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Smoking Hazard to the Fetus

No reasonable doubt now remains that smoking in pregnancy has adverse effects on the developing fetus. The effects range from retardation of fetal growth and prematurity to an increased risk of perinatal death from all causes.[1-4] The influence of smoking is said to be most evident in the latter months of pregnancy,[3] but the observation that the growth-retarded babies of smoking mothers remain small after birth[2] does suggest an effect dating earlier than the third trimester of pregnancy. This view is supported by a report of an increased incidence of spontaneous abortion among smokers.[2]

Many careful studies on pregnant women as well as animals suggest that it is the smoking itself rather than the type of woman who smokes that is responsible for these effects. Though smoking is commoner among women in social classes 3 to 5 than in 1 and 2,[5] the Perinatal Mortality Survey[6] showed that even after correction for social class, age, and parity the increase in perinatal risk is directly related to how heavily the mother smoked.

Nicotine is a powerful vasoconstrictor, but its effect is probably limited to the time when a cigarette is being smoked, and it is possible that the subsequent vasodilatation adequately compensates for this effect.[7] It is possible that the increased incidence of placental separation reported among women who smoke[8] is evidence of a nicotine effect though this complication is also associated with fetal growth retardation in non-smokers. The discovery that the blood content of carbon monoxide is higher in smokers than non-smokers,[9] which was subsequently confirmed in pregnant women,[10] provides another possible clue to the effects on the fetus. Carboxyhaemoglobin levels were also found to be higher in fetuses of mothers who smoked than in fetuses of non-smokers. A recent study by P. V. Cole and colleagues[11] has done much to elucidate the manner in which smoking can be harmful to the fetus. These workers showed that smoking raises the level of carboxyhaemoglobin in the blood; it was three times higher in mothers who smoked than in non-smokers. They also showed that the fetal concentration of carboxyhaemoglobin was about twice that of the mother, though other workers[10 12] had been unable to find a significant difference between maternal and fetal concentrations.

So far these studies had confirmed only what had been found in other studies and did not answer the question how the carboxyhaemoglobin might adversely affect the fetus. This was done by an in-vitro study of the influence of carbon monoxide on the oxygen-carrying capacity of fetal blood. Cole and colleagues showed that carboxyhaemoglobin in fetal blood shifted the oxygen dissociation curve to the left, with the result that at an oxygen saturation of 35% the Po_2 was reduced from 18 to 13 mm Hg. Though the carbon monoxide concentrations used in laboratory studies are not often reached in fetal blood, there can be little doubt that the fetal oxygen reserve is diminished. This hypoxic effect is further enhanced firstly by the reduced blood Po_2 of mothers who smoke, and secondly because (as was found) the concentration of carboxyhaemoglobin is higher in the fetus than in the mother. This phenomenon is interesting because carbon monoxide is a highly diffusible gas, and one would expect a state of transplacental equilibrium between mother and fetus. However, it has been suggested that the feto-maternal difference can be accounted for by the lower oxygen saturation and different affinity for carbon monoxide of fetal than of maternal blood.

What do these observations mean in practical terms? Almost certainly intrauterine hypoxia is responsible for the growth retardation of smokers' babies. For instance, babies of mothers living at high altitudes, who consequently have a reduced Po_2, are smaller than those of mothers living at sea level.[13] Even under optimal conditions the Po_2 of the fetus is low, and the intrauterine environment has been likened to life on top of Everest. Though fetal tissues are well oxygenated under normal conditions, even a modest fall in Po_2 may seriously interfere with fetal oxygenation. Moreover, babies of mothers who smoke have a tendency to an increased metabolic acidosis up to 12 hours after birth, which may be a consequence of asphyxia in utero.[14] It is clear that when there is a complication of pregnancy, such as a pre-eclamptic toxaemia, the additional hazard of smoking could seriously jeopardize the fetus.

Apart from the problem of survival the long-term effects of carbon monoxide poisoning on the offspring of mothers who smoked during pregnancy must be considered. Recently P. Astrup and colleagues[15 16] have reported on well-controlled studies in pregnant women who smoked. They also carried out parallel studies with rabbits showing that rabbit mothers with a high concentration of carboxyhaemoglobin (16-18%) had a greatly increased incidence of still-born and malformed babies. These effects were not observed

among the human infants, though birth weight was reduced. They concluded from these results that serious developmental defects did not occur unless carboxyhaemoglobin levels were well above those normally found among women who smoke.

