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| **Comparisons of Selected Characteristics of Analytic Epidemiologic Studies Reporting Investigating γ-Tocopherol with some potential biomarkers** | | | | |
| **Study** | **Population/Subjects** | **Biomarker** | **Results** | **Comments** |
| Robert V. Cooney,  et al, 2008  (prostate cancer) | 657 men serving as controls | **α-T** | r= -0.40 | All baseline serum levels  γ-T would increase inflammation |
| **Urinary 15-isprostane F2t level** | r= 0.14 |
| **CRP** | r= 0.13 |
| **Smoking** | Smokers: non-smokers  Mean serum level (2.3-1.9μg/ml) |
| **25-OH Vitamin D** | r= 0.23 |
| Stephanie J. W et al. 2012 | Controls:824 (age 55-74) (from Nested case-control study within the PLCO Screening Trial) | **α-T** | r= -0.24 | all baseline serum level  (Elevated risk of prostate cancer with increased serum γ-T level, but not statistically significant) |
| **α-T (adjusted for serum total cholesterol)** | r= -0.39 |
| **VE intake (diet), mg/day** | r= -0.06 |
| **VE intake (supplement), mg/day** | User : Nonuser serum level  3.5: 1.4 mg/L |
| **VE intake (diet &supplement)** | r= -0.54 |
| **α-T: γ- molar ratio** | r= -0.88 |
| **β-Carotene** | r= -0.22 |
| **Cholesterol** | r= 0.29 |
| **BMI** | r= 0.19 |
| Weiwen Chai et. al, 2012(prostate cancer) | Serum samples coming from 92 adults(In general health but with a history of at least one pathology- confirmed adenomatous colorectal polyp within the past 36mo) | **25-OH Vitamin D2** | r= - 0.31 | all baseline serum level |
| **α-T** | r= - 0.10 |
| **leptin** | r= 0.24 |
| Heather Brady  et al. 2007 | 257 infants in a cohort study | **ETS(environment Tobacco Smoke)** |  |  |
| **Plasma lipid concentration** |  |
| Weiwen Chai et al. 2010 | 180 women | **BMI** | r: Normal-Obese(1.5-2.0) |  |
| Geoffrey C Kab et al. 2009 | 5450 postmenopausal women( randomly chosen from a clinical trial and an observational study) | **β-Carotene** | r=- 0.30 | all baseline serum level  (Elevated risk of breast cancer  with increased serum γ-T level, but not statistically significant) |
| **β-Cryptoxanthin** | r=- 0.16 |
| **Lutein + zeaxanthin** | r=- 0.09 |
| **α- Carotene** | r=- 0.25 |
| Emily White et al. 2001 | 1047 of postmenopausal **women** sampled from a clinical trial | **α-T** | 0.69\* | all baseline serum level  \*Pearson partial correlations adjusted for age, ethnicity, cholesterol, triglycerides,  and body mass index (BMI) (*n=* 1047).  \*\*Restricted to those with no supplemental vitamin E intake (including multivitamins) (*n* =599).  \*\*\*Adjusted for age, ethnicity and BMI only  \*\*\*\*Nutrients from diet adjusted for energy intake  ® Ratio of serum concentration in category to concentration in reference category (see Methods),  adjusted for age, ethnicity, cholesterol, triglycerides, and (BMI). |
| **Total cholesterol\*\*\*** | 0.23\*, 0.26\*\* |
| **HDL-C\*\*\*** | -0.13\*, -0.09\*\* |
| **LDL-C\*\*\*** | 0.21\*, 0.21\*\* |
| **Retinol** | -0.16\*, -0.13\*\* |
| **α- Carotene** | -0.14\*, -0.14\*\* |
| **β-Carotene** | -0.30\*, -0.19\*\* |
| **β –Cryptoxanthin** | -0.19\*, -0.14\*\* |
| **Lutein + zeaxanthin** | -0.07\*, -0.10\*\* |
| **Supplemental vitamin E** | - 0.54\* |
| **Supplemental vitamin C** | -0.18\*, -0.10\*\* |
| **γ-T from food\*\*\*\*** | 0.16\*, 0.113\*\* |
| **Total fat\*\*\*\*** | 0.17\*, 0.14\*\* |
| **Total carbohydrates\*\*\*\*** | -0.11\*, -0.10\*\* |
| **Dietary fiber\*\*\*\*** | -0.13\*, -0.12\*\* |
| **Cholesterol\*\*\*\*** | 0.05\*, -0.03\*\* |
| **Servings of fried foods** | -0.13\*, 0.11\*\* |
| **Servings of table fats** | 0.09\*, 0.11\*\* |
| **BMI** | 0.22®, 0.27\*\* |
| **Age** | 0.80®, 0.90\*\* |
| **Annual income** | 0.89®, 1.01\*\* |
| **Smoking** | 1.05®, 0.99\*\* |
| **Alcohol** | 0.75®, 0.79\*\* |
| **Episodes of moderate or strenuous activity/week** | 0.89®, 0.88\*\* |
| **Hormone replacement therapy** | 0.78®, 0.89\*\* |
