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Biology of hyaluronan: insights from genetic disorders of hyaluronan metabolism

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Abstract

Hyaluronan is a rapidly turned over component of the vertebrate extracellular matrix. Its levels are determined, in part, by the hyaluronan synthases, HAS1, HAS2, and HAS3, and three hyaluronidases, HYAL1, HYAL2 and HYAL3. Hyaluronan binding proteins also regulate hyaluronan levels although their involvement is less well understood. To date, two genetic disorders of hyaluronan metabolism have been reported in humans: HYAL1 deficiency (Mucopolysaccharidosis IX) in four individuals with joint pathology as the predominant phenotypic finding and HAS2 deficiency in a single person having cardiac pathology. However, inherited disorders and induced mutations affecting hyaluronan metabolism have been characterized in other species. Overproduction of hyaluronan by HAS2 results in skin folding and thickening in Sharpei dogs and the naked mole rat, whereas a complete deficiency of HAS2 causes embryonic lethality in mice due to cardiac defects. Deficiency of murine HAS1 and

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