Myocardial infarction (MI) is a complex disease. Multiple genes and their interaction with various environmental factors influence the pathogenesis of MI that is thought to be tightly regulated by inflammatory pathways. Recent progress in genetic analysis includes the use of large-scale genome-wide association studies that have proven to be powerful tools even in the analysis of multifactorial phenotypes. However, certain genes are only sparsely represented on the available gene chips and additional candidate gene approaches are necessary. One such example is the CNR2 gene, encoding the cannabinoid receptor 2 (CB2), which has been implicated in mediating anti-inflammatory and anti-atherosclerotic effects in vivo. We therefore hypothesized that genetic variations within the CNR2 gene are associated with the development of MI or classic cardiovascular risk factors. In a large case-control study, 1,968 individuals from the German MI family study were examined with 13 single nucleotide polymorphisms (SNPs) covering CNR2 and the adjacent genes. The association of these SNPs with MI or cardiovascular risk factors, such as arterial hypertension, obesity, hypercholesterolemia and diabetes mellitus, was determined. In allelic and genotypic models, none of the SNPs
showed a significant association with MI. Separate analyses for men and women revealed no
gender-specific relationship between common genetic variations within the CNR2 gene and MI.
Moreover, no significant association between CNR2 gene variants and common cardiovascular risk
factors was observed. We therefore provide evidence in a large German population that common
polymorphisms within the CNR2 gene confer no susceptibility to MI or to cardiovascular risk factors.