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Lifestyle, IGF-I and prostate cancer

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In previous studies we reported that a low-fat, high-fiber diet and exercise program altered serum factors that resulted in reduced growth and increased apoptosis of serum- stimulated, androgen-dependent LNCaP and LAPC-4 cell lines. In the present study we investigated the role of IGF-I in mediating these responses by comparing pre and post intervention serum stimulation of LNCaP and PC-3 (androgen independent cells). Growth was assessed by the CellTiter 96AQ, MTS assay and apoptosis by the Cell Death Detection ELISA Plus assay. A low-fat, high-fiber diet and daily exercise intervention reduced growth of LNCaP cells by 43.6% and increased apoptosis by 354%. When the IGF-I receptor blocker aIR3 was added to the pre- and post-intervention serum, growth of LNCaP cells was reduced to the same level (~41% of pre) and apoptosis increased to the same level (~425% of pre). When IGF-I (60 ng/mL) was added back to the post- intervention serum, the reduction in growth and increase in apoptosis were eliminated. In androgen independent PC-3 cells, diet and exercise intervention only reduced growth by 26.4%. When aIR3 was added to the pre samples, growth was reduced by only 32.8%. Diet and exercise increased apoptosis in the PC-3 cells by only 31.8% and when aIR3 was added to the pre samples apoptosis was increased by only 214.6%. When the PI3 Kinase blocker (LY294002) or the p38 MAP Kinase blocker (SB202190) were added to the pre serum-stimulated LNCaP cells there was an additional 10% reduction in growth compared to the aIR3 results. With the two blockers apoptosis was increased by an additional 299.6% (LY294002) and 308.3% (SB202190) compared to the aIR3 results. These data indicate that in early stage, androgen dependent PCa, diet and exercise intervention significantly reduces growth and induces apoptosis of LNCaP cells primarily by reducing serum IGF-I. In addition, some other serum factor(s), not responsive to diet and exercise, stimulate growth and especially suppress apoptosis in the LNCaP cells. Diet and exercise is far less effective in reducing growth and inducing apoptosis in androgen independent PC-3 cells.

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Role of positive family history in the development of gastric cancer: a meta-analysis study

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Background: The role of family history in gastric cancer (GC) has been showed by several studies. In this study we aim at doing a meta-analysis of case-control studies evaluating the rate of positive family history of GC in patients versus controls.

Methods: A comprehensive English literature search was performed using PubMed from 1966 to September 2005 based on the keywords: Gastric, stomach, cancer, family history, and risk factor. 120 potential articles were reviewed, and review articles on relevant topic were complementarily scrutinized. Only case-control trials comparing the rate of positive family history in patients with gastric cancer versus non-cancer controls were included. All articles retrieved were appraised critically. Meta-analysis of pooled odd ratio (fixed model) was performed by Review Manager 4.2.

Results: Nine case control studies were included (3 from Europe, 3 from Japan, and one from each of North America, Turkey and Taiwan). One study was excluded after analysis as it had an out of range result (OR: 10.3) so the meta-analysis included 8 studies with 2494 patients in GC group and 6628 in control group. 27% of the patients and 15% of the controls had at least one relative with GC ($P<0.0001$). Odd Ratio to have a positive family history for patients with GC to the controls was 1.99 (95% CI: 1.82-2.17) (figure 1). By considering positive history only in the first-degree relatives 28.2% of the patients and 13.8% of the controls had at least one relative with GC ($P = 0.0062$). Odd Ratio to have a positive family history for patients with GC to the controls was 2.16 (95% CI: 1.93-2.42).

Conclusions: It seems that patients with gastric cancer have a double chance of having at least a case of GC in family. This is even more if this positive family history is in the first-degree relatives.

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Seasonal occurrence of spontaneous mammary tumors at mice C3H/He

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A well-studied model of neoplastic transformation and progression is the breast cancer system of the C3H mouse. Spontaneous and transplanted tumors are described. Nevertheless the moment of occurrence of spontaneous tumors is not adequately studied. The major focus is on a experimental approaches to the study of mechanisms and crucial moment of a rise of spontaneous mammary tumors of C3H/He mice. Change of a level of estrogens receptors is studied also. The results obtained to date suggest that tumor cells may originate in mice shortly after pairing. This moment is calculated from experimental data. Both the originating of tumors and a breeding equally depends on seasons (daylight hours). Obtained data confirm a greater role of steroid hormones for initialization of formation of tumors. Probably hormonal influence is a final stage of a beginning tumorogenesis. Steroid hormones do not act as carcinogens. They play a role only as regulators of a transcription. Hormones change a state of cells. Where the formalized state of a cell is history their gene transcription and a pattern gene transcription at present. Experience Bittner is successfully repeated. Synthetical steroid hormones were as the "factor of milk". No histological feature has been found which might explain the variation in levels of estrogen receptors in mice mammary tumors. Growth of transplated ER-positive mammary tumors was modulated by oestrus cycle of mice. Emphasis is given to common mechanisms of action steroids in mice or/and human as a guide to therapy and prevention in men. Ability of tumoral cells to adapt for change of a surrounding microenvironment answers on a question on a paradoxicality of a hormone therapy. Series of parameters of a tumor such as the invasiveness,