

Accepted Manuscript

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PII: S0378-1119(17)31086-7
DOI: <https://doi.org/10.1016/j.gene.2017.12.033>
Reference: GENE 42419
To appear in: *Gene*
Received date: 23 July 2017
Revised date: 7 November 2017
Accepted date: 18 December 2017

Please cite this article as: Lian Duan, Junyuan Hu, Xingjiang Xiong, Yongmei Liu, Jie Wang , The role of DNA methylation in coronary artery disease. The address for the corresponding author was captured as affiliation for all authors. Please check if appropriate. *Gene*(2017), <https://doi.org/10.1016/j.gene.2017.12.033>

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The Role of DNA Methylation in Coronary Artery Disease

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Abstract: Epigenetic studies have identified DNA methylation in coronary artery disease (CAD). How the critical genes interact at the cellular level to cause CAD is still unknown. The discovery of DNA methylation inspired researchers to explore relationships in genomic coding and disease phenotype. In the past two decades, there have been many findings regarding the relationship between DNA methylation and CAD development, and the DNA methylation of critical genes have been found to be significantly changed during CAD, including DNA methylation at homocysteine, Alu and long Interspersed Element 1 (LINE-1) repetitive elements. Here, we provide a brief overview of the biology and mechanisms of DNA methylation and its roles in CAD. We also discuss recent findings regarding DNA methylation of homocysteine, Alu and LINE-1 and some genes on CAD in vitro and in vivo. Finally, we provide some perspectives on DNA methylation in CAD.

Keywords: DNA methylation, CAD, homocysteine, Alu, LINE-1

Over the past few decades, coronary artery disease (CAD) has been among the leading cause of morbidity and mortality worldwide. The number of deaths due to CAD is 56 million globally[1]. CAD is caused by a variety of factors, in which atherosclerosis is the main pathological basis of CAD (Fig. 1[2]). Epigenetic factors, including DNA methylation, histone modification, chromatin remodelling and noncoding RNA regulation, have been reported to cause CAD by altering the interaction between genes and the environment[3]. Among them, DNA methylation is a key epigenetic process for CAD and its risk factors.

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