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A Reproductive Epidemiological Study on Pesticide Workers

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INTRODUCTION

Pesticides have become inevitable in the field of agriculture. A majority of the pesticides are insecticides and many of the insecticides are organophosphates. Organophosphates are highly toxic and they affect the nervous system of vertebrate and invertebrate pests. These organophosphorus pesticides have been proved mutagenic in several in vitro and in vivo test systems (Vijayaraghavan et al., 1994; Pluth et al., 1996; Saxena et al., 1997).

Agricultural workers especially those who spray the pesticides have a great risk as they are directly exposed to organophosphates along with other pesticides. There are wide range of studies available on health hazards (Richter et al. 1992; Brega et al. 1998), mutagenicity (Scarpato et al., 1996a,b; Brega et al., 1998) and reproductive performance (Kharazzi et al., 1980; Rita et al., 1987; White et al., 1988) of these workers.

Besides agricultural workers, men who are employed in the production of pesticides also have occupational exposure to pesticides. However, they are also exposed to several toxic chemicals which are used in the synthesis of pesticides. Studies on health hazards reported decreased plasma and serum cholinesterase activity and alterations in liver function profiles (Kashyap, 1984; Kossmann, 1997; Brega et al., 1998). Cytogenetic studies have reported increased frequency of chromosomal aberrations and SCEs in pesticide workers. (Stocco et al., 1982; Padmavathi et al., 2000). However studies on reproductive performance in pesticide workers are meagre (Whorton et al., 1977), hence we attempted to study the reproductive histories of male workers employed in a organophosphorus pesticide industry.

MATERIALS AND METHODS

A total of 256 male workers in the age group of 21-50 years who were employed in the pesticide industry for a period of 1-27 years were selected for the study. 204 individuals with the same age group and socio-economic status were studied as controls. The workers were clinically examined and information on duration of service, health status, medication, exposure to chemicals, smoking and drinking habits, tobacco and pan chewing habits, type of marriage and reproductive history was collected using a standard questionnaire. Reproductive history included data on number of children, abortions, stillbirths, premature births, neonatal deaths and congenital defects.

The pesticide industry workers were involved in the manufacturing process of five different organophosphorus pesticides, monocrotophos, chloropyrifos, phorate, phosalone and ethion. During the process the workers handled a wide range of chemicals like chlorine, caustic lye, hydrogen chloride, phosphorus pentasulphide, methylene bromide, farmaldehyde, ethylmercaptan,dichloroethane, acrylonitrile, organic solvents etc., that were used as raw materials. Except for wearing boots and uniform the workers were not using any other protective equipment during their work. They worked for 8 hours per day for six days a week throughout the year. The statistical analysis of the data was done using χ^2 test.

RESULTS AND DISCUSSION

The frequency of livebirths decreased significantly from 96.76% in the non-smoker control group to 89.46% in the non-smoker exposed group. A significant increase in the incidence of abortions (7.20%) was observed in the non-smoker exposed group compared to 2.51% abortions in the non-smoker control group. There was an increase in stillbirths (1.29%), neonatal deaths (0.30%), congential defects (0.77%) and premature births (0.26%) in the exposed group when compared to the controls. A slight decrease was observed in the number of fertile males (98.21%) in the exposed group when compared with the

Table 1: Reproductive histories of male workers in a pesticide industry

Group/ Duration of exposure in years	*No. of Males*	*No. Fertile Males*	*No. Pregnancies*	*No.of live-births*	*No. of abortions*	*No. of still-births*	*No. of neo-natal deaths*	*No. of congenital defects*	*No. of pre-mature births*
Control Group									
Non-Smokers	143	142 (99.30)	678	656 (96.76)	17 (2.51)	3 (0.44)	2 (0.30)	0 (0.00)	0 (0.00)
Smokers	61	60 (98.36)	247	226* (91.50)	15* (6.07)	2 (0.81)	2 (0.81)	1 (0.41)	1 (0.41)
Exposed group (1-27 Years)									
Non-Smokers	168	165 (98.21)	389	348* (89.46)	28* (7.20)	5 (1.29)	4 (1.03)	3 (0.77)	1 (0.26)
Smokers	88	86 (97.73)	182	139* (76.37)	27* (14.84)	7* (3.85)	5 (2.75)	2 (1.10)	2 (1.10)

Note: Values in the parantheses indicate percentage
* $p<0.05$

control group (99.30%). However, the increase in the stillbirths, neonatal deaths, congenital defects, premature births and decrease in fertility rate observed in the workers was statistically insignificant when compared to the control group.

