

Role of polymorphisms of CC-chemokine receptor-5 (CCR5) gene in acute myocardial infarction and biological implications for longevity

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Supplementary Table 1

Table 1. Genotype distributions and allelic frequencies of CCR5 59029 A/G and 59353C/T promoter SNPs and CCR5 Δ 32 deletion in AMI patients and controls from Sicily. 2x2 Comparisons between the different groups with Odd Ratio (OR) and (95% Confidence interval).

Genotypes	AMI Patients (N=133)	Controls (N=136)
- 59029 A/A	118	82
A/G	9	30
G/G	6	24
- 59353 C/C	116	109
C/T	14	23
T/T	3	4
	AMI Patients (N=133)	Controls (N=136)
wt/wt	130	119
wt/ Δ 32	3	14
Δ 32/ Δ 32	0	3
Alleles (%)	AMI Patients (N=133)	Controls (N=136)
59029A	245(92.1%)	194(71.3%)
59029G	21(7.9%)	78(28.7%)
59353C	246(92.5%)	241(88.6%)
59353T	20(7.5%)	31(11.4%)
	AMI Patients (N=133)	Controls (N=136)
wt	263 (98.8%)	252 (92.6%)
Δ 32	3 (1.2%)	20 (7.4%)

All the genotypes were in HWE. The CCR5 Δ 32 genotypes were significantly differently distributed between the 2 cohorts: $p=0.005$ (by χ^2 test). We found a significantly lower frequency of the CCR5 Δ 32 genotypes (indicated as wt/ Δ 32 and Δ 32/ Δ 32) among AMI patients compared to controls; no patients were CCR5 Δ 32-homozygous compared to controls (OR=0.16, 95%CI=0.046-0.56, $p=0.003$ by Fisher's exact test). Accordingly, the CCR5 Δ 32 allele frequency shows a statistically significant difference between AMI patients and controls ($p=0.0007$, by χ^2 test with Yates' correction). In particular, the frequency of CCR5 Δ 32 deletion in controls (7.3%) was six times higher than observed in AMI patients (1.2%). So, in controls the OR was lower than in AMI patients (OR=0.14 95%CI=0.04-0.48, $p=0.0008$ by Fisher's exact test).