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Associations of World Trade Center exposures with pulmonary and cardiometabolic outcomes among children seeking care for health concerns

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Abstract

Objective—Prior research on the physical health of children exposed to the World Trade Center (WTC) attacks has largely relied on parental report via questionnaire. We examined the impact of clinically-reported exposures on the physical health of children who lived and/or attended school in downtown Manhattan on September 11, 2001.

Study design—We performed a cross-sectional study of 148 patients who presented to the WTC Environmental Health Center/Survivors Health Program, and were ≤18 years old on September 11, 2001.

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Results—38.5% were caught in the dust cloud from the collapsing buildings on September 11; over 80% spent ≥1 day in their home between September 11 and 18, 2001; and 25.7% reported home dust exposure. New-onset nasal/sinus congestion was reported in 52.7%, while nearly one-third reported new gastroesophageal reflux (GERD) symptoms. Prehypertension or hypertension was identified in 45.5%. Multivariable regression with exposure variables, body mass index category, and age as covariates identified strongest associations of dust cloud with spirometry (17.1% decrease in maximum midexpiratory flow). Younger children experienced increased peripheral eosinophils (+0.098% per year, p=0.023), while older children experienced more new-onset GERD (OR 1.17, p=0.004), headaches (OR 1.10, p=0.011), and prehypertension (OR 1.09, p=0.024). Home dust exposure was associated with reduced high-density lipoprotein (−10.3 mg/dL, p=0.027) and elevated triglycerides (+36.3 mg/dL, p=0.033).

Conclusions—While these findings cannot be assumed to generalize to all children exposed to the WTC attacks, they strongly suggest the need for more extensive study of respiratory, metabolic, and cardiovascu lar consequences.

Keywords

Disaster medicine; World Trade Center; Children's environmental health; Asthma; Obesity; Blood pressure

1. Introduction

The collapse and burning of the World Trade Center (WTC) towers exposed thousands to a complex mixture of dust, debris, and jet fuel combustion byproducts (Landrigan et al., 2004). Physical health consequences for rescue/recovery workers and local community members are documented in multiple and distinct studies (Crowley et al., 2011; Dalton et al., 2010; Izbicki et al., 2007; Prezant et al., 2002; Reibman et al., 2005, 2009). Community members, including children, had potential acute exposures from the fires and dust cloud from collapsing buildings on the September 11, 2001, sub-acute exposures from the fires and resuspended dust within the first week, and/or chronic exposures from resuspended dust and incompletely cleaned homes/schools (Friedman et al., 2011). Exposed pregnant women have been studied for consequences in their children (Berkowitz et al., 2003; Lederman et al., 2004; Wolff et al., 2005), and authors have related psychological stress in children to the disaster (Chemtob et al., 2008). However, little is known about physical health impacts on the nearly 46,000 children who lived and/or attended school in downtown Manhattan (Landrigan et al., 2004), a large number of whom continued to reside near the WTC site in the months after the tragedy.

The WTC Health Registry (WTCHR), a national monitoring program sponsored by the National Institute for Occupational Safety and Health, includes questionnaire-derived parental reports on their children exposure and health consequences. A prior report identified 45% of respondent children with WTC dust cloud exposure, over half of whom reported ≥1 new or worsened respiratory symptom post-exposure; 5.7% reported new post-September 11 asthma, a cumulative prevalence well above expected (Thomas et al., 2008b). Possible etiologic origins include exposure to alkaline dust/smoke in the immediate aftermath (Lioy et al., 2002) and combustion products released by fires burning >3 months after the event (Landrigan et al., 2004). Psychological trauma (Islam et al., 2011), as well as consequent psychopathology, may also contribute.

Physiologic/physician corroboration of these parent-reported, questionnaire-derived findings is lacking. Szema et al. investigated asthma diagnosis, medication use, and limited spirometry data in a group of schoolchildren in Chinatown, reporting increased asthma medication use with greater proximity to the WTC site (Szema et al., 2009). To our knowledge no study has performed comprehensive physiologic assessment of respiratory/ other physical health effects in exposed children.

