Health and Clean Air Newsletter

Fall--2002

1. Schelegle ES, Siefkin AD, McDonald RJ. Time course of ozone-induced neutrophilia in normal humans. Am Rev Respir Dis 1991; 143:1353–58.

2. Graham DE, Henderson F, House DE. Neutrophil influx measured in nasal lavages of humans exposed to ozone. Arch Environ Health 1988; 43:229–233.

3. Graham DE, Koren HS. Biomakers of inflammation in ozone-exposed humans. Am Rev Respir Dis 1990; 142:152–156.

4. Bascom R, Naclerio RM, Fitzgerald TK, Kagey-Sobotka A, Proud D. Effect of ozone inhalation on the response to nasal challenge with antigen of allergic subjects. Am Rev Respir Dis 1990; 142:594–601.

5. Bascom R, Naclerio RM, Fitzgerald TK, Kagey-Sobotka A, Proud D. Effect of ozone inhalation on the response to nasal challenge with antigen of allergic subjects. Am Rev Respir Dis 1990; 142:594–601.

6. Smith CE, Koren HS, Graham DG, Johnson DA. Mast cell tryptase is increased in nasal and bronchial alveolar lavage fluids of humans after ozone exposure. Inhalation Toxicol 1993; 5:117–127.

7. Lippman M. Health effects of ozone: a critical review. J Air Pollut Control Assoc 1989; 39:672–95.

8. Van Louveren HS:, Wagenaar S, Walvoort HC, Vos JG. Effect of ozone on the defense to a respiratory Listeria monocytogenes infection in the rat. Suppression of macrophage function on cellular immunity and aggravation of histopathology in lung and liver during infection. Toxicol Appl Pharmacol 1988; 94:374–93.

9. Gardner DE. Use of experimental airborne infections for monitoring altered host defenses. Environ Health Perspect 1982; 43:99–107.

10. Gilmour MI, Park P, Selgrade MK. Ozone-enhanced pulmonary infection *with Streptococcus Zooepidemicus* in mice: The role of alveolar macrophage function and capsular virulence factors. Am Rev Respir Dis 1993; 147:753–60.

11. Gilmour MI, Selgrade MK. A comparison of the pulmonary defenses against Streptococcal infection in rats and mice following O3 exposure: A possible mechanisms of disease resistance in rats. Toxicol Appl Pharmacol 1993.

12. Harkema JR, Plopper CG, Hyde DM, George JAS, Wilson DW, Dungworth DL. Response of the macaque nasal epithelium to ambient levels of ozone: a morphologic and morphometric study of the transitional and respiratory epithelium. Am J Pathol 1987; 128:129–144.

13. Devlin RB, McDonnell WF, Mann R, Becker S, House DE, Schreinemachers D,Koren HS. Exposure of humans to ambient levels of ozone for 6.6 hours causes cellular and biochemical changes in the lung. Am J Respir Cell Molec Biol 1991; 4:72–81.

14. Harder Sd, Harris DT, House D, Koren HS. Inhibition of human natural killer cell activity following in vitro exposure to ozone. Inhal Toxicol 1990; 2:161–73.

15. Van Louveren HS; Wagenaar S, Walvoort HC, Vos JG. Effect of ozone on the defense to a respiratory Listeria monocytogenes infection in the rat. Suppression of macrophage function on cellular immunity and aggravation of histopathology in lung and liver during infection. Toxicol Appl Pharmacol 1988; 94:374–393.

16. Gardner DE. Use of experimental airborne infections for monitoring altered host defenses. Environ Health Perspect 1982; 43:99–107.

17. "Executive Summary," *Air Quality Criteria for Ozone and Related Photochemical Oxidants*, United States Environmental Protection Agency, (Washington, DC: Office of Research and Development), February 1994.

18. McDonnell WF, Horstman DH, Hazucha MJ, Seal Jr E, Haak ED, Salaam SA, House DE. Pulmonary effects of ozone exposure during exercise: dose response characteristics. J Appl Physiol 1983; 5:1345–52.

19. "Executive Summary," *Air Quality Criteria for Ozone and Related Photochemical Oxidants*, United States Environmental Protection Agency, (Washington, DC: Office of Research and Development), February 1994.

