Institute of Experimental Morphology, Pathology and Anthropology with Museum
Bulgarian Anatomical Society

Acta morphologica et anthropologica, 25 (3-4)
Sofia ● 2018

Evidence of Neuronal Damage in Elderly

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Blood sera from 13 psychologically and neurologically healthy volunteers from Sofia aged 90-95 years were investigated. Our previous studies have shown that GD1a antibodies can be considered as biomarkers for neurodegenerative changes. According to the performed ELISA technique, no elevated IgG titers of these antibodies were detected. This shows that the neurons of this people are healthy independently of the ageing.

Key words: GD1a, old elderly people, neurodegeneration

Introduction

Health is the ability of a biological system to acquire, convert, allocate, distribute, and utilize energy sustainably. The World Health Organization (WHO) defined human he-alth in a broader sense in its 1948 constitution as "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" [12].

Over the last 150 years, life expectancy has increased by 2 years for every decade. Today in the developed countries it is 75-80 years, but the duration of health increases by about 1.7 per decade. More people suffer from age-related illnesses – cancer, cardiovascular disease, diabetes, hypertension, dementia, osteoporosis, eye and hearing problems and others. Life time duration over the years is: 1850-40 years; 1900-47 years; 1950-68 years; 2000-78 years; 2050-88 years. The solution is: moderate sports and exercise, nutrition – excessive intake of carbohydrates definitely shortens life (and in what form it is taken), it is good not to eat as many hours as possible, repairing the damage to the body from outside [11]. Our studies since 2002 have convincingly demonstrated that the titer of GD1a gangliosides may be a marker for neuronal damage. We have found in our experimental model of multiple sclerosis (MS) – chronic remitting experimental allergic encephalomyelitis (CREAE) that before the destruction of the myelin sheath, there is an increased titer of GD1a gangliosides, which is a sign of neuronal damage [5].

Materials and Methods

The health status of 76 randomly chosen volunteers between 90 and 104 years of age from all parts of Bulgaria is described. The illnesses that appear on our list are just like those described by prof. E. Verdin as diseases of the elderly all over the world [11]. A group of all 13 persons who are from Sofia are presented. Their sera were estimated by enzyme-linked immunosorbent assay (ELISA) technique for detecting IgG anti-GD1a antibodies [6]. People are selected one at a time and they do not have any neurological or psychiatric indications – their neurons are healthy.

All these 13 patients were without any neurological or mental illness. The ELISA protocol was selected by slight modifications of the method of Mitzutamari et al [6].

Results

Blood sera from 13 psychologically and neurologically healthy volunteers from Sofia aged 90-95 years were studied. In the ELISA technique, no elevated IgG titers of anti-GD1a antibodies were detected. It is important to note that in all 13 blood samples, there was no need for centrifugation because the serum was self-excreted. This is because all healthy old people who were tested take preparations for decrease of blood viscosity and thus they are in great condition **(Table 1)**.

Table 1. Titers of anti-GD1a antibodies in sera from elderly (90-95 years old)

Age / **OD Optical density**	90-95 year old n=13
IgG anti – GD1a Mean ± SEM	(-) ≤ 0.06 ± 0.02

Data are presented as a mean value x± standard error of mean (SEM)
Legend: OD – optical density; SEM – standard error of mean; n – number of patients

Discussion

Screening at the level of anti-GD1a antibodies indicates that neuronal damage can be traced in many patients. The mechanism by which ganglioside GD1a participates in the cell cohesion is not known. So we decided to study elderly with different diagnoses but without dementia. When there is no evidence for dementia, for impaired function of the neuron, the elderly are mentally healthy no matter how old they are. In all 13 elderly there was no increase in the antibody titers against GD1a meaning that there were no neuronal damages.

The production of fibrinolytic enzyme plasmin, produced by vascular endothelial cells, decreases with the age. This is the main reason for increased coagulation (blood clotting). On the other hand, fibrinogen increases with the age. This causes increased platelet aggregation, which increases the risk of stroke and heart attack. The elevated fibrinogen is a more significant risk factor for strokes than high blood cholesterol levels. To this should be added the combination of risk factors with different relative weight [7].

Plasma lipids could increase the cerebrovascular risk through alteration of the hemorheological profile [9]. Parallel with whole blood viscosity (WBV) many hematological parameters, including the erythrocyte morphology have also been evaluated [3].

Nicotinamide adenine dinucleotide (NAD(+)) is a coenzyme found in all living cells. Here we review factors that regulate NAD(+) and discuss how supplementation with NAD(+) precursors may represent a new therapeutic opportunity against aging and associated disorders, particularly neurodegenerative diseases [10]. The ketone body β-hydroxybutyrate (βOHB) is a convenient source of energy from adipocytes to peripheral tissues during fasting or exercise. These regulatory functions of βOHB serve to link the outside environment to cellular function and gene expression, and have important implications for the pathogenesis and treatment of metabolic diseases including diabetes type 2 [8].

Factors that support human health are: income and social status; social support network; education and literacy; work conditions; social environment; physical environment; personal health practices and coping skills; healthy development in childhood; biology and genetics; health services; culture. Longevity is a side effect of health [12].

The causes of stroke have been tested in 47 countries and are described as follows: 1. Hypertension – 24%; 2. Lack of regular physical activity – 18%; 3. Not administering statins – ratio of apolipoproteins B/A1 – 13%; 4. Harmful feeding habits – 12%; 5. Waist to hip ratio – 9%; 6. Psychosocial factors – stress, bullying at the workplace – 8.5%; 7. Smoking – 6%; 8. Cardiovascular diseases – 4.5%; 9. Alcohol consumption – 3%; 10. Diabetes mellitus – 2% [7].

Global grey matter volume decreases linearly with age, with a significantly steeper decline in males. Local areas of accelerated loss were observed bilaterally in the insula, superior parietal gyri, central sulci, and cingulate sulci. Areas exhibiting little or no age effect (relative preservation) were noted in the amygdala, hippocampi, and endorhinal cortex. Global white matter does not decline with age, but local areas of relative accelerated loss and preservation were seen [4]. From the brain regions affected by ageing, the hippocampus and the prefrontal cortex (PFC) seem to be particularly vulnerable, but even within and between these regions the impact of ageing on the neuronal function can differ. The morphology of neurons in the PFC is more susceptible to age-related changes, as these cells show a decrease in dendritic branching in rats and humans [1].

In 2017 David Cundiff reports 19 different relative risk factors for cardiovascular disease in 168 countries. They include: consumption of products of animal origin; refined carbohydrates; alcohol; tobacco; vitamin K2 intake; level of exercise; body mass index; blood sugar/hemoglobin A1c; problems with the blood pressure; medicines for hypertension; cholesterol/HDL ratio; the incomes of individuals; level of education; gender; age; ethnic origin; vitamin D level; air pollution; fetal, baby and childhood stress [2].

There is no evidence of neuronal damage in elderly who are born in 1924 to 1928 and are in good mental and psychological health.

Acknowledgements: I would like to express my gratitude to Svetlana Vladova, Ivayla Ivanova-Pandourska and Irina Evstatieva for provided excellent technical assistance.

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