associated with fruit and vegetable intake were included. Random effects models were used to estimate summary RRs.

**Results** Nineteen cohort studies were included in the meta-analysis. The summary RR for the highest vs the lowest intake was 0.92 (95% CI 0.86 to 0.99) for fruit and vegetables combined, 0.90 (95% CI 0.85 to 0.98) for fruit and 0.91 (95% CI 0.86 to 0.96) for vegetables. The inverse associations appear to be restricted to colon cancer. In linear dose-response analysis only intake of vegetables was significantly associated with colorectal cancer risk, summary RR 0.98 (95% CI 0.97 to 0.99) per 100 g per day. However, significant inverse associations emerged in non-linear models for fruits (p non-linearity <0.001) and vegetables (p non-linearity =0.001). The greatest risk reduction was observed when increasing intake from very low levels of intake. There was generally little evidence of heterogeneity in the analyses and there was no evidence of small-study bias.

**Conclusion** This meta-analysis indicates that there is a weak, but statistically significant non-linear inverse association between fruit and vegetable intake and colorectal cancer risk.

**3.4 THE EPIDEMIOLOGY OF COGNITIVE RESERVE IN AGEING**

**Chair: Dr. Fiona Matthews, UK**

**03-4.1 COGNITIVE LIFESTYLE, DEMENTIA PROTECTION AND THE BRAIN’S RESERVE MECHANISMS**

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Cognitive lifestyle refers to acquired patterns of cognitive and mental activity over the course of one’s lifetime, and in our group has been formalised to include information about educational achievement, occupational complexity, social engagement and cognitively-loaded leisure activities. Many large-scale prospective cohort studies have shown that maintaining a more active cognitive lifestyle is linked with a reduction in incident dementia risk. Recently, we have further shown that the combination of a higher educational level with either a more cognitively challenging job in mid-life, or enhanced social activity in later life, is more important for minimising dementia risk than either factor in isolation.

Brain reserve and cognitive reserve are typically suggested to mediate this protective relationship. In this presentation, a brief history of the terms will be given, as well as a deconstruction into more specific biological mechanisms. An evaluation of the role of disease modifying, neuroprotective and compensatory mechanisms is then possible, and will be discussed in relation to the design of clinical trials and community-based studies.