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Warm Season Temperatures and Emergency Department Visits in Atlanta, Georgia

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Abstract

Purpose—Extreme heat events will likely increase in frequency with climate change. Heat-related health effects are better documented among the elderly than among younger age groups. We assessed associations between warm-season ambient temperature and emergency department (ED) visits across ages in Atlanta during 1993-2012.

Methods—We examined daily counts of ED visits with primary diagnoses of heat illness, fluid/electrolyte imbalances, renal disease, cardiorespiratory diseases, and intestinal infections by age group (0-4, 5-18, 19-64, 65+ years) in relation to daily maximum temperature (TMX) using Poisson time series models that included cubic terms for TMX at single-day lags of 0-6 days, controlling for maximum dew-point temperature, time trends, week day, holidays, and hospital participation periods. We estimated rate ratios (RRs) and 95% confidence intervals (CI) for TMX changes from 27 °C to 32 °C (25th to 75th percentile) and conducted extensive sensitivity analyses.

Results—We observed associations between TMX and ED visits for all internal causes, heat illness, fluid/electrolyte imbalances, renal diseases, asthma/wheeze, diabetes, and intestinal infections. Age groups with the strongest observed associations were 65+ years for all internal

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causes [lag 0 RR (CI)=1.022 (1.016-1.028)] and diabetes [lag 0 RR=1.050 (1.008-1.095)]; 19-64 years for fluid/electrolyte imbalances [lag 0 RR=1.170 (1.136-1.205)] and renal disease [lag 1 RR=1.082 (1.065-1.099)]; and 5-18 years for asthma/wheeze [lag 2 RR=1.059 (1.030-1.088)] and intestinal infections [lag 1 RR=1.120 (1.041-1.205)].

Conclusions—Varying strengths of associations between TMX and ED visits by age suggest that optimal interventions and health-impact projections would account for varying heat health impacts across ages.

Keywords
Temperature; time series; emergency department visits; climate change; heat

1. Introduction
Climate change is expected to lead to higher warm-season temperatures and an increased frequency, intensity and duration of heat waves (Meehl and Tebaldi, 2004; Watts et al., 2015). A growing body of evidence demonstrates that extreme heat can have adverse health effects (Kravchenko et al., 2013; O’Neill and Ebi, 2009). An understanding of the health effects of extreme heat is important for developing the infrastructure, policy, and public health messages to help vulnerable populations avoid unhealthy exposures and to prepare for and adapt to future climate change (Portier et al. 2010).

Associations have been well established between high ambient temperatures and increased all-cause mortality as well as mortality from specific causes such as cardiovascular disease, cerebrovascular disease, respiratory disease, and heat-related causes (e.g. heat stroke) (Basu, 2009; Kilbourne, 1999; Kravchenko et al., 2013; O’Neill and Ebi, 2009; Portier et al., 2010). Some studies have also found associations between high ambient temperatures and morbidity from these types of health conditions using outcome measures such as counts of hospitalizations or emergency department (ED) visits (Green et al., 2010; Knowlton et al., 2009; Lin et al., 2009; Michelozzi et al., 2009; Pudpong and Hajat, 2011; Schwartz et al., 2004; Semenza et al., 1999; Turner et al., 2012; Ye et al., 2012). Assessment of associations between high temperatures and morbidity using ED visits may be particularly useful for some clinical conditions and for younger populations that tend to have more ED visits than hospital admissions or deaths. A study by Knowlton et al. (2009) of the health effects of the 2006 heat wave in California found that the increase in ED visits during the heat wave was more pronounced than the increase in hospital admissions. Nevertheless, relatively few US studies have assessed the health effects of heat waves or high temperatures using ED visits (Basu et al. 2012; Buckley and Richardson 2012; Jones et al. 1982; Knowlton et al. 2009; Lippmann et al. 2013; Rydman et al. 1999; Saha et al. 2015).

The elderly have been found to be particularly susceptible to health effects of extreme heat (Astrom et al., 2011; Basu, 2009; Green et al., 2010; Knowlton et al., 2009; Ye et al., 2012). However, there is comparatively less information regarding heat health effects in children, partially due to the fact that population-based data are sometimes restricted to elderly populations by nature (e.g., mortality, cardiovascular outcomes) or by source (e.g., Medicare data on hospitalizations). Children may also have higher susceptibility to adverse health
impacts from heat, particularly in relation to morbidity rather than mortality (Bartlett, 2008; Ebi and Paulson, 2007; Kravchenko et al., 2013; Sheffield and Landrigan, 2011; Xu et al., 2012; Xu et al., 2014). Several studies conducted outside the US have found associations between high temperatures or heat waves and various measures of morbidity among children (Checkley et al., 2000; Chou et al., 2010; Hashizume et al., 2007; Kovats et al., 2004; Lam, 2007; Leonardi et al., 2006; Nitschke et al., 2011; Onozuka and Hashizume, 2011; Pudpong and Hajat, 2011; Xu et al., 2012; Xu et al., 2013; Xu et al., 2014). In the US, assessment of age-stratified associations between ambient temperature and morbidity with evaluation of effects across the full age range, including children, has been conducted with limited geographic coverage (Basu et al., 2012; Fletcher et al., 2012; Green et al., 2010; Lin et al., 2009; Li et al., 2012; Lippmann et al., 2013). There is a need for expanding the literature on age-specific risk to additional US locations for proper population-wide risk assessment. Assessment of age-specific risks across the full age range has significant implications for targeted interventions (e.g. school athletic programming) and for enhanced accuracy of health impact projections that account for demographic shifts over time.