Especially for those children with asthma and other respiratory consequences directly related to the WTC disaster, obesity, insulin resistance and other cardiometabolic consequences may well exist. Increased risk of obesity among children from asthma is well documented, possibly due to decreased reduced functional lung and exercise capacity (Bibi et al., 2004; Figueroa-Muñoz et al., 2001; Gilliland et al., 2003; Shore, 2008; To et al., 2004). Further dietary imbalances may have been produced by exposure to WTC dust or by witnessing the events of September 11, 2011 and experiencing resultant mental health consequences, such as depression, anxiety and post-traumatic stress (Goodman and Whitaker, 2002; Richardson et al., 2003).

The cardiovascular consequences of outdoor air pollutants are best documented in the context of adult exposure, dating to the Six Cities study (Dockery et al., 1993; Krewski et al., 2004). While the cardiovascular consequences in children are not well documented, children's unique vulnerability to these pollutants is well documented for respiratory illnesses (Roy et al., 2011; Sheffield et al., 2011; Trasande and Thurston, 2005). It is within the context of the emerging obesity epidemic (Freedman et al., 1999; Ogden et al., 2008) in the United States in which the plausibility of accelerated and early cardiovascular disease in children arises, through the coupling of oxidative stress from outdoor air pollutant exposures and increased lipid deposition in early life (Ceriello and Motz, 2004; Griendling and FitzGerald, 2003; Heitzer et al., 2001). Indeed, DNA adducts have been identified as a marker of oxidative stress due to air pollution leading to cardiovascular diseases (Autrup et al., 1999; Risom et al., 2003). These same DNA adducts have been identified as elevated among highly exposed newborns born to mothers with increasing proximity to the WTC site (Perera et al., 2005). Adolescence may also mobilize highly lipophilic chemicals absorbed into fatty stores earlier in life, increasing bioavailability and potentially acutely increasing toxicity to multiple organ systems.

The WTC Survivors Health Program (WTCHP) at Bellevue Hospital is funded to provide clinical care to children who lived/attended school near the WTC site. We describe results from 148 clinical histories, physical examinations, and laboratory data of patients who presented to the WTCHP, and were ≤18 years of age on September 11, 2001. We examined associations of acute exposure to the dust cloud, sub-acute exposure (within the week after the towers fell) and chronic household exposures with respiratory, metabolic and cardiovascular outcomes.

2. Methods

2.1. Patient population

The WTC Environmental Health Center was established in 2005, and children were first seen in July 2006. In addition to self referrals, patients were identified through the WTCHR, and community-based organizations. Patients underwent a standardized questionnaire, spirometry, and blood tests. All patients, parents and/or guardians signed New York University IRB-approved consents. Child assent was obtained as appropriate. Data were analyzed from the initial clinical visit, occurring on average 7.83 years after September 11, 2001 (range: 4.83–10.19).

2.2. Questionnaire data

Data included date of birth, gender, home and school/employment address on September 11, 2001, race, and ethnicity. Home and school linear distance to the WTC site was calculated using Google Earth (Mountain View, CA) software and categorized into b1 mile and ≥1 mile. An initial question asked patients whether they were caught in dust cloud or not, with a subsequent question asking to what degree hair, skin and clothing were covered with dust, and whether the dust was easily brushed off/otherwise noticeable. We utilized the latter question to develop a high dust cloud exposure category within the dust cloud exposure group, corresponding to reporting being covered in dust. Patients identified whether they were evacuated from their home, and if so, when they were evacuated and returned, if at all. We categorized these subacute exposures into categorical presence/absence at their home residence ≥1 day in the period September 11–18, 2001. Follow-up questions inquired about degree of dust in home (self-reported as heavy/light, collapsed for this analysis to presence/absence).

2.3. Clinical history

Patients and/or their parent/guardian were asked about medical history and symptoms before September 11, 2001 including provider-diagnosed asthma, persistent headache, sinus congestion and/or nasal drip apart from a cold, rash, allergic symptoms and persistent heartburn or acid indigestion. They were then asked whether any of these symptoms/conditions developed after the WTC disaster, and to identify when. We categorized development of a new clinical symptom/condition based upon its development after September 11, 2001, excluding preexistence of the symptom/condition, and quantified time to symptom/condition development as a latency variable.

