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A human relevent rat model of breast cancer

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Because women experience a bewildering array of chemicals, foods and lifestyles, only profound effects on preventing or promoting breast cancer are detectible in human studies. Subtle or delayed effects can be detected in animal models. Mammary tumors in ACI rats share important similarities with the majority of human breast cancers. The link between life time estrogen exposure and breast cancer risk in humans is well established. A high percentage of human breast cancers express ER, are stimulated to grow by the addition of exogenous estrogen, and respond to the antiestrogen tamoxifen. The ACI rat is the only rodent model in which estrogen-sensitive tumors are induced by estrogen. The ACI.COP-Ept2 substrain, derived from the ACI rat, develops mammary tumors similar to those of the ACI rat, but with reduced pituitary hyperplasia. We show that estrogen-induced mammary tumors in ACI.COP-Ept2 express ERα and respond to tamoxifen. Furthermore, tumors express ERβ, progesterone receptor and Her2/neu. The average latency was 183±6 days (n=24) and average tumor burden 1,107±415 mm3. The similarities of ACI.COP-Ept2 tumors to human breast cancers make this a valuable model for determining which of the myriad of lifestyle and diet choices reportedly protecting women from breast cancer actually reduce cancer incidence.