In the smoker exposed group, there was a significant decrease in the percentage of livebirths (76.37) when compared to the value (91.50) observed in the smoker control group. The percentage of abortions and stillbirths in the workers was 14.84 and 3.85 while the control group showed 6.07% abortions and 0.81% stillbirths. This increase in the incidence of abortions and stillbirths in the exposed group when compared to the control group was found to be statistically significant. The frequencies of neonatal deaths, congenital defects and premature births were higher in the exposed group than in the control group. The percentage of fertile males was 97.73 in the exposed group and 98.36 in the control group. However, no statistically significant differences in the frequencies of neonatal deaths, congenital defects, premature births and fertile males were observed between the exposed and control groups.

Environmental pollutants induce mutations in the persons exposed and thereby increasing the risk for genetic defects in the subsequent generations. End points such as congenital malformations and spontaneous abortions following paternal exposure can also be considered as indicators of heritable mutagenic effects (Narod et al., 1988). The results of the present study clearly presented evidence for the impaired reproductive performance in the male workers of a pesticide industry. The results are comparable with those of Rita et al. (1987) who reported high incidence of spontaneous abortions and stillbirths in the wives of grape garden workers exposed to nine different pesticides during their work. Earlier, increased infertility was reported in men involved in pesticide spraying (Kharazzi et al., 1980) and in pesticide industry workers (Whorton et al., 1977). In a case control study, association between the congenital abnormality spinabifida and maternal agricultural occupation was reported (Blatter et al., 1996). In another study association between exposure to agricultural chemicals and three anomalies, stillbirths, spinabifida and hydrocephalus was reported (White et al., 1988).

Several epidemiological studies have revealed a wide range of reproductive effects of occupational exposure in the workers employed in a variety of occupational set ups like crystal glass industry (Wingren and Persson, 1998), rubber industry (De Celis et al., 2000) metal industry (Weizu et al., 1993), chemical plants (Pimenova et al., 1997), dry cleaning workshops (Olsen et al., 1990), hairdressing saloons (Kersemaekers et al., 1997), cement factory (Fatima et al., 1994) and agriculture (White et al., 1998).

The present study has shown more intensified impaired reproductive performance in the smoking groups when compared to the non-smoking groups suggesting the ill effects of smoking

on the reproductive performance. In contrast to this, a study on women occupationally exposed to chemicals reported high incidence of malformations in the new borns in non-smoking women compared to smoking women (Hruba et al., 1999). The adverse effects on the reproductive performance of the pesticide industry workers may be not only due to exposure to organophosphorus pesticides but also due to exposure to a variety of chemicals that are used as raw materials. Studies in animal models have established the genetic effects of different organophosphorus pesticides (Pawan and Khatdare, 1983; Jayashree et al., 1994; Fahmy and Abdalla, 1998) and some of the chemicals such as 1,2-dichloroethane, benzene, ethylalcohol that are used in the industry for the manufacture of pesticides also have been reported to induce sterility, foetotoxicity and teratogenecity in rats (Tatrai et al., 1980; Kuna and Kapp, 1981; Raymond et al., 1982; Slozina et al., 1990). In a reproductive epidemiological study on women occupationally exposed to benzene, gasoline and hydrogen sulphide, increased risk of spontaneous abortions was reported (Xu et al., 1998). So occupational exposure to organophosphorus pesticides and chemicals could be a potential hazard.

In the present study, since the workers are exposed to a variety of pesticides and number of chemicals, it is difficult to attribute the adverse reproductive effects to any particular pesticide or chemical. This might be due to the cumulative effect of exposure to different organophosphorus pesticides and chemicals. Hence measures should be taken to minimise long term exposure to the pesticides and the chemicals at the work place. Otherwise, undue exposure might affect the reproductive performance of the workers and increase birthdefects in their children.

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