such as depression and social isolation, whereas others cannot: e.g. age. Furthermore, only a few of them are directly related to the disease with a rather obvious biologic mechanism while others are still under investigation due to their complexity.

It is a common try of physicians, nurses, social workers and psychologists to modify positively as many of them as possible, even before the clinical onset of the disease. However, from all these risk factors, age is the most crucial, considering that the incidence of sporadic AD increases significantly after 65 years old and is becomes double every 5 years (Alves, Correia, Miguel, Alegria, & Bugalho, 2012).

Biologic Markers

The biologic markers can be examined in vivo and are related to specific pathophysiological mechanisms of AD. Their presence in even pre-clinical stages is connected to the AD prognosis and differential diagnosis (Engelborghs, 2013). Commonly accepted biomarkers are the Cerebrospinal Fluid (CSF) markers i.e. Aβ-42, Tau protein and phospho- Tau-181 (Martin & Allen, 2013; Tapiola et al., 2009), the amyloid plaques observed via PIB-PET, glucose metabolism ([18]F fluodeoxyglucose PET), structural Brain atrophy observed via MRI (Trzepacz et al., 2013; Weiner et al., 2013), as well as, the Event related Potentials as a neurophysiologic marker (Olichney et al., 2008; Papaliagkas, Kimiskidis, Tsolaki, & Anogianakis, 2011). EEG has also been proved to be an effective research and clinical tool for differential diagnosis of dementia (Babiloni et al., 2013; Jeong, 2004; Rossini, Rossi, Babiloni, & Polich, 2007). Hence, recent neurophysiological studies propose indexes of MCI progression to AD with an accuracy about 88.3% (Moretti et al., 2011; Tsolaki et al. 2014).

Genetics

There are two primary types of AD as defined by the age of disease onset. The first is early onset AD (EOAD), and the second type is late onset AD (LOAD). Each one has a unique set of causative or risk related genetic factors. In EOAD the genes has been characterized as rather causative.
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