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Reply to Novelli

European Journal of Human Genetics (2006) **14,** 895. doi:10.1038/sj.ejhg.5201637; published online 17 May 2006

We would like to thank Giuseppe Novelli and colleagues for their comments on our paper:[1] we have appreciated the importance of their demonstration of functional effects of intronic SNPs in the OLR1 gene.[2]

In our publication, we did not find an association with acute myocardial infarction (AMI) in 677 subjects, but observed an association of the 501G>C polymorphism with CAD severity in a survey of 350 individuals.

We have now extended that study (first set) in a new replication sample of 637 angiographically documented CAD patients (second set). Of the 637 CAD patients, 397 were with and 240 without AMI. All these subjects were consecutively recruited from the same clinical center as the first set, according to the previously described enrollment criteria. The IVS4−73C>T SNP, which is associated with the expression of full-length transcript or the LOXIN detected transcript in their study,[2] as well as the exon 4 K167N (501G>C) SNP, were genotyped.

As indicated in Table 1, no association was found in the replica set with the IVS4−73C>T or the K167N SNP ($P=0.15$, $P=0.89$, respectively). The grouped sample (first and second set) confirmed no association with either polymorphism ($P=0.29$, $P=0.84$, respectively).

Table 1 OLR1 IVS4−73C>T (a) and K167N (501G>C) (b) genotype frequencies in the replication sample

	AMI (N=397)	*AMI free (N=240)*
(a) IVS4—73C>T		
C/C	0.207	0.212
C/T	0.456	0.521
T/T	0.337	0.267
(b) K167N (501G>C)		
G/G	0.864	0.877
G/C	0.126	0.115
C/C	0.010	0.008

The association we previously observed between the K167N GG genotype frequency and CAD severity was not confirmed in the second set that included 322 patients with three, 159 patients with two, and 116 patients with one stenosed vessels, respectively ($P=0.86$). In the grouped sample, no significant association was detected ($P=0.73$).

In conclusion, we confirm no association with AMI in an extended sample; moreover, the apparent association with CAD severity was not replicated. Further studies are needed to elucidate the role of the OLR1 gene in cardiovascular disease.

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