Letters to the Editor

Misleading Information on Environmental Tobacco Smoke in the French Lay Press

From CYRILLE COLIN AND HERVE MAISONNEUVE

Sir—Most countries in the World are still fighting the dangers of smoking.¹ In France it was widely believed that the government could regulate smoking and control some of the tobacco industry’s strategies. For example, in January 1991 a law was passed which banned the use of cigarette brand names in advertising.

However the fight continues. For example in June 1996 the tobacco industry launched an extensive campaign in the lay press. Documents were sent to leading health care professionals to encourage them to train media personnel to interpret scientific data. It is often suggested that the results of research are misunderstood. Exposure to environmental tobacco smoke (ETS) was chosen as an example by the industry (while admitting that ETS causes cancer). Health care professionals were provided with a selected bibliography of reports and expert opinion which showed that ETS was not a significant risk for non-smokers. They concluded that neither ‘scientific data’, nor ‘European opinions’ (they excluded North American and Japanese opinions) gave strong reasons for banning smoking in private and public work places. They also encouraged private and public institutions to set up appropriate policies to enable smoking in the workplace.

The tobacco industry’s campaign in the lay press started on 17 June 1996 and used six different advertisements at a cost of US$ 1.3 million to target nine different newspapers. They graded the public health risk of ETS compared with other risk factors according to Table 1.

Using the notion of relative risk to compare figures it was argued that a relative risk of between 1.0 and 2.0 did not give strong evidence of a causal association between ETS and lung cancer. Table 1 gives false or incomplete information and should have been completed by researchers who performed the data collection for each topic and by those who critically appraised the studies. It might be asked what is the relevance of information on the association between vegetarian diet and heart disease or biscuit consumption and heart disease, or pepper consumption and mortality.


However, these figures are not quoted in the advertisements published in the lay press. Most of these studies concern groups of women exposed to ETS who had never smoked themselves. Perhaps the industry is suggesting that these population groups do not exist in France. There is evidence from the US² that population groups exposed to ETS have high serum cotinine levels and that the environment at home and in the workplace significantly contributes to ETS exposure. In France it is likely that such exposure is as bad if not worse.

The scientific community should not allow this misleading information to continue to be published in the lay press. The selected use of scientific data to mislead the public should be prohibited. Public health funding is insufficient to fight such strong lobby groups. As stated by Radford ‘people tend to get most of their information beyond work and family horizons from the press, radio and television’.³ The tobacco industry is aware of this. Even tenuous evidence can be supported in people’s minds by repetition and old adages such as ‘mentez, il en restera toujours quel-quechose’ (go ahead and lie, there is always something to be gained from it). The tobacco industry is also aware that when journalists decide that a story is ‘going down well’, all the newspapers start telling the same story and since the general public tend not to read the medical press there is no way of correcting misleading information.

In France one particular tobacco firm has now been successfully sued by the French National Committee

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against Tobacco and was found guilty on 25 June 1996. The advertising campaign was stopped but by then most of the misleading statements had been published.

REFERENCES
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The Constitutional Hypothesis Negated by Twins Discordant for Smoking and Mortality

From P H JONGBLOET

Sir—In a recent study 1 on smoking-discordant twins undertaken to test the constitutional hypothesis, an elevated risk of death was found among the smokers: for lung cancer (monozygotic (MZ) pairs: RR = 5.0; dizygotic (DZ) pairs: RR = 11.0) and for cardiovascular diseases (MZ pairs: RR = 3.9; DZ pairs: RR = 2.8). This strong association in smoking-discordant MZ twins, as in the population as a whole, would support the causal relation between tobacco and premature mortality and negate the constitutional hypothesis 2 that ‘genetic or early shared familial influences underlie this significant association’ or in other words, ‘that the association of tobacco smoking with personality traits would form a synergistic relationship which predisposes to cancer and cardiovascular mortality’. 2

This conclusion would only be correct if the phenotype of MZ twin pairs, including personality traits predisposing to smoking, would be exclusively dependent on the shared genome and not on any exogenous factor. There is, however, growing disagreement about whether concordance in MZ twin pairs is due to shared genes or shared environment, and uncritical acceptance of the results of classical twin studies may have misled generations of researchers. 3 In this context it is important to notice that in animals, experimentally-induced ageing of eggs before ovulation and/or fertilization resulted in a tendency of axial duplications, taking the form of twins, either of equal size and normal appearance, or of unequal dimensions; this teratogenic component may be apparent either in both embryos or in the smaller one. 4–6 The same tendency towards both MZ twinning and developmental abnormalities has also been observed in humans after delayed ovulation. 7 These unequal dimensions of MZ embryos could explain why the shorter twin is more likely to die of heart disease than the taller one. 8 The same accounts for the well-known low concordance rate of

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schizophrenic (and other constitutional) disorders in MZ twin pairs, and for the high rates of abnormal brain lateralization, besides left-handedness and enlarged cerebral ventricles, in the diseased co-twin.\textsuperscript{9,10} In addition, more often than not, the sick twin has the lower birth weight and the greater number of birth complications; he or she remains smaller, weaker, and slower in development. Additionally, in 9% of the cases the less affected co-twins are said to have reactive psychosis or borderline states, and in 29% neurosis, including character disorders, anxiety states, depressive or somatic neuroses and alcoholism.\textsuperscript{10}

A relationship between MZ twinning and personality traits in one or both of the co-twins and its dependence on a non-genetic factor not only discards the predominantly genetic considerations but also emphasizes non-optimal maturation of the oocyte as a possible cause of a constitution characterized by both personality traits and constitutional diseases.\textsuperscript{11,12} The latter include cancer development, as Witschi concluded in his review on overripeness of the egg as a cause of twinning and teratogenesis;\textsuperscript{13} the persistence of an embryonic appearance of the cells, designated as aplasia or progressive failure of cells to differentiate is the most constant effect produced by overripeness; if combined with considerable growth, it leads to the formation of neoplasm.

Moreover, the DZ twin pair incidence is modulated by the temporal relationship between follicle growth and rupture which is not as strict, even in spontaneous cycles of healthy volunteers.\textsuperscript{13} Asynchrony of follicle maturation and ovulations of up to 81 hours has been registered in induced plural ovulations.\textsuperscript{14} Maturation of the oocyte in animals and humans is known to be modulated by maternal factors, such as reproductive age, pregnancy interval, seasonality of conception, endocrinological diseases (diabetes mellitus, thyroid disease), undernutrition, use of drugs, etc. The maturation of the ultimately splitting oocyte leading to MZ twins and of both oocytes to DZ twins thus differs from that in singletons. Intrapair differences, therefore, may be dependent on the intriguing cascade of hormonal events involving optimal and non-optimal maturation of the oocyte(s).

In line with this concept, the mortality among twins has been found to be 1.14 times higher than in the general population, at least in females aged 60–89.\textsuperscript{15} Isch- has been found to be 1.14 times higher than in the gen- oocyte(s).

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In conclusion, the elevated risk of death among smoking-discordant MZ twins does not negate the constitutional hypothesis because twinning and personality variables (and their interaction with smoking) may be dependent on a non-genetic factor, namely, non-optimal maturation of the oocyte. It even substantiates the possibility of a synergistic relationship between personality traits predisposing to cigarette smoking and cancer or cardiovascular mortality.

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