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Changes in diet are likely to reduce cardiovascular disease, but after decades of active research and heated discussion the question still remains: what is the optimal diet to achieve this elusive goal? Is a low fat, as traditionally recommended by multiple medical societies? Or a high monounsaturated fat as predicated by the Mediterranean diet? Perhaps a high polyunsaturated fat based on the cholesterol lowering effects? The right answer may be all of the above but not for everybody. A well-known phenomenon in nutrition research and practice is the dramatic variability in interindividual response to any type of dietary intervention. There are many other factors influencing response, and they include, among many others, age, sex, physical activity, alcohol, and smoking as well as genetic factors that will help to identify vulnerable populations/individuals that will be benefit from a variety of more personalized and mechanistic based dietary recommendations. This potential could and needs to be developed within the context of nutritional genomics that in conjunction with systems biology may provide the tools to achieve the holy grail of dietary prevention and therapy of cardiovascular diseases. This approach will break with the traditional public health approach of "one size fits all." The current evidence based on nutrigenetics has begun to identify subgroups of individuals who benefit more from a low fat diet, whereas others appear to benefit more from a high monounsaturated or polyunsaturated fat (PUFA) diets. Of interest is the increasing evidence showing that when it comes to cardiovascular health, n-6 and n-3 families of PUFAs interact very different with genetic variants to modulate cardiovascular risk factors. Thus, while some subgroups of individuals may be at higher risk from high consumption of PUFA n-6 [i.e., carriers of the minor allele at the APOA5-1131T>C single nucleotide polymorphism], others may greatly benefit from increased consumption of PUFA n-3 (i.e., carriers of the APOA5-1131C or the A allele at the IL1beta 6054G > A SNP). The continuous progress in Nutrigenomics will allow us to identify those persons for whom diet plays no major role in their risk of CVD as well as those persons who may benefit from specific gene-based dietary advice. However, in order to gain knowledge in this area, the overwhelming amount of genetic data being generated needs to be balanced with reliable and comprehensive phenotypic information gathered over time in very large numbers of subjects. Unfortunately, the existing longitudinal studies lack, individually, the size needed to deal with the complexity of the gene-environment interactions modulating human health and disease, nor are the statistical tools ready to deal with these complex interactions. Moreover, the evidence needs to be supported by properly designed intervention studies. Therefore, whereas it is accepted that our responses to the environment (i.e., diet) are largely determined by our genetic make up, our current knowledge is insufficient to successfully implement the practical use of genetics to predict disease or to personalize dietary advice for the prevention of diabetes and CVD. However, personalized nutrition is a valid concept and it is important to keep in mind that "the future belongs to those who prepare for it today".