**GENETICS OF CORONARY ARTERY DISEASE**

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Susceptibility to coronary artery disease (CAD) is claimed to be 40-60% inherited, but until recently genetic risk factors predisposing to CAD have been elusive. Comprehensive prevention of CAD requires manipulation of genetic risk. The availability of microarrays of single nucleotide polymorphisms enabling genome-wide association studies (GWAS) led to the discovery of 33 genetic risk variants for CAD. Surprisingly, 23 risk variants mediate their risk through unknown mechanisms, with only 10 associating with hypertension or lipids. Thus, there are several mechanisms contributing to the pathogenesis of CAD yet to be elucidated. The first risk variant discovered by GWAS was 9p21.3 which occurs in 75% of all populations except African, with a mean increased risk of 25% per copy. Of the 33 variants for CAD, the increased risk varies from 6-92% with a mean increased risk of 18%, occurring on average in 47% of the population. The maximum number of risk alleles per individual would be 66. In the CARDIoGRAM study of 23 variants, the average per individual was 17, the minimum 7, and the maximum 37. The top 10th percentile has an odds ratio of 1.88 and the lowest percentile an odds ratio of 0.55. Routine genetic screening is unlikely until management is improved by genetic testing. Risk variants should provide patho-physiological insights and targets for novel therapy. While risk variants are less potent predictors of CAD, compared to a biomarkers, they have the advantage of not changing in one's lifetime and are unaffected by diet, gender, age or medication.