| **Supplemental vitamin E intake** | 0.28® |
| Weiping Yu et al. 2009 | Immune incompetent Nu/Nu female BALB/c mice at 6wk of age, injected subcutaneously with a total of 2\*106 MDA-MB-231-GFP human breast cancer cells, Treated with different supplemented vitamin E forms.  In this study the researchers proved thatα-T not only failed to exhibit anticancer properties but also **reduced** anticancer diets actions of γ-T. (Based on tumor volume and cell apoptosis) | | |  |
| A.A Shamim et al. 2013 | A systematic sample of 285 women in early pregnancy selected from a biochemical sub-study. | **Hb concentration (g/l)** | -0.121, -0.142 | Two methods of linear regression, **Model 1**, all seven micronutrients adjusted for an interaction term of vitamin B12 and Zn found to be the only one significant (P <0. 05) among all possible first-order micronutrient interactions;  **Model 2**: additionally adjusted for gestational age (weeks), mid-upper arm circumference (<21.5 cm), inflammation status (four stages) and season |
| Andrzej Sobczak et al. 2004 | 117 volunteers (61 women and 56 men) aged 19–60 (mean age 40*.*3±11), ethnically homogenous, (Sosnowiec, Poland), with no history of coronary heart disease, diabetes and liver disease | **Passive smoking** | r= - 0.190 | In this study the researchers use cotinineas exposure to smoking |
| **Active smoking** | r= 0.346 |
| Jean-Marc Zingg et al. 2013 | Mice | Gamma-T activates gene clusters that reduce lymphocyte proliferation and increase apoptosis and inflammation. | | |
| Tomono Uchida et al. 2013 | Male Wistar rats | Alpha-tocopherol intake did not influence depletion of c-tocopherol and tocotrienol in various tissues as well as excretion of their metabolites into urine, and thus dietary a-tocopherol decreases the tissue concentration of the other isoforms of vitamin E mainly because of the higher affinity of a-TTP for a-tocopherol. | | |
| Adam Prokopowicz et al. 2013 | 340 healthy men (then employed at the Mine and Metallurgial Plant in southern Poland | **lead** | Decrease as the dose of exposure to lead increase | |
| Morimoto Y et al. 2013 | A total of 215,251 men and women of ages 45 to 75 years were included at baseline by mailing a FFQ | Circulating tocopherols, at levels likely reflecting adequate dietary intakes, may be protective against NHL (Non-Hodgkin Lymphoma), whereas higher intakes from supplementation may not be beneficial.  A U-sharp association between tocopherols and NHL. | | |
| Yoshikawa et al. 2005 | A total of 13 healthy adult male volunteers (gamma-T group: 2 g-T capsules each containing 186.5 mg gamma-T and 5mg alpha-T for 28days; control group: only 5 mg d-alpha-T/day) | **Plasma gamma-T** | Increased markedly during administration | |
| **plasma g-CEHC** | Increased markedly during administration | |
| **plasma a-tocopherol** | Decreased during administration | |
| **plasma concentration of a-CEHC** | Decreased significantly | |
| **Urinary excretion of a-CEHC** | Increased in the gamma-T group | |
| **Urinary sodium secretion** | Significantly increase at 1 week after administration | |
| Yeum, K,J et al 1999 | 56 American Caucasian, 25 Chinese and 53 Korean | American serum alpha-T were significantly higher (P<0.005) than those of Chinese, whereas gamma-T were significantly lower (P<0.001) than those of Chinese | | |
| Klein EA et al 2011 | A total of 35,533 men from 427 study sites in the United States, Canada, and Puerto Rico, randomized clinical trial | Supplementation of all alpha-T (400 IU) caused a 50% decrease in the median plasma gamma-T level | | Dietary supplementation with vitamin E significantly increased the risk of prostate cancer among healthy men. |
| Wolf George, 2006 | Review | The reduction in plasma gamma-T during enhanced intake of alpha-T can be explained by the more rapid metabolism of gamma-T occurring when alpha-T intake is increased. | | |
