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Much of the genetics of human aging is determined by the structural and functional pathways that regulate processes such as cellular senescence, DNA repair, immune function, and the circadian clock. We are very far from an understanding of how the genetic network that regulates these pathways interacts to determine age and healthspan. Genetics is challenging to approach because of the multifactorial nature of these processes. Moreover, the genetic causes of age and disease are often different from the genetic causes of the pathological consequences of age, and they arise at different times in life. To gain traction on the genetic causes of aging, it will be necessary to focus on the multifactorial aspects of these pathways, the common features of the genetic backgrounds that determine them, and how both of these are influenced by other genetic and environmental factors. Identification of a missense mutation in the type VI collagen gene in a patient with corneal dystrophy. A systematic approach to the molecular diagnosis of a patient with clinical signs of a corneal dystrophy was used to diagnose ocular disease. This approach, combined with a search for mutations in the known genes for major types of inherited keratopathies, was used to identify a missense mutation in the type VI collagen gene in a patient with corneal dystrophy. To establish the genetic cause of the corneal dystrophy, we applied a systematic approach to the clinical presentation, pattern of inheritance, tissue distribution, and linkage to known mutations of major forms of inherited ocular disease. Nucleotide sequence analysis of the gene encoding the type VI collagen chain, COL6A3, was used to identify a missense mutation in the proband. Polymerase chain reaction amplification followed by nucleotide sequence analysis of this region of the gene was used to identify the mutation. Mutations in the type VI collagen gene were not identified in 13 patients with corneal dystrophy or with an autosomal dominant form of macular corneal dystrophy. A missense mutation was identified in the proband. This patient has typical clinical signs of corneal dystrophy with peripheral punctate opacities in the stroma, but the opacity occurs in only one eye. Linkage to the locus for corneal dystrophy is excluded. Other corne 6d1f23a050

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