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Editorial

will be doubled in 25-30 years [1,2].

Now two molecules are thought to be the most important in the the maintenance of amyloid plague was mostly expressed what allows to assume direct participation of -amyloid in the process of neuronal death in AD in this mice [2].

Tau-protein is the stabilized phosphoprotein, functionally linked with microtubules and providing the structurally functional cytoskeleton organization [4]. In the patients with AD tau-p 0.5 (tn)4 (t1hi)3 (s mic des)5 (t)-5 (r.)3 (a)(h)4 [th6 9 Tw T* ei(n t)-6 (h disease (AD) manifestation is con rmed nally only at autopsy using speci c immunohistochemical methods [2]. Undoubtedly, the problem of life-time diagnosis of AD is one of the most actual problem of biomedicine. is circumstance dictates the necessity to develop lifetime markers for diagnosis and prognosis of AD [1,3-5].

We suppose that the study of extra-brain tissues and cells which are available for biopsy could be very promising for this purpose. Most publications on the use of peripheral cells for diagnosis of AD are devoted to study skin broblasts. From our point of view the peripheral blood lymphocytes (PBL) could be considered as more suitable samples for the research in this eld of study for many reasons (one of them is the ability of lymphocytes to produce biologically active substances, which could be involved in the pathogenesis of AD).

In 2000 we have tried to detect the expression of tau-protein in human peripheral blood lymphocytes (PBL) in patients with AD,

and our attempt was successful: the expression of tau-protein was Alzheimer's disease (AD) is characterized by progressing loss studied [6]. e International Consortium of AD studying, immunocytochemically shown in PBL in absolute majority of AD of the memory, bright dementia and is one of the most serious reated in the USA on the basis of National Institute of Ageing and neurodegenerative frustration. Now AD is observed in more than 20 eigan's Institute species in the recommendations that the pathology million people all over the world, and their quantity as it is expected of tau-protein is one of the most important components of AD pathogenesis.

mechanism of AD development: -amyloid and tau-protein [3]. eld on the great cohort of patients, and we found a very interesting During last 10 years we continue and extend the research in this Existence of pathogenetic connection between -amyloid accumulation act: the AD developed signi cantly less frequently in the group of and neuro brillial damages was proved in transgenic mice. e selective so-called "children of blockade' Leningrad" as well as tau expression neuronal destruction was marked in departments of a brain in which their lymphocytes was also less pronounced than in patients of the

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