observed with expected birthweight. On this basis, babies in group S were 123 g heavier on average (p<0.02) than babies in group N. This is a reverse of a passive smoking effect. The groups were similar in other respects except for the proportion of unemployed husbands. The amounts smoked by the partners in group S showed no dose-response relationship with birthweight, such as might have suggested a causal association (data not shown).

Our evidence does not support a passive smoking effect and indeed suggests the reverse. Why should fetal growth be greater in those exposed to paternal smoking than in those not exposed? One possibility is that a woman who managed to stop smoking because she was pregnant, despite her partner’s smoking, must be highly motivated and may adopt other behaviours (eg, dietary) which are advantageous to pregnancy. It is difficult to disentangle the effects of maternal and paternal smoking when both are highly correlated. We also question the omission of maternal height from the regression analysis of Rubin et al because small stature is closely associated with smoking and with low birthweight.

Department of Social Medicine, Medical School, University of Birmingham. Birmingham B15 2TJ

C. MACARTHUR

PARKINSON’S DISEASE AND PESTICIDES

SIR,—Early-onset Parkinson’s disease is unusual and stimulates a search for a causative factor. However, the use of pesticides by the two men referred to by Dr Bocchetta and Dr Corsini (Nov 15, p 1163) may be purely coincidental, and it is highly speculative to involve the use of pesticides. Furthermore, these seem to be the only processing mills and industrial areas, neither of which are likely to neurotoxic producing a parkinsonian syndrome. London: Academic Press, 1986: 285-89.


FREE RADICALS, LIPID PEROXIDATION, AND PARKINSON’S DISEASE

SIR,—We were not surprised to learn (Sept 13, p 639) that parkinsonian nigral tissue contains slightly increased levels of malondialdehyde (MDA), an indicator of lipid peroxidation. The tissue will contain many dead and dying cells which will peroxidise during their pathological breakdown. Control nigral tissue presumably contains fewer dead cells and will therefore show lower peroxidation levels. Thus, the increased lipid peroxidation observed by Dr Dexter and colleagues may merely be the result rather than the cause of the nigrostriatal cell death. Moreover, although it has been postulated that the parkinsonism-inducing compound methylphenyltetrahydropyridine (MPTP) may kill cells via its metabolite methylphenylpyridinium (MPP+), generating oxygen radicals and subsequent lipid peroxidation,1 we find this evidence for the very convincing.

The cytotoxicity of MPTP and MPP+ in vitro is not blocked by antioxidants.2 MPTP and its primary metabolite MPDP (methylphenylidylydroxypiridinium) have potent direct antioxidant properties themselves.3 Systemic administration of antioxidants to prevent MPTP neurotoxicity has produced very promising results in animal models.4 Furthermore, MPP+ is a very poor generator of oxygen radicals5 and causes very little lipid peroxidation in subcellular fractions. Depleting cellular reduced glutathione (GSH) or compromising the ability of isolated cells to detoxify hydrogen peroxide has no effect on either MPTP or MPP+ cytotoxicity nor does the presence of chelators of transition metals.2,3 Finally, the decrease in GSH observed in the brains of mice injected with MPTP as well as in the substantia nigra of patients with idiopathic Parkinson’s disease5 has been questioned as being causally related to MPTP toxicity.6 Thus, MPP+-induced parkinsonism is unlikely to involve oxygen radicals or lipid peroxidation. Whether or not there is a role for this mechanism in “naturally” induced parkinsonism must remain the subject of further investigation, but Dexter and colleagues’ findings add little to support this hypothesis.

Department of Biomedical and Environmental Health Sciences, School of Public Health, University of California, Berkeley, California 94720, USA

MARTYN T. SMITH

MAKTSA S. SANDY

DONATO DI MONTE


MPTP, SELEGILINE, AND PARKINSONISM

SIR,—Reports of irreversible parkinsonism induced by methylphenyltetrahydropyridine (MPTP) and other chemicals have implications for prevention and treatment. Research into environmental causes of Parkinson’s disease is difficult because the disease has a low rate of genetic concordance, the offending environmental event or events occur in early or middle adult life, and clinical features appear decades later as normal ageing reduces...