

Neonatal vitamin A supplementation for prevention of mortality and morbidity in infancy: systematic review of randomised controlled trials. Siddhartha Gogia and Harshpal Singh Sachdev. *BMJ* 2009;338:b919.

Objective: To evaluate the effect of neonatal vitamin A supplementation on infant mortality, morbidity and early adverse effects. **Design:** Systematic review, meta-analysis, and meta-regression of randomised controlled trials. **Data sources:** Electronic databases and hand search of reviews; abstracts and proceedings of conferences. **Review methods:** Randomised or quasi-randomised or cluster randomised, placebo controlled trials evaluating the effect of prophylactic, neonatal (<1 month) supplementation with synthetic vitamin A on mortality or morbidity within infancy (<1 year), and early adverse effects (≤ 7 days). **Results:** The six included trials were from developing countries. There was no convincing evidence of a reduced risk of mortality during infancy (relative risk 0.92, 95% confidence interval 0.75 to 1.12, $P=0.393$ random effect; $I^2=54.1\%$) or of an increase in early adverse effects including bulging fontanelle (1.16, 0.81 to 1.65, $P=0.418$; $I^2=65.3\%$). No variable emerged as a significant predictor of mortality, but data for important risk groups (high maternal night blindness prevalence and low birth weights) were restricted. Limited data (from one to four trials) did not indicate a reduced risk of mortality during the neonatal period (0.90, 0.75 to 1.08, $P=0.270$; $I^2=0\%$), cause specific mortality, common morbidities (diarrhoea and others), and admission to hospital. There was, however, evidence of an increased risk of acute respiratory infection and a reduced risk of clinic visits. **Conclusions:** There is no convincing evidence of a reduced risk of mortality and possibly morbidity or of increased early adverse effects after neonatal supplementation with vitamin A. There is thus no justification for initiating such supplementation as a public health intervention in developing countries for reducing infant mortality and morbidity.

Dietary Carotenoid Intake Is Associated with Lower Prevalence of Metabolic Syndrome in Middle-Aged and Elderly Men. Ivonne Sluijs et al. *J Nutr* 2009;139(5):987-992.

Carotenoids have antioxidant properties. Little is known about the relation of dietary carotenoid intake on metabolic syndrome risk. We examined whether dietary carotenoid intake was associated with metabolic syndrome and metabolic syndrome risk factors. We conducted a population-based, cross-sectional study in 374 men aged 40–80 y. Intakes of β -carotene, α -carotene, β -cryptoxanthin, lycopene, lutein, and zeaxanthin were estimated using a validated FFQ. Presence of metabolic syndrome was determined using fasting serum glucose, triglyceride, and HDL-cholesterol concentrations, waist circumference, and systolic and diastolic blood pressure. Metabolic syndrome was present in 22% of the men. After adjustment for confounders, total carotenoid and lycopene intakes were inversely associated with presence of metabolic syndrome [relative risk (RR) quartile 4 vs. quartile 1 (95% CI) 0.42 (0.20–0.87), P -trend 0.02; and 0.55 (0.28–1.11), P -trend 0.01, respectively]. For β -carotene, a decreased risk was observed for each quartile of intake compared with the first [RR quartile 4 vs. quartile 1 (95% CI) 0.58 (0.33–1.02)]. Higher total carotenoid, β -carotene, α -carotene, and lycopene intakes were associated with lower waist circumferences and visceral and subcutaneous fat mass. Higher lycopene intake was related to lower serum triglyceride concentrations. In conclusion, higher total carotenoid intakes, mainly those of β -carotene and lycopene, were associated with a lower prevalence of metabolic syndrome and with lower measures of adiposity and serum triglyceride concentrations in middle-aged and elderly men.

Intakes of Fruit, Vegetables, and Carotenoids and Renal Cell Cancer Risk: A Pooled Analysis of 13 Prospective Studies. Jung Eun Lee et al. *Cancer Epidemiol Biomarkers Prev* 2009;18(6):1730–1739.

Fruit and vegetable consumption has been hypothesized to reduce the risk of renal cell cancer. We conducted a pooled analysis of 13 prospective studies, including 1,478 incident cases of renal cell cancer (709 women and 769 men) among 530,469 women and 244,483 men followed for up to 7 to 20 years. Participants completed a validated food-frequency questionnaire at baseline. Using the primary data from each study, the study-specific relative risks (RR) were calculated using the Cox proportional hazards model and then pooled using a random effects model. We found that fruit and vegetable consumption was associated with a reduced risk of renal cell cancer. Compared with <200 g/d of fruit and vegetable intake, the pooled multivariate RR for ≥ 600 g/d was 0.68 [95% confidence interval (95% CI) = 0.54–0.87; P for between-studies heterogeneity = 0.86; P for trend = 0.001]. Compared with <100

g/d, the pooled multivariate RRs (95% CI) for ≥ 400 g/d were 0.79 (0.63-0.99; P for trend = 0.03) for total fruit and 0.72 (0.48-1.08; P for trend = 0.07) for total vegetables. For specific carotenoids, the pooled multivariate RRs (95% CIs) comparing the highest and lowest quintiles were 0.87 (0.73-1.03) for α -carotene, 0.82 (0.69-0.98) for β -carotene, 0.86 (0.73-1.01) for β -cryptoxanthin, 0.82 (0.64-1.06) for lutein/zeaxanthin, and 1.13 (0.95-1.34) for lycopene. In conclusion, increasing fruit and vegetable consumption is associated with decreasing risk of renal cell cancer; carotenoids present in fruit and vegetables may partly contribute to this protection.