Keratin type I binds keratin type II

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references


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https://reactome.org
The first step in keratin assembly is the formation of coiled-coil heterodimers consisting of an acidic type I keratin and a basic or neutral type II keratin (Coulombe & Fuchs 1990, Hatzfeld & Webber 1990, Steinert 1990). In humans, the type I keratins are K9-24, K25-28, which are specific to the inner root sheath of hair, and the hair-specific keratins K31-K38. The type II keratins are K1-8, K71-80 and K81-86 (Bragula & Homberger 2009). Binding between dimer pairs is remarkably strong and can form even in 9M urea (Coulombe & Fuchs 1990). The ~50 nm long middle rod region of keratin protein aligns with its partner in a parallel orientation (Pauling & Corey 1953, Hanukoglu & Fuchs 1983, Parry et al. 1985, Steinert et al. 1994). The rod region is sufficient to form a heterodimer and subsequent tetramers, but the assembly of keratin filaments requires the non-helical head and tail regions (Wilson et al. 1992). The assembly of rod domain heterodimers has asymmetric salt bridges, hydrogen bonds and hydrophobic contacts, and surface of the heterodimer interface exhibits a notable charge polarization (Lee et al. 2012).

In vitro, virtually any type I keratin can dimerize with any type II keratin, leading to the formation of 10-nm long filaments (Franke et al. 1983, Hatzfeld et al. 1987). In vivo, the composition of keratin heterodimers is probably determined by expression. Differing keratin combinations are not characteristic of entire tissues, but probably confer particular functional properties to cells and tissue regions (Bragulla & Homberger 2009). Certain combinations are characteristic of a cell type, e.g. K18/K8 in simple epithelia. At least some keratins can be replaced with no loss of functionality of the keratin filament, e.g. K1/K10, K1/K9, K2/K9, K2/K10 in epithelia (Coulombe & Omary 2002). Suprabasal cells of stratified epithelia express different keratin pairs in different tissues, e.g. skin epidermis predominantly expresses K1/K10, the anterior corneal epithelium produces K3/K12, esophageal epithelium produces K4/K13 (Eichner & Kahn 1990) while hyperproliferative suprabasal cells are characterized by K6/K16 (Sun 2006).

**Literature references**


**Editions**

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