Effect estimates for the association between extreme heat and health outcomes vary by location, possibly due to population adaptation to local temperatures (Anderson and Bell, 2009; O’Neill and Ebi, 2009; Saha et al., 2015). Multicity studies can give an overall estimate of heat-related health effects across locations, but location-specific information about heat health effects is also needed for local public health planning and emergency preparedness related to climate change. Extreme heat events in sprawling metro areas, including Atlanta, have increased at a faster rate than in compact metro areas (Stone et al., 2010). In Atlanta, the frequency and duration of heat waves have already significantly increased over the period from 1961-2010, like in other US cities, but the rate of increase in Atlanta has been higher than the national average (Habeeb et al., 2015). Assessment of the Atlanta population is thus timely and relevant.

We build on prior studies to conduct a comprehensive assessment of heat-related morbidity among all age groups in Atlanta using ED visits as the morbidity measure. Data come from the Study of Particles and Health in Atlanta, a major research effort examining air quality and acute morbidity (Darrow et al., 2012; Metzger et al., 2004; Peel et al., 2005; Sarnat et al., 2010; Strickland et al., 2010; Tolbert et al., 2000). The 20-year study period makes this one of the largest studies of heat and morbidity to date, offering high power for assessing age-specific associations for a range of outcomes. Objectives of our analysis were to examine effects of heat on ED visits for specific health conditions, with consideration of effects of heat by age group across all ages, using analyses that examine possible non-linear effects, effects at various lags, and the impact on the observed associations of decisions regarding outcome definitions, heat metrics and modeling decisions.

2. Materials and methods

2.1. Data Sources

Hourly meteorological data for Atlanta were obtained from the National Solar Radiation Database for the automated surface observing station (ASOS) located at Atlanta Hartsfield International Airport for the period January 1, 1993 through December 31, 2012 (Wilcox, 2015).
These data were used to calculate daily metrics (i.e., minimum, maximum, and average) of temperature, dew-point temperature, apparent temperature, and wind speed, as well as daily average barometric pressure and total precipitation. Daily data on major ambient air pollutant concentrations (including 1-hour maximum carbon monoxide, nitrogen dioxide, and sulfur dioxide; 8-hour maximum ozone, and 24-hour average particulate matter less than 10 microns in diameter) were obtained from ambient monitoring networks in Atlanta. Daily pollutant measurements across monitors were combined using population weighting (Ivy et al., 2008); these air pollutant data were only available through 2010 at the time of this analysis.

Data on ED visits to hospitals in the Atlanta metropolitan area were obtained from individual hospitals (for the period 1993-2004) and from the Georgia Hospital Association (for the period 2005-2012). Data were restricted to patients whose ZIP code was at least partially within one of the 20 counties in the Atlanta metropolitan area. We assessed daily counts of ED visits for 17 outcomes of interest. In our primary analyses, these outcomes were defined based on the primary international classification of disease, version 9 (ICD-9) codes only, and secondary analyses considered these outcomes as defined by the presence of the selected codes in any of the diagnoses listed on patients’ ED records. The outcomes of interest included ED visits for all internal causes (INTERN; ICD-9 codes 001-799), heat illness (HEAT; ICD-9 code 992), fluid and electrolyte imbalances (FLEL; ICD-9 code 276), all renal disease (RENAL; ICD-9 codes 580-593), nephritis and nephrotic syndrome (NEPH; ICD-9 codes 580-589), all circulatory system disease (CIRC; ICD-9 codes 390-459), hypertension (HT; ICD-9 codes 401-405), ischemic heart disease (IHD; ICD-9 codes 410-414), dysrhythmia (DYS; ICD-9 code 427), congestive heart failure (CHF; ICD-9 code 428), ischemic stroke (STK; ICD-9 codes 433-437), all respiratory system disease (RESP; ICD-9 codes 460-519), pneumonia (PNEU; ICD-9 codes 480-486), chronic obstructive pulmonary disease (COPD; ICD-9 codes 491-492, and 496), asthma/wheeze (ASW; ICD-9 codes 493 or 7896.09/.07), diabetes mellitus (DIA; ICD-9 codes 250 or 249), and intestinal infection (GI; ICD-9 codes 001-009). Use of the ED data was in accordance with agreements with the hospitals and the Georgia Hospital Association and this study was approved prior to its conduct by the Emory University Institutional Review Board.