2.4. Pulmonary function

Spirometry (Masterscreen IOS; Viasys Healthcare, Yorba Linda, CA) was performed in accordance with American Thoracic Society/ European Respiratory Society standards (Pellegrino et al., 2005). Forced expiratory volume in the first second (FEV_1), forced vital capacity (FVC), FEV_1/FVC and maximum midexpiratory flow (MMEF) were referenced to published predictive equations for Whites, African Americans and Mexican Americans (Miller et al., 2005). White reference equations were utilized for Asian and unidentified race/ethnicity groups, and Mexican-American reference equations were utilized for all

Hispanics. Spirometry patterns were classified as normal (FEV_1/FVC and FVC≥5th percentile); obstructive (FEV_1/FVCb5th percentile); or isolated low FVC (FVCb5th percentile with normal FEV_1/FVC) (Hankinson et al., 1999). For purposes of comparison with the study population, we analyzed spirometry from 1962 b20 years old children in NHANES 2007–10 using the identical reference equations.

2.5. Anthropometry and blood pressure

Weight was collected using a digital scale (Seca, Hamburg, Germany) to the nearest 0.1 kg. Height was measured to the nearest 0.1 cm using a Seca 225 stadiometer. Blood pressure was measured once using a Philips SureSigns VS3 oscillometric sphygmomanometer (Eindhoven, Germany) with appropriate cuff size for arm length, following American Heart Association guidelines (Urbina et al., 2008). Body mass index (BMI) Z-scores were derived from 2000 Centers for Disease Control and Prevention (CDC) norms, incorporating patient height, weight and gender; overweight and obese were categorized as BMI≥25 and ≥30, respectively or BMI Z-score≥1.036 and ≥1.64 (Grummer-Strawn et al., 2010; Ogden et al., 2002). Calculation of systolic/diastolic blood pressure (BP) Z-scores utilized mixed-effects linear regression models derived using data from 1999 to 2000 CDC National Health and Nutrition Examination Survey (NHANES) (National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents, 2004). Height Z-scores derived from CDC norms, gender and age were input to compute expected systolic/diastolic BPs (Ogden et al., 2002), and BP Z-scores were calculated from measured BPs using the formula $Z=(x-\mu)/\sigma$, where x is measured BP, μ is expected BP, and σ is derived from NHANES data. We categorized BP outcomes into present/absent prehypertension (BP≥90th percentile for age/height Z-score/gender or systolic BP≥ 120 mm Hg or diastolic BP≥80 mm Hg) and present/absent hyper-tension (BP≥95th percentile for age/height Z-score/gender or systolic BP≥140 mm Hg or diastolic BP≥90 mm Hg). For purposes of comparison with the study population, we analyzed anthropometric and blood pressure data from 6 to 19 year old children in NHANES 2003–8 using an identical approach.

2.6. Metabolic evaluation

Random, nonfasting blood samples were analyzed for thyroid stimulating hormone (TSH), triglycerides (log-transformed to account for skewed distribution), high-density lipoprotein (HDL), aspartate aminotransferase (AST), alanine aminotransferase (ALT) and percent and (log-transformed) total eosinophils from the whole blood count differential. We chose to analyze nonfasting triglycerides and high-density lipoprotein (HDL) in light of recent literature suggesting minimal differences relating to length of fasting in children (Steiner et al., 2011) and adults (Langsted et al., 2008), recognizing ongoing debate about utility of nonfasting levels (Mora et al., 2008; Nordestgaard and Benn, 2009). In subsequent logistic regressions, we followed cutpoints previously applied in analyzing metabolic syndrome prevalence in NHANES 2001–6 (Johnson et al., 2009). TSH was categorized into high/normal/low following age-specific, American Association for Clinical Chemistry criteria (Demers and Spencer, 2003). We followed the practice of utilizing 30 U/L ALT as a cutpoint to identify non-alcoholic fatty liver disease (NAFLD) (Fraser et al., 2007). For

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purposes of comparison with the study population, we analyzed anthropometric and blood pressure data from 6 to 19 years old children in NHANES 2003–8 (for ALT, AST and HDL) and NHANES 2009–10 (for TSH and eosinophils) using an identical approach.

2.7. Statistical analysis

Multivariable linear/logistic regressions were performed with the following dependent variables: development of new clinical symptom/condition; latency to development of each condition/symptom; and spirometry, BP and laboratory outcomes. Independent variables included: age; overweight/obese/normal BMI; dust cloud exposure; home dust exposure; and remaining in home ≥1 day between September 11 and 18, 2001. Anthropometric outcomes were examined as dependent variables against all other independent variables. Multivariable regression of the dependent variable was performed against all independent variables, with two exceptions. Given findings from a previous study associating eye irritation (a proxy for severity) with increased respiratory consequences of the WTC disaster (Thomas et al., 2008b), dust cloud was categorized as absent/not covered in dust/covered in dust in the multivariable analysis of spirometry results. BMI category was also excluded from the multivariable analysis of total/percent eosinophils.

