

Stephen Hursting, Ph. D., cancer research expert, comments:

"If you have high insulin and high IGF-1, you're going to drive a signal through receptors that activates a growth and survival pathway. That's exactly what cancer cells capitalize on."

Here is more from Dr. Hursting:

Cancer Prevention Studies in p53-Deficient Mice

SD Hursting, SN Perkins, LA Donehower, BJ Davis.

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Future progress in mechanism-based cancer prevention research may be facilitated by animal models displaying specific genetic susceptibilities for cancer, such as mice deficient in 1 (+/-) or both (-/-) alleles [Alternative forms of the same gene, e.g. the dominant and the recessive genes for hair color] of the *p53* tumor suppressor gene. We observed in *p53*^{-/-} mice that calorie restriction (CR) increased the latency of spontaneous tumor development (mostly lymphomas) by approximately 75%, decreased serum insulin-like growth factor-1 (IGF-1) and leptin levels, slowed thymocyte [a lymphocyte, produced in the thymus, that develops into a T cell] cell cycle traverse, and induced apoptosis [programmed cell death] in immature thymocytes.

In *p53*^{+/-} mice, CR and a 1 d/wk fast each delayed spontaneous tumor development (a mix of lymphomas, sarcomas, and epithelial tumors) and decreased serum IGF-1 and leptin [A peptide hormone produced in adipose tissue; it plays a role in regulating appetite and metabolism] levels, even when begun late in life. In *p53*^{+/-} *Wnt-1* transgenic mice, a mammary tumor model, the same interventions increased mammary tumor latency and reduced mean serum IGF-1 and leptin levels to <50 % of those of control mice. We capitalized on the susceptibility of *p53*^{+/-} mice to chronic, low-dose aromatic amine—induced bladder carcinogenesis to develop a useful model for evaluating bladder cancer prevention approaches. These examples clearly indicate that mice with specific (and humanlike) genetic susceptibilities for cancer are powerful models for testing interventions that may inhibit carcinogenesis in humans.

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