

The change in excess risk of lung cancer attributable to smoking following smoking cessation: an examination of different analytic approaches using CPS-I data

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Abstract

Background Lung cancer risk is modified by smoking cessation. However, the inclusion in the group of former smokers of those who quit after developing symptoms or being diagnosed with lung cancer distorts the apparent risk in the first several years following cessation. This bias is termed the *quitting ill* effect.

Methods Lung cancer mortality data from the American Cancer Society's CPS-I were used to calculate the excess mortality among white male former smokers compared to the predicted risk had those individuals continued to smoke. Alternate approaches to minimizing the quitting ill bias were investigated. Goodness-of-fit of the models was assessed graphically and formally.

Results Poisson models were built for the absolute lung cancer risk for never smokers and the excess risk, over never smokers, for continuing smokers. The decrease in excess risk in former smokers was modeled by a negative exponential function. The models for the three smoker subgroups (continuing, never, and former), all fit the data well. Assuming that the fraction of excess risk remaining for former smokers does not decline for the first two years following cessation and that the quitting ill effect does not influence those who are five or more years post-cessation allowed a reasonable estimation of the change in risk of lung cancer with increasing duration of abstinence.

Conclusions The reduction in the excess risk of lung cancer in former smokers can be estimated, and the quitting ill effect minimized, by the inclusion of a lag between cessation and onset of reduction in risk.

Keywords Lung cancer · Tobacco · Risk factor · Methodological studies

Introduction

Smoking cessation and continued abstinence modifies a smoker's risk of developing lung cancer [1–4]. However, the overall group of former smokers includes those who quit because they were diagnosed with lung cancer, and these individuals artificially elevate the death rate from lung cancer for all former smokers. This *quitting ill* effect was characterized by Garfinkel and Stellman [5], and was discussed by Halpern et al. [6], who provide additional documentation of the effect. Eliminating former smokers who quit after diagnosis can moderate the quitting ill effect, but some individuals quit smoking because of conditions or symptoms such as pneumonia that may precede a lung cancer diagnosis by several months. This influence of the development of lung cancer on cessation, and the high case mortality rate from lung cancer, makes estimation of the change in lung cancer risk in the first several years following cessation problematic.

The biology of lung cancer precludes a rapid change in the lung cancer death rate following cessation. The principal effect of smoking cessation on lung cancer risk is a change in the frequency with which normal cells transform into a malignancy. A substantial interval in time elapses between malignant transformation and growth of the cancer to a size where it can be detected, and a further interval

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