PASSIVE SMOKING AND HEALTH EFFECTS

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The area of the most difficulty in passive smoking is one of establishing exposure or dose. We have, in terms of monitoring, several different pollutants that have been studied. Some are better than others in giving us information about exposure (2, 6). Nitrosamine monitoring is relatively new, and it appears to give us better specific information. The measurement results seem to correlate very well with environmental tobacco smoke concentrations, whereas others may not, because they have other sources (e.g. NO_2, formaldehyde). On the other hand, in a chamber, since nitrosamine measurements are not very cheap, CO can be used although it does not represent other pollutants. Total suspended particulate and respirable suspended particles correlate very highly but have other sources as well. Nicotine would be good, but it adsorbs on surface easily, de-gases randomly, and is more expensive to measure.

What we are aiming at for as a measurement of dose is a valid and reliable method of biological monitoring (2, 4). There are several techniques that have been developed over the past few years. Cotinine, which is a metabolite of nicotine, has a long half life, appears to be quite excellent, and new evidence shows extremely good correlation with exposure. It's reliable, it's good for long exposures, and it can be measured from serum, saliva, or urine. The technique may be method dependent, and is not very cheap. Another method is the measurement of the hydroxyprolineto-creatinine ratio as a measure of NO_2 exposure, but it is affected by other NO_2 sources; this is still being tested.

Other, measurement methods, such as nicotine, are good for very short exposures only; carboyhemoglobin is reasonable to measure for 4-8 hours exposures but thiocyanate has few qualities to recommend it for this purpose. There should be more testing of these methods, and evaluation of their time and concentration determinants.

There are also various attempts at modeling of dose (3, 4).This appears to be the weakest way of estimating dose right now, because there is at least a hundred-fold difference in estimates. The estimates have to be different for different compounds, and they should be more time, concentration, and chemical specific. Also, they are not physiologically meaningful at the present time (4).

In health studies, methodologically we have to contend with many confounding factors (1), with a large amount of misclassification of exposure, and with many interactions that are possible (but which are usually ignored).

In terms of studying the effect side of the equation, we have only certain effects on which we agree and certain in which we don't (1, 3, 5).

There is no doubt in anyone's mind that passive smoking does produce annoyance (6). Annoyance is rapid but plateaus with time. It is subjective and not blinded. The levels required are uncertain. We are at a state of knowledge where we can actually say both smokers and non-smokers prefer not to be exposed. The degree of the preferences is based on where the exposure occurs.

All agree that sensory irritation does occur, as measured objectively and subjectively. There does appear to be a threshold, but the different studies have given different results. Eye irritation does increase with time and concentration. Other irritation (nose, throat, stomach) have not been measured objectively (3, 6).

There are various possibilities of overcoming thus problem: making threshold limit values, reducing emissions, increasing ventilation, protecting patients. Further studies should be in terms of interactions with other air pollutants and sources, since passive smoking occurs within a background of some other pollutants, studying specific populations, and maybe more sensitive individuals.

Infections in children do appear generally to be correlated with mother's smoking and by amount of smoking per day (1). This may be an intrauterine effect and/or a neo-natal effect; we still have to pursue that research. It was thought that one could include warning pregnant females and mothers not to smoke around children.

In terms of pulmonary function, passive smoking possibly affects children's lung funtion growth, but we don't exactly know how much. There is a wide range of results so far, and several studies are under way to see why the differences occur. The effects are mostly on pulmonary flow rates and not on volumes. It also appears to have what can be considered of blunting effect, like active smoking, in terms of response to other irritants, that has to be pursued further. The associated effect of passive smoking on attained height (.45 - .65 cm), but not on growth rate, in one study, is at least due to neo-natal influence.

Passive smoking appears to affect asthmatics; sometimes more specifically those who are reacting allergically. More quantification of that response is needed. As asthma is a highly variable disease, especially research on statistical methods to see why the differences occur is required. Other thoughts include trying to work out other methods of function (like pulmonary clearance), and more studies of chronic disease are needed as well.

In terms of lung cancer, we all know that there are carcinogens, more in side-stream than main-stream tobacco smoke. The research to date needs expansion and improvement especially on dose and response, because of the importance of the issues (4).

Conclusions

1. There is complete agreement on the importance of annoyance reactions towards passive smoking. The annoyance reaction is rapid, but plateaus with time, and subjective, but not blinded. The annoyance threshold level not to be exceeded remains uncertain.

2. Smokers and non-smokers prefer not to be exposed (dependent on place). Therefore it is recommended smokers be curteous.

3. Sensory irritation threshold (objective and subjective) increases with time and concentration.

4. It is recommended that threshold limit values or maximum concentration values standards are being made, emissions reduced, ventilation increased, patients protected, and interactions with other air pollutants and sources studied as well as "sensitive" populations.

5. Infections in children are correlated with mothers' smoking (and amount/day smoked). There are also interactions with parental illness. The effect may be intrauterine and/or neo-natal. Therefore pregnant females and mothers should be warned not to smoke around children.

6. Passive smoking probably affects children's lung function growth some but the effects are different in various studies. The effects mostly are in flow rates (not in volumes). Passive smoking has a blunting effect on the response to other irritants. It affects asthmatics more than others, specifically those reacting "allergically".

7. More research is required on statistical methods, and on measurement methods, methods of pulmonary function (e.g. clearance), more studies of chronic disease, and more research on carcinogens in smoke and lung cancer, especially on dose-response relationships to know of any association effects.

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