20. Kinney and Ozskaynak. Associations of Daily Mortality and Air Pollution in Los Angeles County. Environ. Res., 54, 1991.

21. Kinney and Ozkaynak. Associations Between Ozone and Daily Mortality in Los Angeles and New York City. Am Rev. Respir. Dis., 145(4:2):A95, 1992.

22. Gilliland, F.D., Berhane, K., Rappaport, E., Thomas, D.C., Avol, E., Gauderman, W. J., London, S.J., Margolis, H.G., Mcconnell, R., Islam, K.T., & Peters, J.M. The Effects of Ambient Air Pollution on School Absenteeism due to respiratory illness Epidemiology 2001: 12; 43-54.

23. Kinney, et al. A Critical Evaluation of Acute Ozone Epidemiology Results. Arch. Env. Health 43, 168–73, 1988.

24. American Lung Association, *State of the Air: 2001*, http://www.lungusa.org/press/envir/sota2001_release.html

25. Isaak Walton League, "Air Pollution in Our Parks: Shenandoah National Park Fact Sheet," http://www.iwla.org/reports/parkfssh.html. Ozone is also toxic to trees and other vegetation. A decline of 26 percent to 51 percent in the growth rate of eastern white pines in the Blue Ridge Mountains from the late 1950's to mid-1970's has been attributed to ozone pollution. In

Shenandoah National Park, tulip poplar, green ash, sweet gum, black locust, Eastern hemlock, Table Mountain pine, pitch pine and Virginia pine seedlings have all demonstrated growth loss at ozone levels below minimum federal health standards. National Park Service, "Air Quality in the National Parks," http://www2.nature.nps.gov/ard/pubs/aqnps.htm.

26. National Park Service, "Shenandoah National Park Natural Resource Guide," http://www.shenandoah.national-park.com/nat.htm#air.

27. National Park Service, *Air Quality in the National Parks (2d Edition),* Washington, D.C. (Sep. 2002), http://www2.nature.nps.gov/ard/pubs/aqnps.htm.

28. National Park Service, "List of High Ozone in Park Units - 2001 Season," http://www.aqd.nps.gov/ard/gas/exceed2001.htm.

29. National Park Service, "Ozone—the Invisible Poison," http://www.nps.gov/seki/ozone.htm.

30. See e.g. Brasseur GP, et. al. "Tropospheric Ozone and Climate: Past, Present and Future," in *Present and Future of Modeling Global Change: Toward Integrated Modeling*, Eds. T. Matsuno and H. Kida, TERRAPUB, 2001, http://www.terrapub.co.jp/e-library/toyota/pdf/063.pdf.

31. Brauer, M., Blair, J., & Vedal ,S. Effect of ambient ozone exposure on lung function in farm workers Am J Respir Crit Care Med 154, 981–987, 1996 Fraser Valley study. 58 workers. Farms were at Abbottsford and Matsqui, about 70 Km southeast of Vancouver. Workers spent all day in outside work. Ambient monitors for O3 and SO4. PM2.5 mean was 11.4 micrograms/m3. Ozone daily maximum was mean of 40.3 ppb, with single highest value of 84 ppb. Ozone for the workshift time had a mean of 26 ppb, and a maximum of 54 ppb. FEV1 fell -3.3 ml and FVc fell -4.7 ml for each ppb increase in ozone. Deficits still apparent on following morning.

32. Climate Monitoring and Diagnostics Laboratory, National Oceanic and Atmospheric Administration, "Observed Ozone Changes," http://www.cmdl.noaa.gov/ozwv/dobson/papers/wmobro/observed.html. A.S.L. Associates has studies historic ozone levels for the U.S. National Acid Precipitation Program and the U.S. Environmental Protection Agency. According to A.S.L., "In the mid-1800s, surface ozone was the focus of many scientific studies to prove its existence, to discover its functions in the atmosphere, and to define its role in affecting the spread of epidemics. Ozone was commonly measured using the Schoenbein ozonoscope method. Schoenbein papers were coated with iodide; the reaction with ozone formed iodine. Ozone concentration was expressed as Schoenbein numbers based on coloration of Schoenbein's test paper. Starting in the mid-1800s, more than 300 stations recorded ozone exposures in countries such as Austria, Australia, Belgium, England, France, Germany, Russia, and the United States. Based on data evaluated, some scientists have concluded that (1) the average daily maximum of the surface ozone partial pressure in the Great Lakes area of North America was approximately 0.019 ppm, and (2) the European measurements between the 1850s and 1900 were mostly in the range of approximately 0.017 ppm to 0.023." ppm.http://www.asl-associates.com/back.htm

