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Letter

The *BDNF* Val66Met polymorphism is not associated with myocardial infarction in Czech patients

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) variants 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 equalled 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 Czech (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.

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).

BDNF rs6265 G/A (Val66Met)		Czech population	
		MI (*N* = 217)	Controls (*N* = 180)
Genotypes[a]	GG	149 (0.687)	127 (0.706)
	GA	59 (0.272)	44 (0.244)
	AA	9 (0.041)	9 (0.050)
Alleles[a]	G	357 (0.823)	298 (0.828)
	A	77 (0.177)	62 (0.172)
Carriers[a]	A	68 (0.313)	53 (0.294)

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, $p > 0.05$).
[a] $p > 0.05$ for all comparisons (genotypes, alleles, carriage) between the MI patients and controls.

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.

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