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Advances in Brief

Reduced Growth of Human Breast Cancer Xenografts in Hosts Homozygous for the *lit* Mutation[1]

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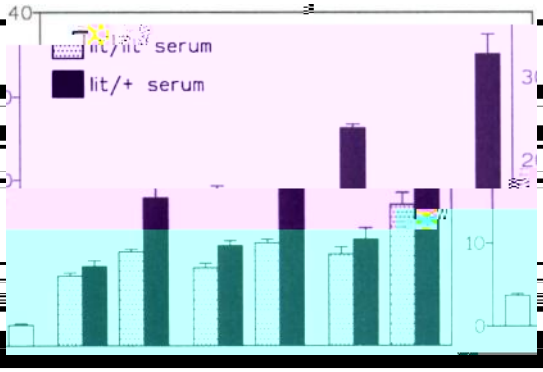
Abstract

Insulin-like growth factor I (IGF-I) is a potent breast cancer mitogen. Growth hormone (GH) up-regulates hepatic *IGF-I* gene expression and circulating IGF-I level. Tissue IGF bioactivity is influenced not only by circulating IGF-I and IGF-II levels but also by autocrine and paracrine

breast cancer behavior is weaker (4, 5, 7), but this is not unexpected given the anticipated influence of potentially confounding variables such as treatment.

GH is a weak mitogen for human breast cancer cells relative to other peptide growth factors (11). On the other hand, significant stimulation of *in vitro* breast cancer cell proliferation by nanomolar

***In Vitro* Cell Proliferation.** MCF-7 cells were obtained from the American Type Tissue Culture Collection (Rockville, Maryland). Stock cultures were



hormone receptor gene to which the *lit* mutation has been mapped are involved in regulation of IGF physiology. Although loss-of-function mutations such as *lit* represent an extreme example, polymorphic variation of various genes involved in regulating host IGF physiology

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