| Ward, N. C et al. 2007 | 58 individuals with type 2 diabetes(double-blind, placebo controlled trial) 500mg alpha-T/day or 500mg mixed tocopherol (60% gamma-T)/day for 6 weeks | The study has demonstrated a significant increase in BP, pulse pressure and HR following supplementation with either alpha-tocopherol or a mixed tocopherol supplement, rich in gamma-tocopherol (60%). | | |
| Q Jiang et al. 2013 | Twenty-four 2-month-old male Fischer 344 rats | Gamma-T supplementation inhibits protein nitration, attenuates inflammation-induced ascorbate oxidation, and generally spares vitamin C in the absence of an effect on alpha-T | | |
| Block et al, 2001 | 116 men aged 35–72 years, Washington County, Maryland, 1989 | **fruit/vegetable consumption** | Spearman correlations Around -0.25 (including unadjusted & adjusted correlations on age/education…) | For alpha-T, the spearman correlations around 0.05 for unadjusted &adjusted… |
| Huang et al, 2003 | 184 adult nonsmokers (two group: placebo or alpha-tocopherol) | **Serum gamma-tocopherol** | Decreased about 60% | Supplementation of alpha-T also decreases delta-T significantly.  Potential health benefits of alpha-tocopherol supplements may be offset by deleterious changes in the bioavailability of other forms of tocopherol and tocotrienol, which might in part account for the null effects of alpha-tocopherol supplementation in most prevention trials of cardiovascular disease and cancer |
| Palan et al. 2005 | Ninety-eight normal non-smoker women (non-fasting venous blood. | **Menopause and hormone replacement therapy (HRT)** | alpha-T increased  gamma-T decreased |  |
| Vasankari et al. 2004 | 104 subjects with CHD and hyper- cholesterolaemia to receive either atorvastatin or simvastatin treatment for 52 weeks | **Statin therapy** | Statin therapy Both alpha-T and gamma-T serum levels decreased (varied on the time period) | |
| Lemcke-Norojarvi et al. 2001 | 46 female healthy veterinary students at the Swedish University of Agricultural Sciences, Uppsala, volunteered (three groups: linola, corn and sesame oil) for 4 weeks | **Corn and sesame oils** | Serum gamma-T increased significantly, and alpha-T/gamma-T decreased in all three groups | Alpha-T did not change during the diet period in any of the three groups  Alpha/Gamma-T varies in three kinds of oils, but basically gamma-T has a bigger advantage |
| Ascherio et al. 1992 | 121 Men- from a sub-sample drawn from a cohort study  186 Women – similarly selected from a Nurses’ Health prospective study | **Total vitamin E intake** | Plasma gamma-T  r = -0.51 in men &  r = -0.42 in women | Plasma concentration of alpha-T is positively associated with the total vitamin E intake |
| Chiu et al. 2009 | 46 smokers and 40 non-smokers  (Cross-sectional study) | Gamma-T for smokers:1.2±0.4, non-smokers: 1.4±0.7 | |  |
| Taylor et al. 2003 | 1072 case patients with incident esophageal squamous cell carcinoma (ESCC), gastric cardia cancer (GCC), or gastric noncardia cancer (GNCC) and in 1053 control subjects  (Case–cohort design) | Serum gamma-tocopherol level was not associated with the incidence of any of these cancers | | The relative risks for comparisons of the highest to the lowest quartiles of serum 􏰆-tocopherol were 0.63 (95% confidence interval [CI] = 0.44 to 0.91) for ESCC, 0.84 (95% CI = 0.55 to 1.26) for GCC, and 2.05 (95% CI = 0.89 to 4.75) for GNCC. |
| Talegawkar et al. 2007 | A subset of participants (n = 499) from the JHS cohort (n = 5302) were selected for the JHS Diet and Physical Activity Sub-Study (DPASS) | **Intake of gamma-T** | Not associated | Total (diet + supplement) intake of alpha-tocopherol was associated with its corresponding measure in serum |
| **Vitamin E supplement** | Negatively associated |
| **BMI, serum total cholesterol** | Positively associated |
| **High school or GED (General Educational Development) completion** | Negatively associated |
