HAZARDOUS EFFECTS OF INVOLUNTARY SMOKING

Pages with reference to book, From 148 To 151

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In USA since 1964 the Surgeon General has identified smoking as the single most important cause of preventable mortality, but it is only recently that attention has been focussed on the adverse effects of involuntary passive smoking on human health[1,2]. Involuntary smoking occurs when non smokers are exposed to tobacco smoke of smokers in enclosed environments[3]. There are two main sources of environmental tobacco smoke, namely the main stream (exhaled smoke of the smoker) and the side stream (smoke emitted from the lit end of the cigarette). Qualitatively, the two types of smokes have similar components but midstream smoke has a higher PH, smaller particles and higher concentration of carbon monoxide[1,2]. Approximately 85% of the smoke generated during cigarette smoking consists of side stream smoke but the adverse effect of involuntary smoking depends upon various factors like filtered or non filtered cigarette, low or high tar/nicotine, smoking rate, room size, ventilation and duration of exposure . Markers which measure tobacco smoke exposure in, non smokers include carbon monoxide thiocyanate, nicotine and cotinine, and urinary mutagenes[4]. Of these nicotine[5-11] and cotinine[12-19] have received maximum attention, with cotinine as the most accepted short term marker in epidemiological studies because of its long half life, lack of fluctuations during smoke exposure and its non invasive determination in urine and saliva. Strong correlation exists between urinary cotinine levels and exposure to tobacco smoke[14-16]. On the basis of biochemical markers, the level of exposure to environmeiflal tobacco smoke is approximately equal to smoking 0.1 - 1 cigarette/day in UK[11,14] and upto 2/day in Japan[17]. In areas which are heavily polluted with sidestream smoke (CO 20 um/L) tobacco smoke inhalation by involuntary smokers is equal to half to one cigarette/day[20]. The health effect of environmental tobacco smoke in non smokers married to smokers is 1.41 to 1.87 which drops to 1.09 to 1.45 in non smokers married to non smokers[2] showing a 7.4[21]. 6.8%[22-26] death rate due to lung cancer in non smokers/100,000 person years. Three prospective studies also showed a slightly higher risk of lung cancer in nonsmokers married to smokers[22,27,28] , while 10 of 15 case controlled studies also showed an increased risk of lung cancer in non smokers, married to smokers as compared to those married to non smokers[29-47] while others reported no increased risk[31,33,36,42,43]. Six of these studies showed a dose response relation between passive smoking and lung cancer[29,30,37-40]. Several studies show a strong association of environmental tobacco smoke to squamous cell and small cell carcinoma than with other cell types affected by lung cancer[32,34,35,37,39,41]. No increased risk of lung cancer was reported from USA[33,43], Hong Kong[31,42] and UK[36] in non smoker females married to smokers, however in view of misclassification of exposure[31,33,36,42,43]. interviewer and observer bias[33,36] and improper matching of case/controls[31,42], the results of these studies should be regarded with caution. Most studies though are not comparable in terms of study design, population size, tobacco type, manner of consumption, extent and duration of tobacco smoke in the working place but almost all showed a positive association of lung cancer in passive smokers. The effect of passive smoking on chronic respiratory symptoms has also been studied. Of 9 studies[48-56], S showed a significant association[49,53] while 4 did not[48,54-56]. In one of the studies extending over 20 years[49], the effect of passive smoke exposure at the work place showed a significant reduction in forced and expiratory flow and FEVi in non smokers exposed to smoke than in non smokers not exposed to environmental smokp. Similarly a French study[50] showed a dose response relation between reduction in pulmonary function and increase in number of cigarettes smoked daily by the spouse, moreover percent reduction in forced

expiratory flow was observed only in non smoking females over the age of 40 years. Significant association of exacerbation of asthma with tobacco smoke exposure has been reported in two studies[57,59] while the third showed no association[58]. Variations in results are probably due to variations in patient's characteristics especially their hyper responsiveness[59]. Cardiovascular diseases are also strongly associated with various risk factors, including smoking[60]. Four of five studies showed association of tobacco smoke exposure in non smokers to the occurrence of heart disease[52,61-63]. In one study, with a 12 years follow up of non smokers aged 25 or over, a significant higher mortality rates from arteriosclerotic heart disease was observed in both sexes when they lived with smokers, but a significant dose response trend was observed in feniales only[63]. The possible effects of exposure to parental tobacco smoke on the frequency and severity of acute respiratory illness in children has been studied in 4 prospective and 9 case controlled studies. Almost all of them showed an increas frequency of both upper and lower respiratory problems in children of smokers than non smokers[64-72], moreover mother's smoking had a stronger influence[68,69]. A positive dose response reduction has been found in the children's lung function and the number of smokers within the house. Younger children are more strongly affected than the older ones. Similarly an association of exposure of parental smoke has been found with increased frequency of chronic middle ear effusions and infections in children aged 4 years or below[73-77]. The long term effects of smoke associated problems and reduction in pulmonary function in children has not been studied but the higher incidence of lung cancer in adults exposed to environmental smoke as a public health problem[78]. Further studies should be undertaken to accurately measure the exposure to tobacco smoke in environment.

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