

Dietary fat and risk of renal cell carcinoma in the USA: a case-control study. Kaye E. Brock et al. *Br J Nutr* 2009;101(8):1228-1238.

Abstract: An increased risk of renal cell carcinoma (RCC) has been linked with obesity. However, there is limited information about the contribution of dietary fat and fat-related food groups to RCC risk. A population-based case-control study of 406 cases and 2434 controls aged 40–85 years was conducted in Iowa (1986–89). For 323 cases and 1820 controls from the present study, information on dietary intake from foods high in fat nutrients and other lifestyle factors was obtained using a mailed questionnaire. Cancer risks were estimated by OR and 95 % CI, adjusting for age, sex, smoking, obesity, hypertension, physical activity, alcohol and vegetable intake and tea and coffee consumption. In all nutrient analyses, energy density estimates were used. Dietary nutrient intake of animal fat, saturated fat, oleic acid and cholesterol was associated with an elevated risk of RCC (OR = 1.9, 95 % CI 1.3, 2.9, Ptrend < 0.001; OR = 2.6, 95 % CI 1.6, 4.0, Ptrend < 0.001; OR = 1.9, 95 % CI 1.2, 2.9, Ptrend = 0.01; OR = 1.9, 95 % CI 1.3, 2.8, Ptrend = 0.006, respectively, for the top quartile compared with the bottom quartile of intake). Increased risks were also associated with high-fat spreads, red and cured meats and dairy products (OR = 2.0, 95 % CI 1.4, 3.0, Ptrend = 0.001; OR = 1.7, 95 % CI 1.0, 2.2, Ptrend = 0.01; OR = 1.8, 95 % CI 1.2, 2.7, Ptrend = 0.02; OR = 1.6, 95 % CI 1.1, 2.3, Ptrend = 0.02, respectively). In both the food groups and nutrients, there was a significant dose-response with increased intake. Our data also indicated that the association of RCC with high-fat spreads may be stronger among individuals with hypertension. These findings deserve further investigation in prospective studies. Key Words: Kidney cancer; Renal cell carcinoma; Case-control studies; Dietary fat.

Meta-analysis of animal fat or animal protein intake and colorectal cancer. Dominik D Alexander et al. *Am J Clin Nutr* 2009;89(5):1402-1409.

Background: In the recent World Cancer Research Fund/American Institute for Cancer Research report of diet and cancer, it was concluded that there is limited but suggestive evidence that animal fat intake increases the risk of colorectal cancer. **Objective:** To clarify this potential relation, we conducted meta-analyses across a variety of subgroups, incorporating data from additional studies. **Design:** Analyses of high compared with low animal fat intakes and categorical dose-response evaluations were conducted. Subgroup analyses, consisting of evaluations by study design, sex, and tumor site were also performed. **Results:** Six prospective cohort studies with comprehensive dietary assessments, contributing 1070 cases of colorectal cancer and ≈1.5 million person-years of follow-up, were identified. The summary relative risk estimate (SRRE) for these studies was 1.04 (95% CI: 0.83, 1.31; P for heterogeneity = 0.221) on the basis of high compared with low intakes. When data from case-control studies were combined with the cohort data, the resulting SRRE was 1.15 (95% CI: 0.93, 1.42) with increased variability (P for heterogeneity = 0.015). In our dose-response analysis of the cohort studies, no association between a 20-g/d increment in animal fat intake and colorectal cancer was observed (SRRE: 1.02; 95% CI: 0.95, 1.09). In a separate analysis of 3 prospective cohort studies that reported data for animal protein or meat protein, no significant association with colorectal cancer was observed (SRRE: 0.90; 95% CI: 0.70, 1.15). **Conclusion:** On the basis of the results of this quantitative assessment, the available epidemiologic evidence does not appear to support an independent association between animal fat intake or animal protein intake and colorectal cancer.

The relation of α -linolenic acid to the risk of prostate cancer: a systematic review and meta-analysis. Joel A Simon et al. *Am J Clin Nutr* 2009;89(5):1558S-1564S.

Background: α -Linolenic acid (ALA; 18:3n-3) has been associated inconsistently with an increased risk of prostate cancer. Additional studies have become available since the publication of 2 previous meta-analyses. **Objective:** The objective was to review the published data on the relation between ALA and prostate cancer. **Design:** We conducted a systematic review to identify studies that included data on ALA and risk of prostate cancer. Data were pooled from studies that compared the highest ALA quantile with the lowest ALA quantile, and risk estimates were combined by using a random-effects model. **Results:** The relation between ALA and prostate cancer is inconsistent across studies. We pooled data from 8 case-control and 8 prospective studies. The summary estimate revealed that high ALA dietary intakes or tissue concentrations are weakly associated with prostate cancer risk (relative risk [RR]: 1.20; 95% CI: 1.01, 1.43). When examined by study type (ie, retrospective

compared with prospective or dietary ALA compared with tissue concentration) or by decade of publication, only the 6 studies examining blood or tissue ALA concentrations revealed a statistically significant association. With the exception of these studies, there was significant heterogeneity and evidence of publication bias. After adjustment for publication bias, there was no association between ALA and prostate cancer (RR: 0.96; 95% CI: 0.79, 1.17). **Conclusions:** Studies examining the relation between ALA and prostate cancer have produced inconsistent findings. High ALA intakes or high blood and adipose tissue concentrations of ALA may be associated with a small increased risk of prostate cancer. However, these conclusions are qualified because of the heterogeneity across studies and the likelihood of publication bias.