| Traber et al. 2005 | Mice | Alpha-T modulates Cyp3a expression, increases g-CEHC production, and limits tissue g-tocopherol accumulation in mice fed high gamma-tocopherol diets | | |
| Wang et al. 2012 | 40 (40–70-year-old), overweight/obese [body mass index (BMI) 25–39.9 kg/m2], nonsmoking postmenopausal women without CVD and other inflammatory diseases.  (A cross-sectional study ) | Plasma TAC (total antioxidant capacity) determined by VCEAC (vitamin C equivalent antioxidant capacity) was positively associated with intakes of γ-tocopherol | | Alpha-T had a opposite correlation with TAC determined by VCEAC compared to gamma-T |
| Talegawkar et al. 2009 | A subset of participants (n = 420) from the JHS cohort (n = 5301) was selected for the JHS DPASS. | Negative association between Total antioxidant performance (TAC) and serum gamma-T | | Serum alpha-T had positive association with TAC |
| Sundl et al. 2004 | Five healthy volunteers aged 24 to 29 years, three females, two males (received a daily supplement of 1,000 IU of RRR α-tocopherol for 4 days) | Plasma alpha-tocopherol concentrations increased significantly, as expected. They leveled off within 48 h.  Gamma-tocopherol concentrations decreased from 6 h onward and leveled off at 24 h.  Ratios of plasma alpha-/gamma-tocopherol decreased and leveled off within 48 h. | | The study suggested that long-term vitamin E supplementation exceeding 2 days does not further decrease γ-tocopherol concentrations. |
| Stone et al. 2004 | **Animal models**  (Alpha-T or gamma-T, half of each group received normal Fe or 8-times Fe)  **SW 480 human cell line** | Both alpha- and gamma-tocopherol unregulated PPAR-γ, mRNA and protein expression.  Gamma-tocopherol was, however, found to be a better enhancer of PPAR-γexpression than alpha-tocopherol at the concentrations tested. | | Dietary Fe levels did not influence tocopherol levels in plasma, liver, or feces. For colonocytes, high dietary Fe decreased tocopherol levels. |
| Smith et al. 2003 | Eleven subjects with End-Stage Renal Disease | **Daily vitamin E supplementation**  **(400 IU *RRR*- alpha-tocopherol).** | Gamma-T decreased  ( from 2.8 ± 0.3 to 1.7 ± 0.2 μM, *P* = 0.001) | Plasma alpha- tocopherol concentrations (from 18 ± 0.5 to 31 ± 1.7 μM, *P* < 0.0001)  Serum alpha-CEHC increased 10-fold (from 68 ± 3 to 771 ± 175 μM (*P* < 0.0001).  Plasma ascorbic acid, Plasma IL-6, CRP, TNF-α, and free F2-isoprostane concentrations were elevated. |
| Gamma-CEHC increased  ( from 837 ± 164 to 1136 ± 230 μM (*P* = 0.008). |
| Sinha et al. 1993 | 65 nonsmoking male volunteers aged 30-59 year-old | **Vitamin E intake** | Negative correlation (r= -0.33, p<0.007) | Alcohol intake decreased gamma-T.  Observed positive correlation between vitamin E intake and alpha-T. |
| Dietrich et al. 2003 | Baseline plasma data of 159 subjects (36 nonsmokers, 40 passive smokers, and 83 smokers). | **Plasma gamma-T** for Nonsmokers: 6.5 μmol/L  Smokers: 7.8 μmol/L  Passive smokers: 7.8 μmol/L | | Alpha-T has no statistical association with smoking status in this study. |
| Ford et al. 2006 | Data from 4087 adults aged >=20 y who participated in the National Health and Nutrition Examination Survey (1999–2000) | No significant difference of gamma-T among varied races and sex. | | Concentrations of alpha-tocopherol increased significantly (*P* for trend < 0.001) with age and were significantly (*P* < 0.015) lower in men than in women. African Americans and Mexican Americans had significantly (*P* < 0.001) lower concentrations of alpha -tocopherol than did whites. |
| Ford et al. 2007 | Data for 1289 participants without self-reported diabetes who were aged $20 years in the National Health and Nutrition Examination Survey (1999–2000). | **Glucose** | Positively associated  (β per mmol/ = 0·09169, SE 0·02711, P=0·001) | Alpha-Tocopherol concentration was inversely associated with glucose concentration and C-peptide. |