33. Schelegle, E.S., Eldridge, M.W., Cross, C.E., Walby, W.F., & Adams, W.C. Differential

effects of airway anesthesia on ozone-induced pulmonary responses in human subjects Am J Respir Crit Care Med 163; 1121-1127; 2001. 0.30 ppm O3 inhaled for 65 minutes by 22 ozone-sensitive healthy subjects. After 50 minutes, FEV1 was reduced 24%, breathing frequency was increased 40%, VT was decreased 31%, and the subjective symptom score increased. Inhalation of tetracaine aerosol (MMD = 3.52 microns) caused marked reductions in throat irritation, cough, shortness of breath, and pain on deep inspiration. But minor and inconsistent rectification of the FEV1 occurred, and respiratory rate not significantly different from effect of saline aerosol. Authors note: "Our data are consistent with afferent endings located within the large conducting airways of the tracheobronchial tree being primarily responsible for ozone-induced subjective symptoms and provides strong evidence that ozone-induced inhibition of maximal inspiratory effort is not dependent on conscious sensations of inspiratory discomfort". Reviews evidence that C-fiber afferent endings initiate response. Note that partial reversal of effect by lidocaine reported by Hazucha, Bates & Bromberg(J Appl Physiol 1989;67; 1535-1541) probably attributable to the time that separated the pulmonary function tests before and after lidocaine.

34. Aris, R.M., Christian, D., Hearne, P.Q., Kerr, K., Finkbeiner, W.E., & Balmes, J.R. Ozone-induced airway inflammation in human subjects as determined by airway lavage and biopsy Am Rev Respir Dis 148, 1363-1372, 1993. Notes that BAL primarily samples effects in peripheral airways, but central airway effects might also be occurring after ozone exposure. Isolated lavage of the left mainstem bronchus together with forceps biopsy of the mucosa performed 18 hours after exposure to 0.20 ppm O3 for four hours during moderate exercise. In 14 healthy athletic subjects. Identical protocol followed in 12 subjects after exposure to filtered air. 8 completed both exposures; 6 only the air exposure and 4 only the ozone exposure.

	AIR Exposed	OZONE exposed
Number of subjects	12	14
Ozone exposure	–	0.20 ppm 4 hours
	Moderate exercise	
Lavage cell count (18 hr)	4.9	13.9
Cells/ml x 10fourth		
Lactate Dehydrogenase U/L	9.6	18.9
Morphometry (PMN/cm2 tissue)	330	2070
FVC pre/post L	5.01/5.0	4.77/4.42
FEV1 pre/post L	4.17/4.19	3.99/3.62
SRaw LxcmH20/L/sec	2.74/2.93	2.54/3.26
Symptom Scores pre/post	0.9/4.2	0.5/13.5
Albumin BAL mg/ml	0.05	0.10

Conclude that "Inhalation of an ambient concentration of ozone can cause morphologic evidence of airway injury in healthy human subjects. Ozone-induced airway inflammation may be an important contributing factor to acute exacerbations of asthma and chronic bronchitis."

35. Fanucchi, M.V., Wong, V., Hinds, D., Tarkington, B., Van Winkle, L.S., Evans, M.J., & Plopper, C.G. Repeated episodes of exposure to ozone alters postnatal development of distal conducting airways in infant rhesus monkeys. Am J Respir Crit Care Med 161: A615, 2000. 30 day old rhesus monkeys exposed to filtered air or ozone for 11 episodes, 5 days each, 0.5 ppm, 8

hr/day, followed by 9 days filtered air; sacrificed during the last fresh air exposure. Details of preparation of lung given. Findings were:
1. ozone decreased the number of branchings to the most proximal respiratory bronchiole (from 13 to about 9):
2. increased density and distribution of goblet cells:
Authors conclude: "We conclude that the periodic cycles of ozone exposure alters postnatal lung morphogenesis and epithelial differentiation in the distal lung of infant primates."

