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Smoking selects mutants

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In the October 24 Proceedings of the National Academy of Sciences Rodin and Rodin propose that smoking leads to increased lung cancer not by causing more mutations, but by selecting for those mutations that do arise (*Proc Natl Acad Sci USA* 2000, **97:**12244-12249). They take advantage of an increase in p53 mutational data in nonsmokers and find, for example, that the frequency of silent mutations in p53 is identical between smokers and non-smokers. In contrast, twice as many lung cancers from smokers have a defective p53 as compared to lung cancers from non-smokers. Rodin and Rodin suggest that the more often that smoking-related stresses such as hypoxia induce p53-related cell cycle arrest or apoptosis, the higher the probability that a p53 mutant cell will be selected. If true, this would mean that eliminating specific carcinogens from cigarettes would have little effect on the occurrence of lung cancer.

References

1. Proceedings of the National Academy of Sciences, [http://www.pnas.org/]

2. IARC TP53 mutation database , [http://www.iarc.fr/p53/]

3. Strand asymmetry of CpG transitions as indicator of G1 phase-dependent origin of multiple tumorigenic p53 mutations in stem cells.

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