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Low-Dose Agrochemicals and Lawn-Care Pesticides Induce Developmental Toxicity in Murine Preimplantation Embryos

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Abstract and Introduction

Abstract

Occupational exposures to pesticides may increase parental risk of infertility and adverse pregnancy outcomes such as spontaneous abortion, preterm delivery, and congenital anomalies. Less is known about residential use of pesticides and the risks they pose to reproduction and development. In the present study we evaluate environmentally relevant, low-dose exposures to agrochemicals and lawn-care pesticides for their direct effects on mouse preimplantation embryo development, a period corresponding to the first 5-7 days after human conception. Agents tested were those commonly used in the upper midwestern United States, including six herbicides [atrazine, dicamba, metolachlor, 2,4-dichlorophenoxyacetic acid (2,4-D)], pendimethalin, and mecoprop), three insecticides (chlorpyrifos, terbufos, and permethrin), two fungicides (chlorothalonil and mancozeb), a desiccant (diquat), and a fertilizer (ammonium nitrate). Groups of 20-25 embryos were incubated 96 hr *in vitro* with either individual chemicals or mixtures of chemicals simulating exposures encountered by handling pesticides, inhaling drift, or ingesting contaminated groundwater. Incubating embryos with individual pesticides increased the percentage of apoptosis (cell death) for 11 of 13 chemicals ($p \leq 0.05$) and reduced development to blastocyst and mean cell number per embryo for 3 of 13 agents ($p \leq 0.05$). Mixtures simulating preemergent herbicides, postemergent herbicides, and fungicides increased the percentage of apoptosis in exposed embryos ($p \leq 0.05$). Mixtures simulating groundwater contaminants, insecticide formulation, and lawn-care herbicides reduced development to blastocyst and mean cell number per embryo ($p \leq 0.05$). Our data demonstrate that pesticide-induced injury can occur very early in development, with a variety of agents, and at concentrations assumed to be without adverse health consequences for humans.

Introduction

Recent epidemiologic studies suggest that parents working in areas of high pesticide application are at increased risk for adverse reproductive outcomes such as infertility (Fuortes et al. 1997; Greenlee et al. 2003; Smith et al. 1997), poor fertilization (Tielemans et al. 1999), fetal death (Arbuckle and Sever 1998; Saxena et al. 1983), and congenital anomalies (Bell et al. 2001a; Garry et al. 1996, 2002). Residential pesticide exposures and their effects on reproductive health are less well understood. A few studies suggest that maternal exposures to pesticides used around the home are associated with risk of stillbirth and fetal deaths (Bell et al. 2001b; Pastore et al. 1997; Savitz et al. 1989). Decreased birth weight and length of newborns have been associated with high levels of chlorpyrifos in plasma samples of urban minority women (Perera et al. 2003).

Timing, combinations of agrochemicals, duration of exposure, and dose may play critical roles in pregnancy outcomes. Bell et al. (2001a) reported that maternal pesticide exposures occurring during the third to eighth weeks of pregnancy have the greatest impact on fetal deaths. This temporal association strengthened when the pesticides were applied within 1 mi² of the maternal residence. Timing of paternal pesticide exposures may also be important. Arbuckle et al. (1999a) reported that exposures to phenoxy herbicides occurring in fathers 3 months before

