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Adolescents exposed to the World Trade Center collapse have elevated serum dioxin and furan concentrations more than 12 years later☆

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ABSTRACT

Background: The collapse of the World Trade Center (WTC) on September 11, 2001 released a dust cloud containing numerous environmental contaminants, including polychlorinated dibenzo-*para*-dioxins and polychlorinated dibenzofurans (PCDD/Fs). PCDD/Fs are toxic and are associated with numerous adverse health outcomes including cancer, diabetes, and impaired reproductive and immunologic function. Prior studies have found adults exposed to the WTC disaster to have elevated levels of PCDD/Fs. This is the first study to assess PCDD/F levels in WTC-exposed children.

Methods: This analysis includes 110 participants, a subset of the 2014–2016 WTC Adolescent Health Study, a group of both exposed youths who lived, attended school, or were present in lower Manhattan on 9/11 recruited from the WTC Health Registry (WTCHR) and unexposed youths frequency matched on age, sex, race, ethnicity, and income. Our sample was selected to maximize the contrast in their exposure to dust from the WTC collapse. Questionnaire data, including items about chronic home dust and acute dust cloud exposure, anthropometric measures, and biologic specimens were collected during a clinic visit. Serum PCDD/F concentrations were measured according to a standardized procedure at the New York State Department of Health Organic Analytical Laboratory. We used multivariable linear regression to assess differences in PCCD/Fs between WTCHR and non-WTCHR participants. We also compared mean and median PCDD/F and toxic equivalency (TEQ) concentrations in our cohort to 2003–4 National Health and Nutrition Examination Survey (NHANES) levels for youths age 12–19.

Results: Median PCDD/F levels were statistically significantly higher among WTCHR participants compared to non-WTCHR participants for 16 out of 17 congeners. Mean and median TEQ concentrations in WTCHR participants were > 7 times those in non-WTCHR participants (72.5 vs. 10.1 and 25. 3 vs. 3.39 pg/g lipid, respectively). Among WTCHR participants, median concentrations of several PCDD/Fs were higher than the NHANES 95th percentiles. After controlling for dust cloud exposure, home dust exposure was significantly associated with higher PCDD/F level.

Conclusions: Adolescents in lower Manhattan on the day of the WTC attack and exposed to particulate contamination from the WTC collapse had significantly elevated PCDD/F levels > 12 years later compared to a matched comparison group, driven by chronic home dust exposure rather than acute dust cloud exposure. PCDD/F and TEQ levels substantially exceeded those in similar-aged NHANES participants. Future studies are

Abbreviations: 9/11, September 11, 2001; AhR, aryl hydrocarbon receptor; DHQ, Diet History Questionnaire; LOD, level of detection; NHANES, National Health and Nutrition Examination Survey; NYU, New York University; PCB, polychlorinated biphenyl; PCDD, polychlorinated dibenzo-*para*-dioxins; PCDF, polychlorinated dibenzofurans; TEF, toxic equivalency factor; TEQ, toxic equivalency quotient; WHO, World Health Organization; WTC, World Trade Center; WTCHR, World Trade Center Health Registry

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warranted to explore associations of PCDD/Fs with health and developmental outcomes among individuals exposed to the WTC disaster as children.

1. Introduction

The collapse of the World Trade Center (WTC) on September 11, 2001 (9/11) resulted in the immediate release of a dust cloud containing large amounts of environmental pollutants from burning jet fuel and pulverized construction material, plastics, and electronics, which were deposited across downtown Manhattan, New York City, as well as from the continuous release of smoke from fires that burned for > 3 months after the disaster (Landrigan et al., 2004). Among these toxic chemicals were polychlorinated dibenzo-*para*-dioxins and polychlorinated dibenzofurans (PCDD/Fs), byproducts of the combustion process (Rappe, 1994). Elevated concentrations of PCDD/Fs were found on samples swabbed from exterior window surfaces (Rayne et al., 2005) and in samples of dust, water, sediment, and sewage collected in and around the WTC site (Litten et al., 2003). Elevated PCDD/F levels have been reported in serum samples from firefighters who responded to the disaster (Edelman et al., 2003) and in plasma samples from pregnant women in the immediate vicinity (Wolff et al., 2005) and from state and National Guard workers assigned to the site in the weeks after the collapse (Horii et al., 2010). To date, no assessment of exposure to these chemicals has been conducted in local children, who may have been uniquely vulnerable to the exposures' potential toxic effects.

PCDD/Fs are two related families of compounds produced when carbon and chlorine combine at high temperatures. Because they are lipophilic and tend to bioaccumulate in the food chain, the primary sources of human exposure are high-fat foods and breastmilk (Centers for Disease Control and Prevention). In the case of the WTC disaster, children would additionally have been exposed to PCDD/F-containing dust particles, especially those that accumulated inside their homes through contaminated ventilation systems and persisted in upholstered furniture and carpeting (U.S. Environmental Protection Agency, 2002). PCDD/Fs have a half-life of approximately 7 years in adults (Michalek et al., 2002). Although they are reported to be eliminated more quickly in those age < 18 years (Kerger et al., 2006), they may pose particular risks to this population, which is undergoing critical periods of biological development.

