Dear Author,

Please check your proof carefully and mark all corrections at the appropriate place in the proof (e.g., by using on-screen annotation in the PDF file) or compile them in a separate list.

For correction or revision of any artwork, please consult [http://www.elsevier.com/artworkinstructions](http://www.elsevier.com/artworkinstructions).

No queries have arisen during the processing of your article.

Thank you for your assistance.
Dear Editor,

Brain-derived neurotrophic factor (BDNF) has been implicated in the pathogenesis of coronary artery disease (CAD) [1]. BDNF Val66Met polymorphism has recently been associated with particular manifestation of CAD (unstable angina) in Chinese population [2]. To further explore role of this polymorphism in CAD we have investigated its association with myocardial infarction (MI) in the Czech Caucasian population.

The frequencies of BDNF Val66Met (rs6265) variant were determined in the group of 217 MI patients [age, median (range): 53 (25–79); males/females: 185/32] diagnosed according to the international consensus criteria [3] and compared to those in healthy control group [N = 180; 29 (18–64); 95/85]. The genotype and allele frequencies of the BDNF Val66Met polymorphism in the MI patients (66Met allele frequency: 17.7%) nearly equaled those observed in healthy control subjects (17.2%, p > 0.05, Table 1). Both investigated groups also did not differ in carriage rates of BDNF 66Val/Met alleles. Similarly, no association of this BDNF variant with MI was observed in the subanalysis according to the gender (data not shown).

In addition to its principal role in neural system, BDNF has also been implicated in the vascular development, repair of vascular injury and CAD pathogenesis [1,4,5]. The gene variants affecting BDNF expression and/or structure may, therefore, be considered as plausible candidates in CAD genetics [6,7]. The report which motivated the present study described the association of less common BDNF 66Met allele with protection from unstable angina in Chinese population [2]; the authors suggested that this SNP may affect local expression of BDNF in inflamed arterial wall and interfere with the plaque rupture.

Our study could not provide further evidence that BDNF gene variability contributes to the genetic component of CAD. However, there are several reasons why the present work could not be strictly considered as a replication of the original Chinese study [2]. (1) Different phenotypes: the genetic component of various CAD manifestations (MI and unstable angina) may partially differ. (2) The Chinese (Caucasian) and Chinese (Asian) populations differ significantly in their genetic background; this is particularly apparent in the frequency of investigated BDNF 66Met allele in Chinese controls (49%) by comparison with Czech controls (17%). Furthermore, the differences in haplotype structure between both ethnicities may mask causal variants within the BDNF gene or nearby. (3) Though our study involved well characterised group of MI patients, it disposed of lower statistical power to detect potential association of BDNF gene variant with CAD in comparison with the original report [2]. In the context of group size, there was a report of an inverse association (as compared with Chinese data [2]) between BDNF 66Met/Met genotype and coronary atherosclerosis in Italian females [8], based only on 12 patients with coronary artery stenosis – this size definitely does not allow to draw any valid conclusions.

In summary, the BDNF Val66Met polymorphism is not associated with myocardial infarction in Czech population. We could not, therefore, provide further positive data which would add up to the observation from Chinese population [2] suggesting that BDNF Met/Met genotype is a genetic modifier in CAD.

Acknowledgments

This study was supported by the institutional grant of the Palacky University (IGA UP Project No. LF_2010_008, Czech Republic) and, in part, by the Czech Ministry of Education, Youth and Sports (Projects MSM619859205 and ME-856). The data were presented in part at the European Atherosclerosis Society Congress, Hamburg, June 20–23, 2010.

References


Table 1

Distribution of BDNF SNP rs6265 G/A (Val66Met) genotypes and alleles in the groups of Czech patients with myocardial infarction (MI) and of healthy control subjects (controls).

<table>
<thead>
<tr>
<th>BDNF rs6265 G/A (Val66Met)</th>
<th>Czech population</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MI (N = 217)</td>
</tr>
<tr>
<td>Genotypes</td>
<td></td>
</tr>
<tr>
<td>GG</td>
<td>149 (0.687)</td>
</tr>
<tr>
<td>GA</td>
<td>59 (0.272)</td>
</tr>
<tr>
<td>AA</td>
<td>9 (0.041)</td>
</tr>
<tr>
<td>Alleles</td>
<td></td>
</tr>
<tr>
<td>G</td>
<td>357 (0.823)</td>
</tr>
<tr>
<td>A</td>
<td>77 (0.177)</td>
</tr>
<tr>
<td>Carriers</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>68 (0.313)</td>
</tr>
</tbody>
</table>

The data are given as absolute numbers with proportion in parentheses. The BDNF rs6265 G/A (Val66Met) SNP was genotyped by “TaqMan” SNP Genotyping Assay (Applied Biosystems, Assay ID C_11592758_10) according to the manufacturer’s instructions. The distribution of BDNF Val66Met genotypes conformed to Hardy–Weinberg equilibrium in both investigated groups (MI/controls, p > 0.05). * p > 0.05 for all comparisons (alleles, carriage) between the MI patients and controls.

BDNF 66Met/Met genotype and coronary atherosclerosis in Italian females [8], based only on 12 patients with coronary artery stenosis – this size definitely does not allow to draw any valid conclusions.

In summary, the BDNF Val66Met polymorphism is not associated with myocardial infarction in Czech population. We could not, therefore, provide further positive data which would add up to the observation from Chinese population [2] suggesting that BDNF Met/Met genotype is a genetic modifier in CAD.


A. Štahelová
Laboratory of Immunogenomics and Proteomics, Department of Immunology, Medical Faculty of Palacky University and Faculty Hospital, Olomouc, Czech Republic

J. Petrková
Laboratory of Immunogenomics and Proteomics, Department of Immunology, Medical Faculty of Palacky University and Faculty Hospital, Olomouc, Czech Republic

N. Motakova
Laboratory of Immunogenomics and Proteomics, Department of Immunology, Medical Faculty of Palacky University and Faculty Hospital, Olomouc, Czech Republic

M. Taborsky
Department of Internal Medicine I, Medical Faculty of Palacky University and Faculty Hospital, Olomouc, Czech Republic

F. Mrazek
Laboratory of Immunogenomics and Proteomics, Department of Immunology, Medical Faculty of Palacky University and Faculty Hospital, Olomouc, Czech Republic

M. Petrek
Laboratory of Immunogenomics and Proteomics, Department of Immunology, Medical Faculty of Palacky University, I.P. Pavlova str. 6, CZ-775 20 Olomouc, Czech Republic.

Tel.: +420 58 563 2770; fax: + 420 58 541 5116.

Div. of Clinical Biochemistry and Immunogenetics, Medical Faculty of Palacky University and Faculty Hospital, Olomouc, Czech Republic

Institute of Molecular and Translational Medicine, Medical Faculty of Palacky University and Faculty Hospital, Olomouc, Czech Republic

E-mail address: martin.petrek@fnol.cz

Available online xxxx