2.7.1. Sensitivity analysis—Recognizing the lack of a control group in our study, we reprised all multivariable analyses across least/most exposed subpopulations to assess the robustness of our results. We created a scale representing the sum of categorical dust cloud, heavy dust cloud, home dust, and residence ≥1 day between September 11 and 18, 2001 for spirometry outcomes, and the sum of categorical dust cloud, home dust, and residence ≥1 day between September 11 and 18, 2001 for all other dependent variables. Valuesb2 represented low exposure, while ≥2 represented high exposure.

All analyses were performed with Stata 10 (College Station, TX) or SAS 9.2 (Research Triangle Institute, Cary, NC).

3. Results

Our sample was ethnically diverse (Table 1). Over one-third of questionnaire respondents (38.5%) were caught in the dust cloud. Many (39.7%) resided <1 mile from the WTC site, while most, 77%, attended school <1 mile from the WTC site. Home dust exposure was identified in 25.7%, with the majority reporting heavy exposure. An increase in a wide range of clinical diagnoses and symptoms was noted, with mean latency onset often within the first 3–4 years.

One-third were overweight, and 15.6% obese (Table 2). Prehypertension or hypertension criteria were met in 45.5% (33% of those <20 years of age), with 10.6% meeting hypertension criteria. An HDL<40 mg/dL was identified in 10%, 24% had triglycerides≥ 100 mg/dL, and 19.4% had elevated ALT. Body mass was similar to normative data from NHANES 2003–8. There was a significant divergence from normative data with respect to other cardiometabolic outcomes. Whereas in 2001–2006 NHANES, 6.9% were prehypertensive and 2.4% were hypertensive, 45.5% and 10.6% in our study population were prehypertensive and hypertensive respectively. Low HDL and elevated triglycerides were similar compared to a reference population (Johnson et al., 2009), while NAFLD prevalence was much higher (19.4%) adult NHANES prevalence (8%) (Clark, 2006). The mean eosinophil count was 168.4/μL and <2% had abnormal TSH. Both were slightly lower than the normative sample from NHANES 2009–10.

As of December 19, 2011, spirometry was completed in 127 (85.8%; Table 3). Four-fifths (82.9%) had normal predicted FEV_1 and FVC, yet mean FEV_1/FVC averaged 84.1%, with 11.3% having $FEV_1 \leq$ 5th percentile, and 10.5% with FEV_1/FVC≤5th percentile. Compared with normative data from NHANES 2007–10, the study population appeared to have lower FEV_1 and FVC percent predicted, as well as more frequent abnormal FEV_1, FVC, and MMEF.

Multivariable linear/logistic analyses of clinical and metabolic outcomes (Table 4) included dust cloud, home dust, residence ≥1 day at home between September 11 and 18, 2001, BMI category and age as independent variables. Spirometry outcomes also included categorical heavy dust cloud as a nominal covariate. Older children more frequently reported gastroesophageal reflux symptoms (+16.6% odds/year older, p=0.004) and headaches (+10.2% odds/year older, p= 0.011), while younger children were diagnosed with asthma more frequently (+16.2% odds/year younger, p=0.001). Increased eosinophils (counts and %) were identified among those residing at home ≥1 day in the week following the WTC disaster (p=0.027) and younger children (p=0.023). Prehypertension was positively associated with older age (+9.5% odds/year older, p=0.024).

Home dust exposure was associated with reductions in HDL (−10.3 mg/dL, p=0.027) and elevations in triglycerides (+36.3 mg/dL, p=0.033). Logistic regressions identified OR 7.00 (p=0.059) for elevated triglycerides and OR 2.85 (p=0.162) for low HDL. Exclusion of nonfasting triglycerides>200 mg/dL (suggestive of a genetic dyslipidemia not related to environmental exposure) (Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents: Summary Report, 2011) attenuated association of home dust with trigylcerides to p=0.059 (+22.3 mg/dL). No associations with TSH, anthropometrics or ALT were identified.

