

Despite the past development of drugs, i.e. statins, that efficiently lower plasma cholesterol levels and thereby reduce the chance of developing atherosclerotic lesions, the risk to die from a cardiovascular event remains very high. More specifically, death of myocardial infarction is still the predominant cause of death worldwide. As such, there is clear need for the development of alternative cardiovascular therapies. In this light, it is of interest to see that many associations have been found between differences in the cellular expression and/or plasma level of a specific class of small non-coding RNA molecules, the ~22 nucleotide long so-called microRNAs, and the occurrence of myocardial infarction. MicroRNAs through binding to their target transcripts negatively impact on the gene expression of a wide variety of proteins, and - in this way - are able to modulate many processes involved in the development of atherosclerotic lesions, the de-stabilization of these lesions, and the generation and recovery from a myocardial infarction. Notably, microRNAs constitute valuable drug targets as they can be readily administered to humans. Furthermore, their activity can also be easily diminished through administration of antagonizing nucleotides, i.e. antagomirs. When taking the aforementioned findings into account, one can foresee a bright future for microRNAs in cardiovascular drug development. Providing insight in the (causal) contribution of microRNAs to cardiovascular disease pathology, i.e. through basic proof-of-principle and clinical studies, will hopefully aid in a speedy development of novel microRNA-based cardiovascular therapies.

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