DELAYED CARDIOPROTECTION INDUCED BY A NOVEL QUINAZOLINE COMPOUND MAY BE SUSTAINED BY INDUCTION OF ANGIOGENESIS AND IMPROVEMENT OF CARDIAC ENERGY METABOLISM

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We have previously demonstrated that the anilinoquinazoline derivative DMA (*4-N-(3'-N,N-dimethylphenyl)amino-6,7-dimethoxyquinazoline*) induces myocardial protection to ischemia/reperfusion injury. The present study was undertaken to examine the myocardial gene expression induced by DMA. The experimental approach was based on the screening of total myocardial RNA taken from mice treated with DMA or vehicle 24 and 48 hours before the sacrifice, and hybridized to microarrays containing 36000 coding sequences present in mouse genome (*Codelink® Bioarrays*). A total of 345 (260 up-, 85 downregulated) and 197 (110 up-, 87 downregulated) genes were found to be differentially expressed at 24 and 48 hours after treatment with DMA, respectively. Functional analysis indicated that most of these genes are involved in angiogenesis and energy metabolism. Interestingly, we could detect differential expression of genes also described as regulated in response to hypoxia. Taken together, these data demonstrate that DMA affects the expression of genes involved in biological processes of relevance to comprehend myocardial protection to ischemia/reperfusion injury.