Children with dust cloud exposure, but not those with heavy dust cloud exposure, were identified as having reduced FEV_1/FVC (−5.4%, p=0.032) and MMEF (−17.1% predicted, p=0.034). Associations were also identified for dust cloud (but not heavy dust cloud) exposure with subnormal FVC (OR 44.36, p=0.003) and FEV_1/FVC (OR 15.06, p=0.002). Dust cloud exposure was associated with higher odds of abnormal spirometry (OR 24.39, p=0.0002), isolated low FVC (OR 21.16, p=0.011) and obstructive pattern (OR 7.05, p=0.026). No associations were identified either in the group not exposed to dust cloud or in the more heavily exposed dust cloud category (all p>0.05).

Our sensitivity analysis confirmed decrements in MMEF Z-score (−7.8% predicted, p=0.069) and FEV_1/FVC (−1.75%, p=0.110) among the heavily exposed group, as well as below normal FVC (OR 5.30, p=0.107) and FEV_1/FVC (OR 4.49, p=0.057). Higher odds of abnormal spirometry (OR 4.20, p=0.027), low FVC (OR 3.92, p= 0.194) and obstructive pattern (OR 2.92, p=0.188) were also identified. No increase in triglycerides was identified in the highly exposed sub-sample (+2.30 mg/dL, p=0.700) though a near-significant decrement in HDL was identified (−7.82 mg/dL, p=0.107). Increases in total and percent eosinophils persisted among younger children (+0.090% or +6.4/μL per year younger). Older age was still associated with prehypertension (+9.3% odds/year older, p=0.010), new GERD (OR +17.1% odds/year older, pb0.001), and headaches (+9.2% odds/year older, p=0.015), while younger children developed asthma more frequently (OR +14.1% odds/year younger, p=0.001). New-onset rash was identified in the most-exposed group (OR 3.88, p=0.023) with reduced latency (−3.4 years compared with least-exposed, p=0.010), and reduced latency to allergies was identified among the most heavily exposed (−1.9 years, p=0.032).

4. Discussion

These findings suggest a wide range of potential health effects in children with WTC dust exposure. Strengths of this study include: use of clinical testing on pulmonary function, anthropometry, and blood pressure, and metabolic evaluations in addition to exposures and symptoms obtained via clinical history. Most of the clinical tests performed were standardized by age and gender according to national standards.

Absent a reference group or baseline data, the increased risks observed could be due to pre-existing, unknown risk factors in downtown New York City rather than to WTC dust exposure. Additionally, data obtained from a clinic population may not apply to the entire population of exposed children. As the initial clinical visit occurred on average 7.83 years after the WTC tragedy, recall bias of symptoms and exposures is also a concern. Our sensitivity analysis diminishes but does not preclude such concerns. Our clinical measurements are also from a single time point quite distant in time from 2001, and other interceding/confounding events represent plausible alternative explanations for the effects identified.

White coat hypertension is also well documented, and the present study is limited by the existence of a “first reading” effect with oscillometric devices. The present analysis also does not control for psychological stress and related symptoms of posttraumatic stress disorder and depression, an important covariate for the outcomes we studied (Wright, 2005; Wright et al., 2002, 2005). Possible confounding of dust cloud by coexistent stressful exposures known to independently influence pulmonary consequences of air pollution exposure (Islam et al., 2011; Wright, 2005) further limits statements regarding causation. The study area may have had high rates of aerodigestive, cardiometabolic, and high eosinophilia rates even before September 11, 2001. The demographics of our sample are similar to WTCHR children (Thomas et al., 2008b) alleviating this concern somewhat.

Recognizing these limitations, our findings of abnormal FEV_1, obstructed pattern, and low vital capacity, mirror those identified in adults exposed to the dust cloud (Banauch et al., 2006; Friedman et al., 2011; Kleinman et al., 2011). The absence of an association of heavy dust cloud exposure with decrements in spirometry is counterintuitive. Children covered in dust may have been more likely to limit ongoing exposure in the days after September 11, 2001. We did not ask about eye irritation from the dust cloud as a proxy for severity of exposure, as the WTCHR did (Thomas et al., 2008a), and so there is limited comparability with those results. We may also have suffered from low statistical power to detect effects among this relatively small subpopulation of children in our study. Yet an acquired deficit in lung growth such as the one we identified has been associated with an accelerated decline in lung function and the development of chronic obstructive pulmonary disease in adulthood (Martinez, 2009) and it is therefore even more important to interrogate the possibility of physiologic decrements in a larger, more representative sample with a diversity of WTC exposures and compare them with a control group.