The other question that has remained unanswered until recently is whether the offspring of mothers who smoke in pregnancy show any evidence of intellectual handicap. There appears to be some conflict of opinion on this point. R. R. Beard and N. Grandstaff[17] showed that even with a low concentration of carboxyhaemoglobin the hearing was affected, while Cole and colleagues[11] cite an unpublished observation from the Perinatal Survey that at 7 years of age the children of mothers who smoked showed some intellectual impairment. However, Janet B. Hardy and D. D. Mellits,[18] in a careful study from the U.S.A., could find no evidence of physical or mental handicap at 7 years of age, though they did observe a progressive increase in neonatal death among infants of mothers who smoked more than 20 cigarettes a day.

If these hazards to the fetus are to be avoided, real efforts should be made to educate women on the dangers of smoking, so that at least if they cannot be dissuaded from smoking at other times they will be prepared to give it up as soon as they become pregnant.

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Road Accidents Epidemic

The increase in road accidents in Europe in the past decade has been such that in most technically developed countries they now account for a third to a half of all male deaths in the age group 15-24. The number of drivers under the age of 25 injured in road accidents in the United Kingdom between 1960 and 1970 increased more than three-fold.[1] If this situation had been caused by poliomyelitis or some other disease traditionally associated in the popular imagination with public health the term "epidemic" would be freely used and the alarm bells would be ringing. But they are not.

A report[2] recently issued by the European Conference of Ministers of Transport makes it possible to compare, within limits, the incidence of deaths and injury on the roads in different European countries. It is based on a special survey and it supplements the very inadequate information to be obtained from official returns of road accidents. The wide variety of criteria whereby the different countries record such information renders comparison difficult, but trends occurring over a decade can yield useful information. It is interesting to note that the U.K. comes out well in comparison with other European countries, with the lowest overall increase in road accident fatalities (9%) from 1961 to 1970. It also has the best record for pedestrians, with only an 8% increase. The increase in fatalities among car users in the U.K. was a staggering 86%, yet even that is lower than in any European country for which a valid comparison is possible. A substantial part of this increase must be due to collisions between cars and heavy lorries. W. Gissane and J. Bull[3] have recently analysed the great and growing danger to car occupants in these circumstances.

For child pedestrians, however, we have the highest incidence of casualties in Europe in proportion to the population at risk up to the age of 5 as well as between the ages of 10 and 14. Only Germany has a worse record for the age group 6-9 years, and that only marginally. It is a cause for concern that we should have proportionately four times as many child pedestrian casualties under the age of 6 as does France, and more than three times as many in the age group 10-14. In comparison with the other European countries our problems are shown to be far more concentrated in built up areas, where 56% of our road deaths and 73% of our casualties occur. The risk is particularly high in the older cities where density of population and volume of traffic are high, and income and social class are low.[4] It may also be significant that the U.K. is the only technically developed country in Europe where pedestrians are allowed to cross the road whenever, wherever, and however they like.

The report is cautious, with good reason, about the causes of road accidents. It lists as the main "presumed causes" failure to give way, unsafe speed, pedestrians at fault, drunken driving, and unlawful overtaking. The experts who drew up the report unanimously agreed that unsafe speed is the principal "behavioural" cause, and the relatively low figure returned for drunken driving by most countries is dismissed as being "not in line with the facts," a criticism which is almost certainly true of the major wine-producing countries, most of which have not yet introduced an effective statutory blood alcohol level.

The failure to control the rising mortality and morbidity from road accidents should be regarded by public health authorities as a major epidemiological challenge. The case control surveys on which the alcohol provisions in the Road Safety Act 1967 were based provide a good example of the effectiveness of modern epidemiological techniques in road accident prevention, and it is unfortunate that the role of public health authorities has been limited to dealing with the consequences of road accidents instead of their prevention. The demarcation agreement which applies in most European countries, whereby transport authorities deal with prevention, may be convenient for administrative purposes, but it should be re-examined in the light of the facts now before us. Both the annual report of our Department of Health and Social Services and that of its Chief Medical Officer on "The State of the Public Health" ignore the prevention of road accidents, and the Transport and Road Research Laboratory assigns a relatively low priority to studies on the behaviour of road users while spending large sums of money on developing sophisticated techniques of road transport