36. Kunzli, N., Lurmann, F., Segal, M., Ngo, L., Balmes, J., Tager, I.B. Association between lifetime ambientozone exposure and pulmonary function in college freshmen--Results of a pilot study. Environ. Res. 1997; 72:8–16.

37. Galizia, A., Kinney, P.L. Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults. Environ. Health Perspect 1999; 107:675–9.

38. McCconnell, R., Berhane, K., Gilliland, F., London, S.J., Islam, T., Gauderman, W.J., Avol, E., Margolis, H.G., & Peters, J.M. Asthma in exercising children exposed to ozone: a cohort study Lancet 2002; 359; 386–391. From the Southern California Children's Study. Relevant numbers; 5762 children completed baseline questionnaires; 479 excluded because they were not at school when the questionnaire was administered; 883 excluded for a history of asthma; 312 excluded because of missing answers to "wheezing" questions; 26 excluded for chest illnesses such as cystic fibrosis: 527 excluded because they had less than one year of follow-up. This left 3535 children with no initial history of asthma; 2752 of these had no history of wheezing; 1934 played sports; 273 played three or more team sports. There were 46 low pollution communities (O3 daytime mean 40.0 ppb); and 46 high pollution communities (O3 mean 59.6 ppb). PM10 twice as high in high ozone communities (43.3 vs 21.6) and PM2.5 three times higher (21.4 vs 7.6). NO2 three times higher in high ozone communities (29.2 vs 10.8 ppb). It was shown that development of asthma prospectively was three times higher in children participating in more than 3 sports in high ozone communities, compared to children who did no sports in both communities or did fewer than 3 sports in high ozone communities. No differences in development of asthma if other pollutants were studied. Excellent discussion; 32 references. Convincing argument as to why standard cross-sectional comparisons might show no differences in prevalence of asthma.

39. Friedman, M.S., Powell, K.E., Hutwagner, L., Graham, L.M., & Teague, W.G. Impact of changes in transportation and Commuting behaviors during the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma. JAMA 2001: 285; 897–905. Comparison of the 17 days of the Olympic Games (July 19–Aug 4) to a baseline period consisting of the 4 weeks before and 4 weeks after the Olympic Games. Peak one hour level of O3 fell to 50–100 ppb during the games from a predicted value of about 70–120 in the comparison periods. PM10 (24 hour level) was 20–45, compared to levels of 30–70; NO2 was only slightly lower running at about 30 ppb peak one hour level compared to values between 20–65. CO also slightly lower. Traffic density measurements showed decreases of 22% in weekday 1-hour morning peak traffic counts during the Olympic Games. Ozone levels fell slightly over the same period in three different places 60 km to 100 km from Atlanta; but these changes were only about one fifth of

the drop in Atlanta. Citywide acute care visits and hospitalizations for asthma were logged. Results showed no changes in nonasthma diagnoses; decreases of 41% in Medicaid claims file, 44% decreases in HMO database; 11% decreases in two emergency pediatric departments; and decreases of 19% in Georgia Hospital Discharge Database. Lack of change in other diagnostic categories indicates that children did not leave Atlanta over the period of the Olympic Games.

40. One of the study's authors, Michael S. Friedman viewed it important because "it provides evidence that decreasing automobile use can reduce the burden of asthma in our cities and that citywide efforts to reduce rush-hour automobile traffic through the use of public transportation and altered work schedules is possible in America." Centers for Disease Control, "CDC study links improved air quality with decreased emergency visits for asthma," Press Release, Feb. 21, 2001, http://www.cdc.gov/od/oc/media/pressrel/r010221.htm.