The relatively high frequency of cardiometabolic risks is also of great concern, and has biologic plausibility. Increased risk of obesity among children from asthma is well documented, possibly due to decreased reduced functional lung and exercise capacity (Bibi et al., 2004; Figueroa-Muñoz et al., 2001; Gilliland et al., 2003; Shore, 2008; To et al., 2004). Further chronic dietary imbalances may have been produced by exposure to WTC dust or by witnessing the events of September 11, 2011 as well as experiencing resultant mental health consequences, such as depression, anxiety and post-traumatic stress (Goodman and Whitaker, 2002; Richardson et al., 2003). While the cardiovascular consequences in children are not well documented, it is within the context of the emerging obesity epidemic (Freedman et al., 1999; Ogden et al., 2008) in the United States in which the plausibility of accelerated and early cardiovascular disease in children arises, through the coupling of oxidative stress from outdoor air pollutant exposures and increased lipid deposition in early life (Ceriello and Motz, 2004; Griendling and FitzGerald, 2003; Heitzer et al., 2001). Indeed, DNA adducts have been identified as a marker of oxidative stress due to air pollution leading to cardiovascular diseases (Autrup et al., 1999; Risom et al., 2003). These same DNA adducts have been identified as elevated among highly exposed newborns born to mothers with increasing proximity to the WTC site (Perera et al., 2005). Although selection bias limits extrapolation, the high prevalence of new-onset GERD (29.1%), compared with 1.0% annual prevalence identified in one study (Sorof et al., 2001), raises the need to consider aerodigestive effects more carefully in this population.

In light of the potential clinical significance of accelerated and adverse pulmonary and cardiometabolic risks in WTC-exposed children, additional research is needed to ensure that longer-latency health conditions do not go unrecognized in this younger and vulnerable population. A comparable control group is needed to exclude other environmental triggers in NYC, high baseline rates due to poor disease management, and limited health care access, as potential confounders.

Finally, we note that this manuscript does not present analyses of depression, post-traumatic stress disorder (PTSD) and anxiety as potential risk factors. We note that a substantial literature indicates that PTSD is associated with increased allostatic load resulting in significant physical and psychological morbidity (Chemtob et al., 2008; Juster et al., 2010). Effects include increased cardiovascular reactivity, disturbed sleep physiology, obesity, autonomic hyperarousal, and altered hypothalamic–pituitary axis activity. Similarly, PTSD symptoms have been found to be associated with a significantly increased likelihood of asthma among 9/11 exposed adults. For example, a recent study examining twins found that those in the highest quartile of PTSD symptoms were 2.3 times as likely to have asthma compared with those in the lowest quartile, even taking into account familial/ genetic factors and other potential confounds (Goodwin et al., 2007).

Brackbill and his colleagues studied consequences of WTC exposure on new asthma case prevalence and also examined the prevalence of PTS symptoms (Brackbill et al., 2009). Among those with post 9/11 asthma, 36% had probable PTSD 5–6 years after the disaster. Although younger age was associated with increased vulnerability among children in the WTCHR (Thomas et al., 2008a), there appeared to be no relationship between event witnessing or PTSD symptoms and increased asthma. The association of younger age with vulnerability both to exposure-related asthma and PTSD suggests the importance of re-examining these associations among children and adolescents directly exposed to the WTC dust cloud. Clearly, such re-examination should include (1) better measures of child and adolescent exposure to trauma other than the WTC attacks, (2) assess exposure to stressful life events, and (3) measure actual diagnosis (nearly all prior studies used self-report) (Perlman et al., 2011).

The accurate assessment of exposure–outcome relationships is a critical challenge in this, as in all studies of WTC-exposed populations. Because the event was a disaster with few immediate studies and exposure assessments, personal exposure cannot be reconstructed completely. Modeling of the plume has provided some information (Stenchikov et al., 2006). Exposure categories as previously described (Thomas et al., 2008b) and analyses of acute/chronic exposures in area residents and workers (Friedman et al., 2011) may be useful in developing composite scales for children as well.