| **Glycosylated haemoglobin** | Positively associated  (β per mmol/ = 0·04954, SE 0·01284, P < 0·001) |
| Olmedilla et al. 2002 | 200 volunteered men and 200 women aged 25–45 in five European regions. | **Alpha-tocopherol** | Decreased gamma-T markedly about 63% when supplied alone. | Increased alpha-T about 35%.  Both the alpha-T and gamma-T returned to baseline value 1 month after discontinuing alpha-T. |
| Leonard et al. 2005 | Fourteen (5 females, 9 males) active, healthy, nonsmoking adults, who were not currently vitamin or antioxidant supplement users. | **Deuterium-labeled alpha- and gamma-tocopherol** | Gamma-tocopherol is rapidly metabolized to gamma-CEHC, and to a greater degree in women than in men, whereas alpha-tocopherol is maintained in the plasma and little is metabolized to alpha-CEHC.  Bile may be an important way of excretion for both gamma-T and gamma-CEHC. | |
| Jeanes et al. 2004 | 15 smokers (males n = 8; females n = 7)  15 non-smokers (males n = 8; females n = 7) of comparable ages | No significant difference of gamma-T in plasma and platelets between smokers and non-smokers, but smokers had significantly lower levels of g-tocopherol in their lymphocytes.  Smokers excreted significantly more urinary gamma-CEHC compared to non-smokers. | | No significant difference of gamma-T in plasma and platelets between smokers and non-smokers, but smokers had significantly lower levels of alpha-tocopherol in their lymphocytes. |
| Ingles et al. 1998 | Plasma alpha- and gamma-T concentration for 332 subjects with colorectal adenomas and 363 control subjects from a previous sigmoidoscopy-based study. | Before adjusted for potential confounders, Increasing alpha-tocopherol and decreasing gamma-tocopherol levels were associated with decreased occurrence of large (> or = 1 cm) but not of small (<1 cm) adenomas.  After adjustment, Subjects in the highest versus lowest quintile of alpha-tocopherol: gamma-tocopherol ratio had an odds ratio of 0.36 (95% confidence interval, 0.14-0.95) for large adenomas.  A high plasma **alpha: gamma-t ratio** may be a better predictor of decreased cancer risk than high plasma alpha-tocopherol alone. | | |
| Djuric et al. 2006 | 122 healthy, premenopausal women with a family history of breast cancer were randomized across four diet arms for one year in a 2 x 2 factorial design study: control, low-fat, high fruit-vegetable and combination low-fat/high FV diets. | Low-fat intervention resulted in significantly decreased both gamma-tocopherol dietary intakes and plasma levels. | | Alpha-tocopherol was not affected |
| Frankenfeld et al. 2012 | Participants (n=2031) comprised women who had participated in a case-control study of diet and breast-related diseases nested within a randomized trial of breast self-examination among textile workers (n=266 064). | **Fruit intake** | Inversely associated with gamma-T | Alpha-T is positively associated with fruit intake. |
| Jiang et al. 2004 | Human prostate cancer cell lines (androgen-resistant PC-3 and androgen-sensitive LNCaP) and lung cancer cells (A549). | Gamma-T, but not alpha-T, inhibited growth of several human cancer cell lines, including prostate PC-3 and LNCaP and lung A549 cells.  Gamma-T induces cell death in a prostate cancer cell line by interrupting *de novo* synthesis of **sphingolipids**. | | |
| Jiang et al. 2002 | Twenty-four 2-month-old male Fischer 344 rats | Gamma-T supplementation inhibits protein nitration, attenuates inflammation-induced ascorbate oxidation, and generally spares vitamin C in the absence of an effect on alpha-T.  These effects were achieved by a moderate supplementation of gamma-T in rats that had supplemented levels of alpha-T. | | |
| Jiang et al. 2000 | Human macrophages and epithelial cells | Gamma-T and its major metabolite gamma-CEHC, but not alpha-T, inhibit COX activity and thus possess anti-inflammatory activity. | | |
