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The ability of GH, via its mediator peptide IGF-1, to influence regulation of cellular growth has been the focus of much interest in recent years. In this review, we will explore the association between GH and cancer. Available experimental data support the suggestion that GH/IGF-1 status may influence neoplastic tissue growth. Extensive epidemiological data exist that also support a link between GH/IGF-1 status and cancer risk. Epidemiological studies of patients with acromegaly indicate an increased risk of colorectal cancer, although risk of other cancers is unproven, and a long-term follow-up study of children deficient in GH treated with pituitary-derived GH has indicated an increased risk of colorectal cancer. Conversely, extensive studies of the outcome of GH replacement in childhood cancer survivors show no evidence of an excess of de novo cancers, and more recent surveillance of children and adults treated with GH has revealed no increase in observed cancer risk. However, given the experimental evidence that indicates GH/IGF-1 provides an anti-apoptotic environment that may favour survival of genetically damaged cells, longer-term surveillance is necessary; over many years, even a subtle alteration in the environmental milieu in this direction, although not inducing cancer, could result in acceleration of carcinogenesis. Finally, even if GH/IGF-1 therapy does result in a small increase in cancer risk compared to untreated patients with GH deficiency, it is likely that the eventual risk will be the same as the general population. Such a restoration to normality will need to be balanced against the known morbidity of untreated GH deficiency.

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