INTRODUCTION
The normal functioning of dopaminergic system is compromised during Alzheimer’s disease (AD). The activity of monoamine oxidase B (MAOB), enzyme involved in degradation of dopamine, is also disturbed in AD. It was observed that MAOB activity is increased during AD. Also, increased expression of MAOB was detected in hippocampus and cortex of people who suffered from AD. It was observed that MAOB rs1799836 polymorphism can affect MAOB transcription, consequently influencing also protein translation and MAOB activity. Our recent study showed that the levels of cerebrospinal fluid (CSF) amyloid β (Aβ) in patients carrying A allele in MAOB rs1799836 polymorphism. The goal of this study was to compare MAOB rs1799836 polymorphism with APOE, the only confirmed genetic risk factor for sporadic AD.

MATERIALS AND METHODS
Study included 253 participants of whom 127 suffered from AD, 57 were mild cognitive impairment patients, 11 were healthy controls and 58 suffered from other primary causes of dementia. Genomic DNA was extracted from peripheral blood using the salting-out method. MAOB rs1799836 single nucleotide polymorphism (SNP) and APOE SNPs (rs7412 and rs429358) were determined using TaqMan SNP Genotyping Assays (Applied Biosystems, Foster City, CA, USA).

RESULTS
There was significant increase in the frequency of APOE ε4/ε4 homozygotes among patients carrying AA MAOB rs1799836 genotype in comparison to patients carrying other MAOB rs1799836 genotypes (Figure 1) or G allele carriers (Figure 1; Figure 2). Also, there was significantly higher frequency of APOE ε4 carriers (ε4/ε4 + ε4/εx genotypes) among AA MAOB rs1799836 homozygotes (Figure 3). Since MAOB gene is located on X chromosome, we analysed if distribution of MAOB rs1799836 genotypes between patients with different APOE genotypes was different with MAOB rs1799836 genotypes being adjusted for sex (Table 1). Logistic regression model revealed that AA MAOB rs1799836 genotype had significant association with APOE ε4 allele, APOE ε4ε4 and APOE ε4/εx genotype even when adjusted for sex (Table 1).

CONCLUSIONS
The present study sheds light on MAOB rs1799836 polymorphism as potential genetic biomarker of AD. We previously proved the association of this polymorphism with Aβ levels measured in CSF (Babić Leko et al., J Alzheimers Dis, 2020), and now we also showed the association of MAOB rs1799836 polymorphism with APOE genotype. In conclusion, the results of this study together with our previous results indicate that the MAOB rs1799836 polymorphism could be strong genetic biomarker of AD.

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