| Jiang et al. 2003 | Male Wistar rats (250–330 g) | Gamma-T inhibits the pro-inflammatory eicosanoids, suppresses pro-inflammatory cytokine, and attenuates inflammation-mediated damage in a rat inflammation model. | | |
| Gysin et al. 2002 | Androgen-independent human prostate PC-3 tumor cell line | Gamma-tocopherol has an evident differentiative capacity on PC3 cells (inhibit the proliferation), leading to an increased expression of TG2 (transglutaminase 2), and reduced cyclin D1 and cyclin E levels, affecting cell cycle progression. | | |
| Fei Guan et al. 2012 | Azoxymethane-Treated F344 Rats | **4-HNE** | Decreased | γ-T could inhibit colon carcinogenesis. |
| **Nitrotyrosine** | Decreased |
| **PPAR- γ** | Increased |
| **Cyclin D1 expression in ACF** | Decreased |
| **PGE2 and 8- isoprostane in serum** | Decreased |
| Gopalan et al. 2012 | MCF-7 and MDA-MB-435 human breast cancer cell lines, and SUM159 human breast cancer cells. | Gamma-T induces apoptosis through activation of pro-apoptotic JNK/CHOP/DR5-mediated events.  An increased level of **ceramide** generated by the de novo ceramide synthesis pathway is a common upstream mediator for both gamma-T- and gamma-T3 (gamma-tocotrienol) -mediated apoptotic events. | | |
| Campbell et al. 2009 | PC-3 prostate cancer cell line | **15-S-HETE**  **(An endogenous PPAR*γ* ligand)** | Increased | These data demonstrate that the growth arrest mediated by  gamma–T and its effectiveness as an anti-proliferative agent in prostrate cancer cells follows a PPAR*γ* –dependent mechanism. |
| **15-Lipoxygenase-2 (a tumor suppressor)** | Increased |
| **Cyclin D1** | Decreased |
| **Cyclin D3** | Decreased |
| **Bcl-2** | Decreased |
| **NF*κ* B protein** | Decreased |
| Campbell et al. 2003 | SW480 human colon cancer cell lines and COS-7 cells. | Both gamma-T and alpha-T could modulate the PPARγ expression in SW 480 colon cancer cell lines, but the up-regulation of PPARγ by γ-tocopherol is more significant than the up-regulation of PPARγ by α-tocopherol perhaps due the ability of γ-tocopherol to accumulate at higher concentrations in the cell. | | |
| Campbell et al. 2006 | The colon cancer cell lines SW480 (RPMI 1640), HCT-116 (McCoy's), HT-29 (Dulbecco's Modified Eagle Medium), and HCT-15 (RPMI 1640), and the CCD-112CoN (normal primary cells, non-transformed phenotype). | Treatment with RRR-γ-tocopherol resulted in significant cell death for all cancer cell lines tested, while RRR-α- tocopherol did not.  Further, RRR-γ-tocopherol treatment showed no cytotoxicity to normal colon cells CCD-112CoN at the highest concentration and time point tested. | | |
| B MCF-7 et al. 2009 | BALB/c mice, endothelial cell line mHEVa and Spleen cells | isoform D-*γ*-tocopherol elevates inflammation in experimental asthma  D-*γ*-tocopherol, at as little as 10% the concentration of D-*α*-tocopherol, ablates the anti- inflammatory benefit of the D-*α*-tocopherol isoform.  A mechanism for these opposing immunoregulatory functions of purified tocopherols at physiological concentrations is not through modulation of expression of several cytokines, chemokines, or adhesion molecules, but is, at least in part, by regulation of endothelial cell signals during leukocyte recruitment. | | |
| Sergejs Berdnikovs  et al. 2013  (Lung inflammation) | BALB/c mice | VCAM-1 activation of PKCα |  | γ-T does ***not*** regulate apoptosis by modulation of cytokines, Chemokines or adhesion molecule. |
| leukocyte recruitment to the lung |  |
| d-α-T inhibition of leukocyte transendothelial migration in vitro |  |
| Amanda K. Smolarek et al. 2012 | Female MMTV/ErbB2/neu transgenic mice | Bcl-2 |  | Dietary supplementation could inhibit tumorigenesis in animal models. |
|  | XIAP |  |
|  | PTEN |  |
|  | p21 |  |
|  | P27 |  |
|  | PPARγ |  |
|  | PCNA |  |
|  | PKC |  |