Dear Sir,

We have appreciated the letter by Drs Mehta and Anand Kumar as it gives us the opportunity to emphasize some of our recent findings.

In our previous report we have demonstrated that an inverse correlation exists between traffic pollutants and semen parameters. Particularly, environmental SO, NO, CO and Pb decrease sperm motility, kinetics and function in exposed subjects (De Rosa et al., 2003).

Multicentric research performed in Austria, France and Switzerland (Kunzli et al., 2000) has demonstrated that exposure to particulate matter (<10 μm, PM<sub>10</sub>) constituted by CO, SO, NO, deriving from vehicle exhaust and diesel emissions, increases mortality in adults >30 years, respiratory and cardiovascular hospital admissions at all ages, incidence of chronic bronchitis in adults >25 years, bronchitis episode in children <15 years, restricted activity days in adults >20 years and asthma attacks in adults and children. Particularly, air pollution caused 6% of total mortality or more than 40 000 attributable cases per year. About half of all mortality caused by air pollution was attributed to motorized traffic, accounting also for: more than 25 000 new cases of chronic bronchitis in
adults and more than 290,000 episodes of bronchitis in children; more than $0.5 \times 10^6$ asthma attacks and more than $16 \times 10^6$ person-days of restricted activities. In the same period, Samet et al. (2000) demonstrated that an increase of PM$_{10}$ levels in 20 American cities was associated with the risk of death from all causes and, particularly, from cardiovascular and respiratory illnesses. Besides the negative effects on the cardiovascular and respiratory system, pollutants affect sperm quality. In fact, Mehta and Kumar (1997) reported an inverse correlation between environmental levels of PM and sperm count and semen quality. Previously, the prolonged exposure to this particulate extract was shown to reduce sperm motility in vitro (Fredricsson et al., 1993). Finally, it has been demonstrated that these exhausts stimulate hormonal secretion of the adrenal cortex, depresses gonadotrophin release and inhibits spermatogenesis in rats (Watanabe and Oonuki, 1999).

In our study we have not reported on particulate matter as further studies are ongoing to evaluate the role of PM in the exposed subjects.

In addition to the detrimental effects of pollutants on semen quality, the prolonged sitting position might have an independent role as it has been implicated in poor semen quality (Figá-Talamanca et al., 1996; Bhattacharya, 2003) by increasing temperature in the pelvic region (Bujan et al., 2000). It should be noted that the exposure to environmental pollutants in our study group compared with an age-matched group of controls equally working in a sitting position confirms the harmful role of chemical pollutants on the spermatogenesis (De Rosa et al., 2003).

Finally, it is a long time since a number of papers have demonstrated that chronic exposure to pollutants induces a modification of human chromosomes. Particularly it has been demonstrated that lead exposure induce a dissociation of the acrocentric chromosomes (Verschaeve et al., 1979) and severe chromosomal aberrations (rings and dicentric) (Deknudt et al., 1977). All these effects should be kept in mind when studying the spermatogenesis in exposed subjects, because they could worsen negative male fertility potential.

References


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