DDT and Male Reproductive System

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A brief on film 3 billion and counting given by Attaran (2010) made us to present facts about DDT. Popularly known DDT is dichlorophenyltrichloroethane (C₆H₄Cl₂), CH₂Cl₂ and widely used as insecticide. This stands as a lead item in controlling malaria. It was established as a pollutant of agriculture soil (Al-Wabel et al., 2011), well water (Sabdon et al., 2008), ground water (El-Saeid et al., 2011), lake water (El-Bestawy et al., 2000) and river water (Kalanter and Ebadi, 2006). Restriction was imposed on its use due to entry into food chain. It was reported as a residue in fish (Musa et al., 2010). Initially believing DDT was not toxic to human but ban was imposed for ecological reasons (Rogan and Chen, 2005). Research from animal experiments, clinical studies and surveys conducted at different parts of the world showed damaging effects of DDT on human and animal life. Action of DDT and its metabolite on male reproductive system alone depicted the seriousness of situation. Some important observations made by different groups of workers in animal and human are presented here.

DDT was identified as a hormone disruptor (Gray, 1998) which directly acted on organ by blocking the action of androgen receptor in animal (Gray, 1998; Kelce et al., 1995; Longnecker et al., 2002; Smith et al., 1972) and in human (Gray, 1998) or through the regulating mechanism of hormone (De Jager et al., 2006).

DDT delayed puberty in rat and human (Gray, 1998). The reduction in size of accessory glands was observed (Gray, 1998). It was shown as antagonist to androgen at the level of prostate gland in mice (Smith et al., 1972). DDT blocked androgen induced gene expression in vitro which altered sex differentiation in male rat (Gray, 1998).

Exposing to DDT during embryonic stage in domestic rooster lead to cloacal deformities in adulthood, causing an abnormal semen flow, deformations in testes and reduction in seminiferous tubular area (Blomqvist et al., 2006). In rat which reduced sperm count (Gray, 1998). Longnecker et al. (2002) suspected increased male reproductive defects like hypospadias and cryptorchidism may be due to the increased use of DDT for insect control. Very recently Bornman et al. (2010) examined 3210 new born boys in a malarial area and found nearly 11% had urogenital birth defects. Tren et al. (2010) disagreed to the entire study. Chemicals with estrogenic or antiandrogenic compound shall lead to hypospadias (Nassar et al., 2010) or cryptorchidism (Weidner et al., 1989). Genetic cause was considered as a rare (Hiort et al., 1994) a minor (Allara et al., 1995) or as a major cause (Wang and Baskin, 2008) for this. A study on 2033 workers who were exposed to DDT at occupational level showed they had a chance of baby with birth defect
Kristensen et al. (1997) had similar observation with Norwegian farmers. Others did not find any relation between DDT/DDE and hypospadias (Longnecker et al., 2007).

Decrease in semen quality due to environmental factors was an established fact (Fritz and Czeizel, 1996). Some workers observed decreasing reproductive health and declining semen quality due to exposure to DDT (Salazar-Garcia et al., 2004). Low semen quality abnormal morphology, low concentration and motility are associated with p, p-DDE (Hauser et al., 2003). A similar study with 47 workers underwent detailed investigation including blood level of inhibin, FSH and E2. Inhibin was low and FSH and E2 level was high (Dalvie and Myers, 2006). Their semen evaluation showed low sperm count and motility (Dalvie et al., 2004; Dalvie and Myers, 2006). The results indicated the damage caused was at the level of hormone regulation. Detailed semen study conducted by Aneck-Hahn et al. (2007) showed decreased semen volume and sperm motility with increase in percentage of sperm with cytoplasmic droplet and in the incident of teratozoospermia and oligozoospermia. Insufficient sperm chromatin condensation, present was positively correlated to p, p-DDE concentration (De Jager et al., 2006). The study also indicated adverse effect on testicular function and or the regulation of reproductive hormone (De Jager et al., 2006). Children born to father with poor semen quality have a chance to be born with hypospadias (Fritz and Czeizel, 1996; Carlson et al., 1996). Assisted reproductive technology is helpful to make a father from a man with low semen quality. This could be a responsible factor for hypospadias. A tendency toward low quality of semen always caused anxiety in infertile couple and they approached available mode to become parents.

Mother might be a responsible factor for male offspring with urogenital defects as amniotic fluid was seen as contaminated with DDE (Foster et al., 2000). A study showed mothers’ consumption of contaminated sport fish with pesticides lead to urogenital defect in male offspring (Mendola et al., 2005). More level of chemicals in mother’s placenta was observed (Fernandez et al., 2007). Waliszewski et al. (2005) observed persistent level of pesticides in blood serum lipid in women bearing babies with cryptorchidism. Such mothers continued to give chemicals through milk at one to three months after giving birth (Damgaard et al., 2006). DDT gets deposited in adipose tissue also (Alle et al., 2009).

Toppari et al. (1996) reported deteriorating in male reproductive health at spread in Europe showing with declining semen quality could be with a reason originated in fetal life like exposure to any substance like DDT, which contain estrogenic or anti-androgenic substances present in environment. High rate of male involuntary infertility reported from two cities, Surat and Ahmedabad, infested with malaria and where DDT was lavishly sprayed, sprinkled and spread (Skandhan and Mazumdar, 1982; Skandhan et al., 1982, 1986). We considered air pollutants from industries could be a responsible factor, though we did not explore the association of the incident with that of DDT.

REFERENCES


