Conclusions ARHL is a complex disorder, influenced by genetic, environmental/lifestyle and stochastic factors. Despite its high prevalence and the recent progress in hear- ing research, few attempts to identify genetic deter- minants of ARHL have been made. In this post- genome era, with high- throughput genotyping plat- forms now developed, a HapMap project to guide marker selection, the constructive challenge we face is to find strategies that are best suited to unravel the genetic basis of complex traits such as presbyscu- sis. The reverse genetics approach, where phenotypes are refined in relation to genetic marker data (linkage and linkage–disequilibrium analysis), although statis- tical challenges remain, may lead to the identifica- tion of susceptibility components for ARHL. On the other hand, forward genetics may also be a promis- ing approach. Isolating all the genes in the human genome, as well as identifying and cataloguing the functional variants within them in the human popula- tion, will allow assessment of the impact of genotype on phenotypic outcome of interest. Additionally, com- plementary strategies, based on functional genomics technology involving microarrays and proteomics, can be used to develop predictors of disease susceptibility based on biological pathways physiologically relevant to ARHL. Nevertheless, choices in study designs will still be the major factor in the probability of suc- cess. Determination of the genetic variants involved in ARHL should provide new insights into the dis- order mechanism, which may uncover new leads for pharmaceutical intervention and could result in the development of screening kits to identify individuals at increased risk.

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