

Smoking

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A study of genetic susceptibility to renal cell carcinoma (RCC) among smokers and non-smokers.

The proposal is that the expression (production) of N acetyl transferase is genetically controlled **and** is required for metabolising tobacco carcinogens. Subjects were categorised as slow or fast acetylators. The proposal is that fast acetylators will metabolise potential carcinogens more effectively.

Cases of RCC and controls (taken from the local telephone book) were classified according to genotype.

189 (60%) of RCC cases agreed to take part 115 donated blood.

259 (61%) of controls completed all parts of the study.

Exposure to tobacco smoke was measured by recall.

With **no** exposure to tobacco smoke, slow acetylators were at increased risk of RCC; odds ratio (OR) = 1.8 (95% CI = 1.1,2.9)

Slow acetylators that had a history of smoking were at higher risk of RCC; OR = 3.2 (95% CI = 1.7,6.1)

Fast acetylators that had a history of smoking were not at statistically significant risk; OR = 1.4 (95% CI = 0.7, 2.9)

Overall, smoking increases the risk of RCC; OR = 2.2 (95% CI = 1.3,3.7).

Comment

Participation rates and methods of exposure assessment were less than ideal. However, the hypothesis seems to be supported by these findings; slow acetylators are at higher risk of RCC with or without a history of smoking. A significant proportion of the excess risk of RCC is innate i.e. not related to smoking history. Accurate determination of relative contributions would require a much more powerful study.

