THE AIR WE BREATHE

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Everybody breathes several times a minute throughout life. Yet how many people stop to think about the quality of the air they inhale?

Respiratory disease, mostly of the airways is second only by a narrow margin, to cardiovascular disease as the commonest reason for admission to our hospital (data form the Health Information Unit). It is also one of the commonest complaints for which patients visit their family doctor and for absenteeism from work.

Air pollution has been associated with respiratory ill health for centuries and in recent years much research effort has gone into quantifying the risks and elucidating the mechanisms. The link between chronic bronchitis and emphysema, tobacco smoking and urban as well as indoor air pollution due to the burning of fuels is well recognised. The infamous London smog of 1952 which killed 4000 people forced the government to legislate and by 1956 Parliament had passed the Clean Air Act. Since then the prevalence and mortality from these diseases has fallen. Other countries have followed suit.

In Malta, asthma is probably the commonest disease affecting airways. The effects of air pollution on respiratory health have been reviewed extensively (1-4) but the relationship to asthma has been the subject of some controversy because of conflicting results (5,6). Many epidemiological studies have shown that asthmatic persons of all ages exposed to short or long term air pollution remains unclear and it becomes more complex as it unravels. There are usually several pollutants involved and the most important seem to be sulphur dioxide, ozone and nitrous oxide. Sulphur dioxide at levels often encountered in real life (9) appears to be the most irritant, producing airway inflammation, hyperresponsiveness and increased sensitivity of afferent airway neurones. The degree of these effects is dependent on the concentration, humidity, air temperature and the previous state of health of the airway. Furthermore, it will depend on concomitant or previous exposure to other pollutants. For example, asthmatics develop symptoms at a much lower concentration than normals when exposed to ozone, (10,11) and nitrous oxide potentiates the effects of sulphur dioxide (12), at levels often below the WHO recommended limites (13). In winter, particulates consisting of soot, water droplets and sulphur and nitrous oxides, in combination with low temperatures and still air, are particularly noxious through their acidic effects on afferent nerve endings and the mucosa of the airway (14). In summer, high temperatures and sunlight act on the same compounds to produce high levels of ozone (photochemical smog) which in turn has detrimental effects on the airway. It is important to simultaneously monitor all known irritants and metereological data if any sense is to be made of the effects on our lungs of the polluted air we breathe.

Why therefore are the prevalence and mortality from this disease steadily rising even where air pollution is being controlled? And why is it that those populations living in heavily polluted areas seem to have a lower prevalence of asthma than those living in relatively clean environments? Some understanding of this apparent paradox is beginning to emerge. There is evidence that low concentrations of pollutants lower the threshold of response to allergens in atopic asthma. Whether or not indoor as well as outdoor pollutants increase sensitization to aeroallergens and therefore increase the prevalence of atopic asthma is unclear. In a study involving over 1000 school children in Sweden and Poland (15), those living in a polluted urban environment had more respiratory symptoms but a lower prevalence of positive skin tests than those from rural areas. However, it seems probable that those living in the country would have had greater exposure to aeroallergens. A recent study from Germany (16) after its reunification also found the prevalence of asthma to be lower in the heavily polluted East. Conversely, bronchitis was commoner in the East than in the West. However, it is now recognised that the age at which there is exposure to aeroallergens and the simultaneous presence or absence of pollutants are important factors which determine the development of respiratory symptoms and asthma. A study from Norway involving more than 500 children showed that those exposed to low

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concentrations of environmental irritants had an increased risk of sensitization to outdoor allergens and this was more marked in those first exposed below the age of three years (17).

It seems that above a certain threshold for both duration of exposure and for concentration, pollution may "protect" against asthma at the expense of producing the more serious conditions of chronic bronchitis and emphysema. One is tempted to speculate therefore, that exposure to low concentrations of pollutants produces only mild inflammation so that simultaneous exposure to aeroallergens allows contact more easily between allergen and the increased numbers of inflammatory cells thus presumably enhancing subsequent sensitization. On the other hand, high concentrations of pollutants induce much more inflammation with the possibility that, like cigarette smoke, they may inhibit immune mechanisms and therefore sensitization. The effects of cigarette smoke on immune mechanisms have been reviewed by Holt (18). Cigarette smoking has a variable influence on T-cells and immunoglobulins. Particularly with increasing age, heavy smoking (> 20/day) suppresses T-cell function and circulating immunoglobulins including IgE, whereas a low level (<10/day) of smoking has the opposite effects. This biphasic response might explain the conflicting results of epidemiological studies. It may explain why in the same studies where asthma is found to be less prevalent, chronic bronchitis is more common. It may also be an explanation for the anomalous albeit uncommon situation where a long term cigarette smoker with or without chronic bronchitis and emphysema develops worsening asthma soon after giving up smoking and in whom symptoms improve when smoking is resumed (18). The observation that smoking protects against sensitisation is not new. Farmers and bird fanciers who smoke are less likely to develop extrinsic allergic alveolitis and also less likely to have precipitins detected in their serum than those who do not smoke (19).

In Malta, vehicular traffic has increased dramatically in recent years and a large proportion of vehicles are badly maintained resulting in even higher emissions of pollutants. The notion that Malta is a windy place and therefore polluted air is all blown away is unfounded. On most days in the early morning hours the air is very still. We are beginning to see (literally) days in winter and in summer when smog blankets our low lying towns where traffic is dense and where the power station is sited. We have very little data on the levels of air pollutants on Malta and so the article by Alfred Vella and colleagues published in this issue is of importance and should be of interest to clinicians and public health officials alike.

It is the impression of several clinicians that asthma in Malta is increasing in prevalence and in severity. This places an onus on the medical profession to study the epidemiology of airway disease in our country and to look with interest at the development of air monitoring programs on our Island.

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ERRATUM: in the article "THE AIR WE BREATHE", para. 4, lines 7-8 should read: "... exposed to short or long term air pollution develop worsening symptoms.[7,8] The role of air pollution ....."