PCDD/Fs are associated with a wide range of adverse health outcomes. Acute toxicity commonly results in chloracne, a condition resembling severe acne that can last for years (Sorg, 2014). Longitudinal studies of exposed adults have found elevated incidence of diabetes, multiple cancers, and altered reproductive and immunologic function (Pesatori et al., 2003; Wang et al., 2008; Kuwatsuka et al., 2014; Nishijo et al., 2014; Li et al., 2015), while follow-up studies of children exposed in utero or through breastfeeding have found links with impaired cognitive and behavioral function (Nakajima et al., 2006; Neugebauer et al., 2015; Tran et al., 2016) and, among boys, altered semen quality (Mocarelli et al., 2011). The biological mechanism underlying these associations involves activation of the aryl hydrocarbon receptor (AhR), which stimulates production of xenobiotic-metabolizing enzymes. By persistently activating the AhR, PCDD/Fs disrupt its normal homeostatic function, leading to disruptions in development, cell-cycle control, and tumor suppression (White and Birnbaum, 2009). Toxicity of individual PCDD/Fs is determined by each congener's binding affinity for the AhR relative to that of 2,3,7,8-TCDD, the most potent congener in the series, and these toxic equivalency quotients (TEQs) may be summed to approximate the total toxicity of the mixture (van den Berg

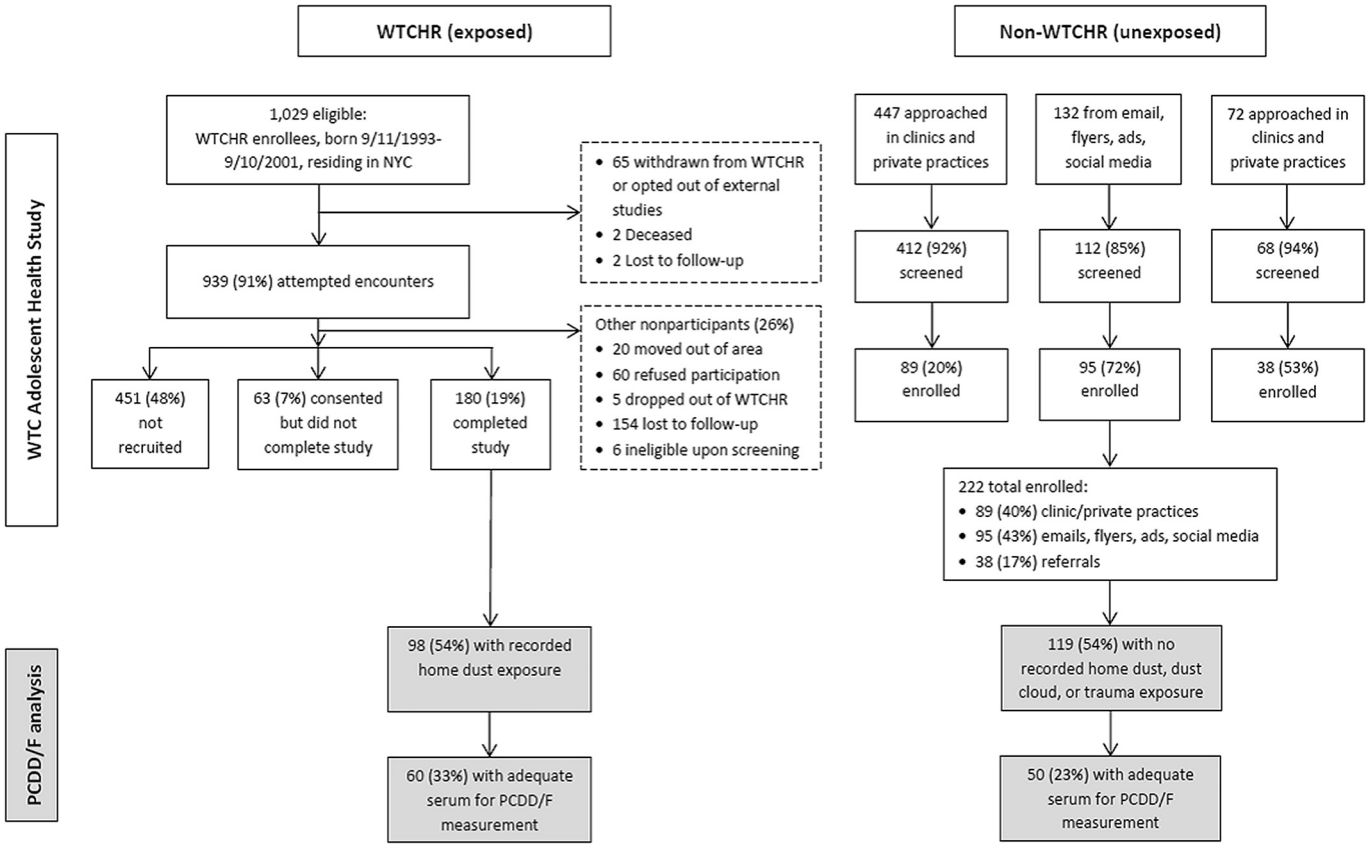

Fig. 1. World Trade Center Health Registry (WTCHR) and non-WTCHR participants in the WTC Adolescent Health Study who were included in the current analysis.