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In the news

LIFTING THE SMOKE SCREEN

Three independent studies provide "the first genetic clues to unravelling the mystery of why some smokers puff their way through life without developing disease while others die young of lung cancer" (*The Financial Times*, 3 April 2008).

The studies, which involved more than 35,000 people in Europe, Canada and the United States, identified variants in two key areas on chromosome 15. Surprisingly, the results "point to areas of the genetic code that are not associated with pleasure and the rewards of addiction" (*The New York Times*, 3 April 2008). Although it is unclear whether the variants lie in one gene or three closely linked genes, it is known that they alter nicotinic receptors.

"Smokers with one copy of the two variants — present in half the population — have a 30 per cent increased risk of lung cancer, and those with two copies — one in 10 of the population — have an 80 per cent increased risk, compared with smokers without the variants" (*The Independent*, 3 April 2008).

But the authors of the studies disagree on "whether the set of variants directly increased the risk of lung cancer or did so indirectly by causing more smoking" (*The New York Times*, 3 April 2008). A smoker who is homozygous for these variants "on average lights up two extra cigarettes a day and has a much harder time quitting" (*The New York Times*, 3 April 2008).

The findings have important medical implications as they might "lead to new treatments and ways of helping people to quit smoking" (*The Times*, 3 April 2008). Moreover, they show "how a disease that clearly has a strong environmental component can also be affected by genetics" (*The Times*, 3 April 2008).

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