41. Burnett, R.T., Smith-Dorion, M., Stieb, D., Raizenne, M.E., Brook, J.R., Dales, R.E., Leech, J.A., Cakmak, S., & Krewski, D. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. Am J Epidemiol 2001: 153; 444–452. 15-year period 1980–1994 in Toronto (including 6 cities of Toronto, North York, East York, Etobicoke, Scarborough & York). Daily number of emergency or urgent hospital admissions for croup, pneumonia, asthma and acute bronchitis/bronchiolitis. Gastroentieritis used as control (second leading cause of hospitalization). Prediction equations, previously validated, used to predict PM2.5 from TSP, and sulfates also used in the summer. LOESS nonparametric smoothing method. Adjustemnt for weather descrivbed, and temporal trednds and day of the week effects taken oput. Mean values for ozone 45.2 ppb; PM2.5 18 micrograms/m3; PM10-2.5 16.2; NO2 ppb 44.1; SO2 ppb 11.8; CO ppm 1.9. Mean admissions/day for respiratory problems 2.9; mean GI admissions 1.2. Percentage increase in daily admissions in May–August period was 14.2% associated with a 45.2 ppb increase in O3. (Daily one hour maximum.) if lag of one day used. If lag of 2 days, was 13.2% increase. Based on five day average, 45.2 ppb increase in ozone associated with 34.8% increase. This became 29.4% after adjustment for either PM10-2.5 or NO2. Correlation of O3 withPM2.5 was 0.58 and with NO2 was 0.52; with CO was 0.24. Increases for asthma were 31.3%; croup 45.3%; acute bronchitis/bronchiolitis 45.7%; and 23.3% for pneumonia. Note of CO relationship since this showed strongest relationship after adjustment for ozone.

42. Petroeschevsky, A., Simpson, R.W., Thalib, L., & Rutherford, S. Associations between outdoor air pollution and hospital admissions in Brisbane, Australia. Arch Environ Health 56; 37–52; 2001. Period of study was 1987–1994. Total of 41,127 emergency admissions for cardiovascular disease, and 33,710 respiratory admissions which included 13,246 for asthma (constituting 39% of all respiratory admissions). All public hospitals in Brisbane area included. Air pollution data from 7 stations, measuring ambient levels every half-hour. 24 hour averages and maximal 1 hour concentrations used in analysis, but an 8 hour average used for ozone. Statistical analytical methods exactly followed the APHEA protocol, which is described in some detail. This is an iterative model-building process after correction for more than 60 day temporal cycles. Abstract notes: "Ozone was consistently associated with admissions for asthma and respiratory disease—with little evidence of a threshold. In two pollutant models, the ozone effect was relatively unaffected by the control for high levels of other pollutants. Particulate pollution (measured by nephelometry) was associated positively with admissions for respiratory disease

and admissions for asthma in summer, whereas a negative association was observed for cardiovascular admissions". SO2 was associated with admissions for the control diagnosis of digestive disorders. Note that ozone levels were similarly elevated throughout the year, with mean 8 hour levels being 1.99; 1.67; 1.61; and 2.23 pphm in summer; autumn; winter and spring respectively. Mean 24 hour NO2 levels were 0.97; 1.29; 1.79; and 1.53 pphm in the same seasons. NO2 was not associated with hospital admissions. Risk ratios for asthma admissions in age groups 0–14; 15–64; and for total asthma were 1.064; 1.084; and 1.090 for a unit increase (pphm) in 8 hour ozone. This represents a 9% increase in asthma admissions per 1 pphm increase in ozone. Figure of RR for asthma all ages versus average 8 hour, O3 lagged 2 days is linear from 1.0 to 3.0 pphm with no evidence of a lower threshold.

43. Bag, R., Frolov, A.Q., Keys, J., Zimmerman, J.L., & Hanania, N.A. Association between ambient ozone levels and emergency department (ED) visits for asthma in Houston, TX, USA. Am J Respir Crit Care Med 161: A308; 2000. Adult visits for asthma (> 17 years) recorded. 7982 asthma visits with 4214 during the ozone high months. Mean age 41.6 years; 61% were women. In ozone months, mean values were 36.8 ppb as 8 hour average and 50.8 for 1 hour maximum levels. 71 days with 8 hour average above 0.08 ppm. Significant association between ozone levels and ED visits in the ozone months.

44. Sunyer, J., Basagana, X., Belmonte, J., & Anto, J.M. Effect of nitrogen dioxide and ozone on the risk of dying in patients with severe asthmaThorax 2002: 57; 687–693. Patients over the age of 14 who died during the period 1985–1995 in Barcelona who had visited the emergency department of one of the four largest hospitals in the city. For asthma during 1985–1989. Total of 467 men and 611 women. "Air pollution was measured at the city monitoring stations which provide a mean for the entire city". Daily values of PM10 - appears to have a mean value of 61.2 micrograms/m3; Black smoke 40.0; 1 hour NO2, 89.7 micrograms/m3; 24 hour NO2, 52.3 micrograms/m3; 1 hour ozone 69.3 micrograms/m3; 8 hour ozone 54.4 micrograms/m3; SO2, 18.8 micrograms/m3; Pollen and spores recorded weekly and measured by 'the Cour method'. Thought to be accurate for pollen grains, but fungal spores get damaged and are underestimates. For cases with more than one admission always for asthma, the odds ratio for interquartile change was as follows:

