Reduced Growth of Human Breast Cancer Xenografts in Hosts Homozygous for the *lit* Mutation¹

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Insulin-like growth factor I (IGF-I) is a potent breast cancer mitogen. Growth hormone (GH) up-regulates hepatic IGF-I gene expression and irculating IGF-I level. Tissue IGF bioactivity is influenced not only by irculating 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 <i>in vitro</i> breast cancer cell proliferation by nanomolar
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In Vitro Cell Proliferation. MCF-7 cells were obtained from the American Type Tissue Culture Collection (Rockville, Maryland). Stock cultures were	1000	T
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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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