5. Conclusion

This cross-sectional, clinically-based study represents relatively brief follow-up in the context of chronic conditions with a longer latency, especially cardiometabolic outcomes. Thus, additional research is needed to ensure that longer-latency health conditions do not go unrecognized in this younger and vulnerable population. Although findings from this help-seeking clinic sample may not generalize to children exposed to the WTC disaster, our results confirm and extend previously-documented respiratory implications, and suggest possible aerodigestive and cardiovascular effects that require longitudinal study in a larger, more representative sample compared with a control group.

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Abbreviations

ALT	alanine aminotransferase
AST	aspartate aminotransferase

BMI	body mass index
CDC	Centers for Disease Control and Prevention
FEV_1	forced expiratory volume in the first second
FVC	forced vital capacity
GERD	gastroesophageal reflux disease
HDL	high-density lipoprotein
MMEF	maximum midexpiratory flow
NAFLD	non-alcoholic fatty liver disease
NHANES	National Health and Nutrition Examination Survey
TSH	thyroid stimulating hormone
WTC	World Trade Center
WTCHR	World Trade Center Health Registry
WTCHP	World Trade Center Survivors Health Program

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HIGHLIGHTS

▷ Children seeking care for World Trade Center-related health concerns frequently reported chronic medical conditions.

▷ Exposure to the dust cloud in childhood is associated with decrements in pulmonary function.

▷ Chronic dust exposure is associated with increases in cardiometabolic risks.

▷ Further extensive study of cardiometabolic and respiratory consequences in exposed children is needed.

Table 1

Demographics, exposure characteristics, conditions reported and latency to development of conditions among patients with pediatric exposure to WTC disaster.

Characteristic (n = 148 unless otherwise indicated)	Result
Demographics	
Female sex, %	55.4
Age on 9/11/01, mean years (range)	11.5 (4 days-18 years)
Years to first visit from 9/11/01, mean (range)	7.83 (4.83-10.19)
Race,%	
White	61.4
Asian	24.2
African American	12.9
Native American/Alaskan native	1.5
No answer	10.2
Hispanic ethnicity, %	24.6
Exposure characteristics	
Home distance < 1 mile to WTC site[a] (n = 136), %	39.7
School distance <1 mile to WTC site[a] (n = 90), %	76.6
Caught in dust cloud[a] (n = 146), %	38.5
Light dust in home[a] (n = 140), %	25.7
Heavy dust in home[a] (n = 140), %	21.4
Residence in home at least 1 day in September 11-18, 2001[a] (n = 145), %	80.0

Clinical conditions/symptoms	Preexisting as of September 11, 2001, %	New onset, after September 11, 2001[b], %	Mean latency to onset after September 11, 2001, years (range)
New onset provider-diagnosed asthma	14.9%	21.4%	3.7 (0.3-8.7)
Persistent heartburn/acid indigestion	2.7%	29.1%	3.2 (0-9)
Nasal congestion/sinus symptoms	12.8%	52.7%	3.7 (0-10)
Rash	4.7%	15.6%	3.8 (0-10)
Persistent headaches	15.5%	40.9%	4.4 (0-10)
Allergic symptoms	12.8%	39.5%	3.7 (0-9)

[a]Missing data.

[b]Among those without preexisting conditions/symptoms.

Table 2

Anthropometric, blood pressure and metabolic measurements among patients with pediatric exposure to WTC disaster, compared with normative data.

Clinical characteristic	Result (SD)	Normative data
Anthropometrics (n = 147 unless otherwise indicated)		
Mean BMI	23.38 (5.18)	22.10 (6.13)[a]
Mean BMI Z score[c] (n = 70)	0.51 (1.02)	0.60 (1.14)[a]
Overweight, %	33.3	37.3[a]
Obese,%	15.6	21.0[a]
Blood pressure (BP, n = 132 unless otherwise indicated)		
Mean systolic, mm Hg	117.0 (14.4)	108.0 (10.6)[a]
Mean diastolic, mm Hg	69.5 (11.6)	57.9 (12.5)[a]
Systolic Z score[c] (n = 65)	0.27 (1.12)	–0.13 (0.90)
Diastolic Z score[c] (n = 65)	0.18 (1.01)	–0.56 (1.05)
Hypertension, %	10.6	2.4[b]
Prehypertension or hypertension, %	45.5	6.9[b]
Laboratory values		
Elevated TSH, %(n = 90)	1.6	1.6[d]
Mean TSH (n = 90)	1.91 (1.23)	1.52 (0.88)[d]
Elevated triglycerides, % (n = 50)	24.0	25.6[b]
Mean triglycerides, mg/dL (n = 50)	94 (64)	86 (56)[a]
Low HDL, % (n = 50)	10.0	19.3[b]
Mean HDL, mg/dL (n = 50)	55.7 (13.8)	54.0 (12.6)[a]
Elevated ALT, % (n = 93)	19.4	9.5[a]
Mean ALT, IU/L (n = 93)	22.5 (14.2)	20.1 (15.9)[a]
Mean AST, IU/L (n = 93)	25.2 (12.1)	24.6 (11.3)[a]
Total eosinophils, number/μL (n = 133)	168.4 (148.9)	232.9 (208.2)[d]
Percent eosinophils, % (n = 133)	2.39 (2.08)	3.39 (2.80)[d]