> NO2, 1.688 (1.074 to 2.652): Ozone 1.755 (0.984 to 3.133) with 95% confidence limits in brackets. These values were not much changed by correction for total pollen and for spore counts. "Patients with severe asthma –that is, those with more than one admission to the emergency department for an asthma exacerbation–had a higher risk of dying on days with higher levels of NO2 regardless of the season, and O3 in the warm season. These associations were not confounded by the weekly levels of pollen and fungal spores"

45. Desqueyroux, H., Pujet, J.C., Prosper, M., Squinazi, F., & Momas, I. Short-Term effects of low-level air pollution on respiratory health of adults suffering from moderate to severe asthma. Environmental Research Section A; 89;29–37 (2002). 60 severe asthmatics mean age 55 studied over 13-month period. Criteria included attendance at Center for Treatment of Respiratory Diseases in Paris, and included more than 15% increase in FEV1 after beta2 agonist inhalation; more than 20% FEV1 fall after provocative dose of methacholine; recurrent wheezing and physician certified moderate to severe asthma. Daily values of SO2 from 28 sites; for PM10 from

7 sites; for NO2 from 15 sites; and from 6 sites for O3. Each subject seen by a physician at each consultation whether scheduled or emergency. An Asthma attack was defined as the need to increase twofold the dose of inhaled beta2 agonist and confirmed by clinical examination. Odds ratio for risk of an asthmatic attack per 10 microgram/m3 increase in PM10 was 1.41. An increase of 10 microgram/m3 (5 ppb) of Ozone was associated with an OR of 1.20. 3–5 day delay for PM10 but 2-day delay for O3. No association with NO2 nor with SO2. Convincing and clinically thorough panel study.

46. Thurston, G., Lippmann, M., Bartoszek, M., & Fine, J. Summer haze associations with asthma exacerbations, peak flow changes, and respiratory symptoms in children at a summer asthma camp. In: Program Abstracts of the Sixth Conference of the International Society for Environmental Epidemiology: Research Triangle Park, NC September 18–21, 1994. Abstract 227: Daily air pollution and health data collected for a week in June 1991, 1992, and 1993 at a camp for asthmatic children. Highest O3 was 160 ppb in 1991, and in 1992 was 63 ppb. Children aged 7–13 had to go to on-site physician for medication. Medication requests monotonically related to ozone levels. Decrements in PEFR also noted. Conclude that there was "a strong association between summertime haze air pollution and asthma exacerbations." Upper respiratory symptoms of sore throat, runny nose, and eye irritation were related to pollen counts in 1992 when pollution levels were low.

47. Honicky, R.E., & Osborne, J.S. Respiratory effects of wood heat: clinical observations and epidemiologic assessment. Environ Health Perspectives 95; 105–109; 1991. These authors in 1983 published a case report (Pediatrics 71; 126–128; 1983) of recurrent chest illnesses with woodburning stove smoke exposure. From Michigan State University. Documents case history of non-atopic child who got recurrent episodes of pneumonia and it was noted that all respiratory symptoms rapidly disappeared on admission to hospital. This study involved 62 randomly selected children aged 1–7 years from those attending a Clinical Center. It was shown that severe respiratory symptoms were commoner in children from homes using wood for heating, when compared to children from homes using fuel oil or gas furnaces.

48. Grosse, S.D., Matte, T.D., Schwartz, J., & Jackson, R.J. Economic gains resulting from the reduction in Children's exposure to Lead in the United States. Environ Health Perspectives 110;563–569; 2002. Calculate improvements in worker productivity as a consequence of lowering the blood lead in children by 15 mug/dl which occurred between 1976 and 1999. Assumed change in cognitive function to be between 0.185 and 0.323 IQ points for each 1 mug/dl change in blood lead. Estimated economic benefit for each year's cohort of 3.8 million 2 year old children ranges from $110 billion to $319 billion.