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ABSTRACT BOOK

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findings in literature, suggesting depression like effects of BDNF administration into nucleus accumbens.

Keywords: Depression, antidepressants, electroconvulsive shock, BDNF, nucleus accumbens

P13

Auditory evoked N100 and P200 potentials in Alzheimer's disease

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Alzheimer's disease (AD) is the most common neurodegenerative disorder and the most prevalent cause of dementia. Prolonged latency and decreased amplitude of P300 in oddball paradigm are well known neurophysiological parameters. However, reports on the changes of N100 or P200 potentials in AD are rather rare and contradictory. In the present study, N100 and P200 waves that are mainly generated in sensory and unimodal association cortices were investigated in order to search for electrophysiological changes arising from these regions that are assumed to be affected in later stages of the disease. N100 and P200 potentials of the auditory event-related potentials of 15 healthy volunteers and 22 patients at early (n=11, GDS<=4 and CDR < 2) to moderate (n=11, GDS>4 or CDR >=2) stages of AD were analyzed. The differences among the three groups were statistically tested with a repeated-measures ANOVA. It was observed that N100 potentials of the moderate AD group to target stimuli of the novelty paradigm peaked later than those of the control group ($p<0.01$). This effect had a topographical pattern such that the latency was especially prolonged in the fronto-central sites but not in the parietal site ($p=0.009$). In both oddball and novelty paradigms, N100 potentials of the early stage AD patients to standard stimuli peaked significantly earlier over the left hemisphere ($p=0.044$ in the novelty paradigm, $p=0.051$ in the oddball paradigm). No other significant differences were found among the groups. The N100 latency changes between moderate AD patients and controls, but not between controls and mild AD patiens, is in accordance with the later onset of damage in sensory and unimodal association cortices. The lateralization effect in N100 latencies to standard stimuli found between control group and mild AD patients might reflect top-down modulation of N100 by structures claimed to be damaged earlier.

Keywords: Alzheimer's disease, dementia, N100, P200, auditory evoked potential

P14

Polymorphisms at the ligand binding site of the vitamin D receptor gene and Alzheimer's disease

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Alzheimer's disease (AD) is a progressive neurodegenerative disorder that effects whole regions of the brain. The key aims in therapeutic strategies of AD are to decrease the neuronal damage, maintenance or regeneration of neurons. 1,25(OH)2D3 (Vitamin D3) can act on cells of the nervous system by modulating the production of neurotrophins. Vitamin D3 also could mediate its neuroprotective effects via the modulation of neuronal calcium homeostasis. Regulation of nerve growth factor (NGF) synthesis by Vitamin D3 indicates that it could be valuable on determining the neuron's fate. Recent therapy studies with neurotrophic factors such as NGF have shown that this factors could be effective on extending the life time of cognitive system cells and regeneration of neurons. In this way, the polymorphisms which could effect the relationship between Vitamin D3 and its receptor (Vitamin D receptor-VDR) may be important on the period and impact of therapy. In addition, the polymorphisms which can be effective on the affinity of Vitamin D3 to its receptor may influence the synthesis of NGF. In support of this, VDR gene polymorphisms can be related with neurodegenerative disease and neuronal damage. In this preliminary study, our aim was to determine if there is an association between VDR gene and late-onset AD. We collected blood samples from 43 cases of dementia of Alzheimer type and from 37 age-matched controls (mean ages 74.7, and 72.2 years, respectively). Patients are clinically diagnosed according to DSM-IV criterias. We used PCR and RFLP to test for an association between AD and Taq 1 polymorphism at VDR gene. As a result we found 41.9% genotype TT, 44.2% genotype Tt, 14% genotype tt for patients, and 48.6% genotype TT, 32.4% genotype Tt, 18.9% genotype tt for healthy control. When the control and patitents were compared we saw that the distribution of genotypes and alleles did not differ according to Chi-square test ($p=0.50$). In our preliminary results we were unable to find an association between the Taq 1 polymorphism on VDR gene and late-onset AD.

Keywords: Vitamin D, VDR, Alzheimer's disease, Taq1, polymorphism

P15

Interleukin 1 alpha gene promotor region polymorphism in Alzheimer's disease

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Interleukin-1 is a pluripotent immunomodulatory cytokine that has an initiating role in cellular and humoral immunity in the periphery. It is reported that a polymorphism in the 5'-flanking regulatory region at –889 of the interleukin-1 alpha (IL-1alfa) gene may cause an over expression of IL-1 alfa, which is also shown to be associated with inflammatory diseases and Alzheimer's disease. In this preliminary study, our aim was to determine if there is an association between IL-1 alfa gene and late-onset Alzheimer's disease. We collected blood samples from 52 cases of dementia of Alzheimer type and from 35 age-matched controls (mean ages 75.1±5.7, and 72.7±7.3, years respectively). Patients are clinically diagnosed by Istanbul University, Cerrahpasa Faculty of Medicine, Department of Geropsychiatry and Istanbul University, Istanbul Faculty of Medicine, Department of Neurology, Behavioral and Movement Disorders Unit according to DSM-IV criterias. Salting-out method with 5M NaCl is used for DNA isolation. We used polimerase chain reaction-confronting two-pair primers (PCR-CTPP) to test for an association between Alzheimer's disease and a polymorphism at -889 of the IL-1 alfa gene. After genetic analysis of the IL-1 alfa gene, we found 63.5% genotype C/C, 32.7% genotype C/T, 3.8% genotype T/T for patients, and 48.6% genotype C/C, 45.7% genotype C/T, 5.7% genotype T/T for healthy control. When the control and patitents were compared for C/C, C/T and T/T genotypes we saw that the distribution of genotypes and alleles did not differ according to Chi-square test ($p=0.39$). Our preliminary results show no significant increase in the risk for the T/T or C/T genotype in late-onset cases. Thus, we were unable to find an association between the C –889T transition on IL-1 alfa gene and late-onset Alzheimer's disease.

Keywords: IL-1, Alzheimer disease, inflammation, polymorphism, PCR-CTPP

P16

Evaluation of the effect of blood and urine samples on Pc 12 cell line viability in Alzheimer's disease

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Alzheimer's disease (AD) is a neurodegenerative disease mostly seen in older ages. The toxic effects of pathological markers, namely amyloid plaques and neurofibrillary tangles, on neuronal cells have been shown. Also, there is a probabilty of being neurotoxic agents in urine and blood in Alzheimer's disease. For that reason, in this study, it's aimed to investigate the effects of Alzheimer patients' urine and blood samples on neuronal cells. In this study, 15 Alzheimer patients 15 control subjects are included. PC 12 cells are used to identify the effects of blood and urine samples on neuronal cell death. The blood samples of patients and control subjects are added on PC12 cell line at a ratio of 5%, 10%, 15%. The urine samples of patients and control subjects are added on PC 12 cell line at a ratio of 1/10. The toxic effect is determined by MTT viability test after 48 and 72 hours for blood and urine samples respectively. As a result, it's concluded that there is no neurotoxic or neuroprotective effect of Alzheimer patients' urine samples. It's determined that Alzheimer patients' blood samples which are added at a ratio of 15% have neuroprotective effect. These results show us that there may be some materials in Alzheimer patients' blood samples which cause neuroprotection.

Keywords: Alzheimer's disease, blood sample, urine sample, neurotoxicity, neuroprotection

P17

The antinociceptive effect of centrally administered CDP-Choline in rats

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