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Growth Hormone Regulated Gene Expression: A Brief Note

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Description

Growth Hormone (GH) exerts a various array of physiological actions that has major roles in growth and metabolism. the first somatomedin hypothesis of Salmon and Daugh each day proposed that a serum factor controlled by GH, or somatomedin, was directly liable for stimulating sulfate incorporation by cartilage, and a number of other lines of investigation have since identified Insulin-like growth factor 1 (IGF-1) because the principal somatomedin liable for this effect. In support of the somatomedin hypothesis, many of the characteristic phenotypic features of GH deficiency are indeed recapitulated with mutations of the gene encoding IGF-1 in both rodents and humans although there are now a way better recognition of the role of local tissue-derived IGF-1 production, instead of strictly endocrine-acting serum IGF-1, on mediating many of the consequences of GH.

GH treatment restores growth velocity in children with GH deficiency, whereas replacement therapy in adults improves body composition, exercise capacity, skeletal integrity, and quality of life measures. GH has been alleged to have an antiaging effect, but diminished signaling of the GH-IGF-1 axis has been related to longevity across many species, intimating deleterious effects from this pathway. Indeed this phenomenon extends even to humans and has been attributed to reduced risks in 2 major contributors to mortality, cancer and diabetes. It follows that better understanding of the molecular mechanisms underlying GH action has important implications to human health and disease. Since the somatomedin hypothesis of somatotropin (GH) action on growth was first formulated in 1957, the key roles of both GH and insulin-like growth factor-I (IGF-I) in human growth are extended to incorporate important effects on tissue maintenance and repair [1].

Newer observations have revealed that this pathway features a negative side, because it has been implicated as a possible contributor to the event of several human cancers and has been linked to diminished lifespan in experimental animals. From the past studies, much has been learned about the physiology of the GH insulin-like growth factor-I (IGF-I) growth axis [2]. The central roles of this pathway in normal pre- and postnatal growth in mammals are extended to incorporate actions on tissue maintenance, regeneration, and repair within the adult [3,4]. Alongside a clearer definition of positive actions of GH and IGF I, an appreciation of their potentially harmful effects has been established. The negative roles of excessive GH on glucose metabolism and on cardiovascular function in acromegaly are long known [5], as have the pathogenic effects of both GH and IGF-I in proliferative diabetic retinopathy. GH and IGF-I actions even have been linked in experimental animals to accelerated aging, and IGF-I has been found in epidemiological studies in humans and in experimental models in animals to contribute too many sorts of cancer. Taken together, these studies emphasize the importance of understanding the essential biochemistry and biology of the GH-IGF-I pathway so as to modulate its effects on human health and disease.

The broad physiological actions of GH and IGF-1 on growth and metabolism and increasing to aging support the detailed study of mechanisms of GH gene regulation [6].

Much has been learned about the signal transduction pathways that are activated by GH, yet there remain key inquiries to address during this field and experiments assessing events in chromatin are underrepresented [7].

Tissue specificity in mechanisms of gene regulation has been highlighted during this review and may be manifest at many for his or her study [10]. Although IGF-1 is that the most crucial effector of GH and, hence, the foremost studied, characterization of its key regulatory domains remains incomplete, with virtually no current insight about the domains that function in humans or those active in tissues aside from liver.

Conclusion

GH and IGF-I play multiple roles in human and animal physiology

and disease. They are essential for normal preand postnatal growth and are key factors in normal tissue repair and regeneration throughout the lifespan. Conversely, actions of GH and IGF-I are linked to accelerated aging and to cancer development and metastasis. A more comprehensive understanding of the essential biochemistry and biology of GH and IGF-I actions is required to plan ways to separate therapeutically useful from deleterious effects of this potent hormonal signaling system. Finally, strategies to modulate signaling pathways to focus on specific genes would be desired to permit enhancement of selected GH effects while not inducing any harm.

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