[a]Normative data from NHANES 2003-8 (analyzed by authors).

[b]Normative data from NHANES 2001-6 (Johnson et al. 2009).

[c]Z scores based upon distribution of BP and BMI in NHANES samples age<20.

[d]Normative data from NHANES 2009-10 (analyzed by authors).

Table 3

Spirometry among patients with pediatric exposure to WTC disaster, compared with normative data.

Clinical result (n = 127 unless otherwise indicated)	Result	Normative data (NHANES 2007-10)
FEV_1, % predicted, mean[a] (n = 123, SD)	96.5 (12.9)	101.8 (14.2)
FVC, % predicted, mean[a] (n = 123, SD)	99.0 (13.2)	103.5 (13.7)
FEV_1/FVC, mean[a] (n = 122, SD)	84.1 (6.4)	86.0 (6.6)
MMEF, % predicted (MMEF, SD)	94.6 (2.4)	99.8 (29.2)
Values below lower limit of normal		
FEV_1, %	11.3	7.2
FVC, %	8.1	7.1
FEV_1/FVC ratio, %	10.5	12.9
MMEF, %	12.3	10.2
Spirometry pattern		
Normal spirometry, %	82.9	86.0
Isolated low FVC, %	6.5	2.7
Obstructed pattern, %	8.9	11.0
Obstructed pattern with low FVC, %	1.6	0.4

[a]Absence of anthropometric data and/or failure to meet rigorous validity standards for components of spirometry measurement contribute to missing data.

Table 4

Predictors of anthropometric, blood pressure, metabolic analyses, clinical conditions and spirometry results among patients with pediatric exposure to WTC disaster.

Significant multivariable regression result	Odds ratio/increment (p value)
Clinical conditions	
Age on 9/11/01 associated with new provider diagnosed asthma	+ 16.2% odds/year younger (0.001)
Age on 9/11/01 associated with new GERD	+ 16.6% odds/year older (0.004)
Age on 9/11/01 associated with new headaches	+ 10.2% odds/year older (0.011)
Laboratory analyses	
Residence in home at least 1 day in the period September 11-18, 2001 associated with total eosinophil count	+ 64.6/μL (0.027)
Residence in home at least 1 day in the period September 11-18, 2001 associated with percent eosinophil count	+ 1.37% (0.007)
Age on 9/11/01 associated with percent eosinophil count	+ 0.098%/year younger (0.023)
Home dust exposure associated with HDL	–10.3 mg/dL (0.027)
Home dust exposure associated with triglycerides	+ 36.3 mg/dL (0.033)
Blood pressure	
Age on 9/11/01 associated with prehypertension	+ 9.5% odds/year older (0.024)
Spirometry results	
Dust cloud associated with FEV_1/FVC	–5.4% (0.032)
Dust cloud associated with <5th percentile FEV_1/FVC	15.06 (0.002)
Dust cloud associated with MMEF percent predicted	–17.1% (0.034)
Dust cloud associated with <5th percentile FVC	44.36 (0.003)
Spirometry pattern	
Dust cloud associated with abnormal spirometry	24.39 (0.0002)
Dust cloud associated with isolated low FVC pattern	21.16 (0.011)
Dust cloud associated with obstructive pattern	7.05 (0.026)

All results are from multivariable linear/logistic regressions including: residence in home at least 1 day in the period September 11 -18, 2001, home dust exposure, dust cloud exposure (as present/absent for all results, except for PFT results when expressed as absent/dust exposure but not covered in dust/covered in dust), age on 9/11/01 and overweight/obese/normal body mass index (with the exception of eosinophils, for which body mass index was excluded). Results not reported imply p>.05.