

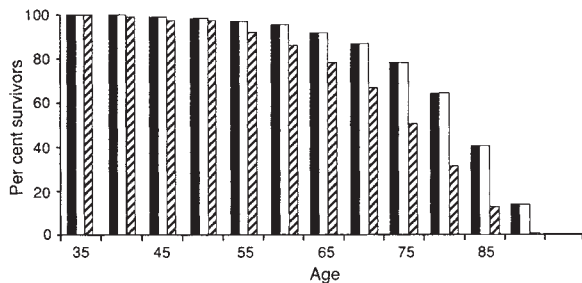
gene deletions were less frequent in uncultured than in cultured tumours, they were still present in 19 per cent of tumours examined. It will also be of interest to determine the two pathways by which p16 and p53 gene defects facilitate growth *in vitro* in bladder and other tumour types.

**Charles H. Spruck III\***,  
**Mirella Gonzalez-Zulueta\***,  
**Atsuko Shibata, Anne R. Simoneau,**  
**Ming-Fong Lin, Felicidad Gonzales,**  
**Yvonne C. Tsai, Peter A. Jones†**  
*Department of Biochemistry and  
 Molecular Biology,  
 USC/Norris Comprehensive Cancer Center,  
 Los Angeles, California 90033, USA*

\*Both authors contributed equally to the work.  
 †To whom correspondence should be addressed.

## Tobacco-related mortality

SIR — Despite widespread knowledge of the health risks of cigarette smoking, many smokers cannot quit because they are addicted to nicotine. B. R. has proposed earlier<sup>1</sup> that inveterate smokers switch to oral smokeless tobacco, consumed as chewing tobacco or moist snuff,



Survival rates for 35-year-old individuals: smokers (hatched bars), non-users of tobacco (black bars) and smokeless-tobacco users (open bars).

which also satisfies a smoker's nicotine addiction. This strategy has not been pursued as a public-health measure because of concern that smokeless tobacco might cause some people to start or to recommence smoking, and because it poses a health hazard. But the only consequential hazard from smokeless tobacco use known so far is oral cavity cancer<sup>2</sup>. In India, use of smokeless tobacco (although usually combined with betel leaf, areca nut, and/or slaked lime) is a major risk factor for oral cancer. Yet in the United States, its use has only a small risk<sup>3</sup>. Even prolonged use of the newer smokeless tobacco products may have a low absolute risk, which would not comprise a meaningful objection to smokeless tobacco substitution for the inveterate smoker.

We estimated the life expectancy of 35-year-old white males with three patterns of tobacco use: non-users, cigarette smokers and smokeless-tobacco users.

For non-users and smokers, we used the mortality rates from the American Cancer Society's second cancer prevention study of smoking and mortality among more than one million Americans<sup>4</sup>.

We employed standard methods to determine the attributable risk for oral cancer among smokeless-tobacco users<sup>5</sup>. A relative risk of 8 (oral cancer risk among users relative to that among non-users) was used to estimate the excess oral cancer mortality in this group. We applied the excess risk equally at all ages after 35 years, even though oral cancer generally occurs in users over age 70 (ref. 6).

The results indicate that the average remaining life expectancy of a 35-year-old smokeless-tobacco user is 45.92 years, only 0.04 year less than that of a non-user (see figure). This 15-day reduction in life expectancy is in sharp contrast to the 7.8 years lost by smokers. Thus, both the 35-year-old non-user of tobacco and the smokeless-tobacco user will live on average to be 80.9 years of age compared with 73.1 years for the smoker. Only 67 per cent of smokers will be alive at age 70, compared with more than 87 per cent of smokeless-tobacco users and non-users of tobacco. Although the effect of smokeless-tobacco on life expectancy may

at first seem surprising, the absolute risk of developing oral cancer from its use is small, the disease is not uniformly fatal, and this type of tobacco use is not associated with the other causes of smoking-related deaths.

Experience of the past 30 years in the United Kingdom and the United States shows that, despite substantial reductions in smoking uptake and continuation rates, many people remain addicted to nicotine, often

with fatal consequences. We suggest that abstinence is not the only approach to reducing tobacco-related mortality: for smokers addicted to nicotine who would not otherwise stop, a permanent switch to smokeless tobacco could be an acceptable alternative to quitting.

**Brad Rodu**  
*Department of Oral Pathology,  
 School of Dentistry,*  
**Philip Cole**  
*Department of Epidemiology,  
 School of Public Health,  
 University of Alabama at Birmingham,  
 Birmingham, Alabama 35294-0007, USA*

- Rodu, B. *Am. J. med. Sci.* **308**, 32-34 (1994).
- Cullen, J. W. *et al. Pub. Hlth. Rep.* **101**, 355-373 (1986).
- Winn, D. M. *et al. New Engl. J. Med.* **304**, 745 (1981).
- Garfinkel, L. *NCI Monographs* **67**, NIH publ. no. 85-2713, 49-52 (1985).
- Cole, P. & MacMahon, B. *Br. J. Prev. Soc. Med.* **25**, 242-244 (1971).
- McGuirt, W. F. & Wray, A. *NCI Monogr.* **2**, NIH publ. no. 93-3461, 91-95 (1993).

## Electrostatic sense in rattlesnakes

SIR — An isolated rattlesnake rattle produces a positive electrostatic charge of 75 V when mechanically vibrated. Friction of snake skin on materials common in the environment also produces positive charges as high as 1,000 V. These facts are a necessary condition for the hypothesis that snakes sense the presence of cover and other environmental features by electrostatic effects involved in tongue scanning. There are two other considerations.

First, in common with all other dry-skinned land animals, snakes acquire electrostatic charges by friction during their sliding movements and are typically observed to have positive charge levels of between 100 and 1,000 V. Possibly because of electrostatic discharge hazards plus the tendency of charged surfaces to attract dust, all dry-skinned land animals other than snakes and snake-like lizards are covered with a multitude of electrical discharge points in the form of hair, feathers, bristles, setae or spinules, or they have more electrically conductive skin (unlike that of snakes). Thus, snake skin appears adapted for acquiring and retaining static in the following ways: it is a good electrical insulator; a positive charge is produced by friction with nearly all common materials; and it has a relative absence of points where discharges may occur.

Second, the explanation that rattlesnakes avoid injury by warnings produced by noise of tail rattling<sup>1</sup> is partly correct, but harks back to times when this was the only explanation. The warning explanation becomes less convincing in the light of the facts that silent rattling is common in juveniles and smaller rattlesnake species, and that the primitive ancestral forms of the rattle were probably silent. Thus the rattle may have other functions unrelated to warnings.

We used the experimental arrangement shown in the figure to determine whether rattlesnake rattles could produce electrostatic charges solely by vibration in air. We attached a rattle (for example, from a western diamondback rattlesnake, *Crotalus atrox*) to an end of a coil of wire so that the rattle could be vibrated. The wire coil was held in place by acrylic plastic mounted over a static voltmeter (709 static sensor, 3M Co.). When vibrated at 60 Hz through an insulating nylon rod with no rattle in place, the voltmeter showed a zero voltage. But when the rattle was in place and vibrated, positive charges of 50-100 V were produced, indicating that a vibrating rattle generates electrostatic charges.

In feeding and in following trails, snakes touch objects and surfaces with