2.2. Statistical methods—Analyses were restricted to the warm season, which was defined as May-September. Analyses were conducted using Poisson generalized linear models allowing for overdispersion. The primary models assessed the relationship between daily maximum temperature and daily counts of ED visits for the selected outcomes, modeling daily maximum temperature with linear, quadratic and cubic terms to allow for non-linear relationships. We selected daily maximum temperature as our primary temperature metric because it may represent the greatest physiological stress due to temperature on a given day, it typically occurs at a time of day when people may be active and exposed to outdoor temperatures (particularly children) (Barnett et al., 2010), and it has been used in some previous studies of temperature and morbidity (Saha et al., 2015; Ye et al., 2001). We assessed models for the daily counts of ED visits for the selected outcomes for all ages combined and models stratified by age group (0-4, 5-18, 19-64, and ≥65 years). Associations were assessed for maximum temperature at single-day lags of 0-6 days, using a
separate model for each lag. Distributed lag models were not considered in this analysis given that non-linear modeling of temperature effects would result in overly complex models that would be difficult to interpret. The primary models controlled for maximum dew-point temperature using cubic terms at the same lag as used for the maximum temperature terms. All models controlled for time trends using indicator variables for year, a cubic spline for day of the warm season with monthly knots within the warm season for each year, and terms for the interaction between the linear term for day of the warm season and each year indicator variable.

Models also controlled for day of week, federal holidays, weekend July 4th days, and periods of hospital participation using indicator variables. Using these models, we estimated rate ratios (RR) for various temperature changes (in degrees Celsius) from a reference maximum temperature of 27 °C (which was the 25th percentile value of daily maximum temperature during the study period) to a given temperature by applying parameter estimate values from the linear, squared, and cubic terms for maximum temperature to the temperature contrast being considered. We assessed the degree of evidence for non-linear effects using a likelihood ratio test for the significance of the squared and cubic temperature terms jointly. Our primary models assumed constant effects for the entire time series (1993-2012). This assumption was assessed in secondary analyses that modeled effects separately for each 5-year interval within the study period for selected outcomes.

In secondary models, we assessed the impact of using outcome definitions that considered the presence of the selected ICD-9 codes in any of the listed diagnoses rather than in the primary diagnosis only. We also compared the results of models assessing daily maximum apparent temperature (a metric that is based on both temperature and humidity) (Steadman, 1984) to those assessing maximum temperature as the exposure metric. Models assessing maximum apparent temperature were the same as primary models, but did not control for dew-point temperature; rate ratios were estimated for various temperature changes (in degrees Celsius) from a reference maximum apparent temperature of 28 °C (which was the 25th percentile value of daily maximum apparent temperature during the study time period).

We also conducted several types of sensitivity analyses to assess the stability of our results to changes in model specification. We considered models that controlled for minimum dew-point temperature (using cubic terms that matched the lag used for maximum temperature) rather than maximum dew-point temperature; and models that controlled for other meteorological variables (in separate models, at lag 0), including average wind speed (using a linear term), mean barometric pressure (using a linear term, in millibars), and the presence of precipitation (using an indicator variable for whether total daily precipitation was >0). We also considered models that controlled for various air pollutant concentrations (modeled using linear terms for the concentration at lag 0, and restricted to the 1993-2010 period during which population-weighted air pollutant data were available). Results of the models controlling for air pollutants were compared with results of primary models restricted to the same time period.
3. Results

3.1. Descriptive analysis results

The year-round maximum temperature in Atlanta during 1993-2012 ranged from −7.8 °C to 40.6 °C, with the warmest temperatures occurring during May-September (the warm season definition used in this analysis) (see Supplemental Material, Figure S1). Descriptive statistics for daily maximum temperature and other meteorological measures for Atlanta during the warm seasons for 1993-2012 are shown in Table 1, and correlations between these measures are shown in Table 2. Daily maximum temperature was highly correlated (Spearman correlation coefficient ≥0.94) with daily maximum apparent temperature. Correlations between maximum temperatures at lag 0 and the other lags considered in our models are shown in Supplemental Material, Table S1. There was no apparent trend in daily warm season maximum temperatures across years during the study period (See Supplemental Material, Figure S2).

The data set for this analysis included a total of 9,856,015 ED visits to Atlanta metropolitan area hospitals during the warm seasons of 1993-2012, of which 6,994,110 had primary ICD-9 codes indicating internal causes (Table 3). The mean daily count of ED visits for internal causes for all ages was 2,285.7, with the mean daily counts for the specific conditions considered in the analysis, for all ages, ranging from 2.8 for heat illness to 294.3 for all respiratory diseases. For several outcomes, mean daily counts were low within some age categories (e.g., on average there were no ED visits for HEAT among 0-4 year olds), preventing estimation of temperature effects for these outcomes for these age groups. Information about counts of ED visits for outcomes defined based on any listed diagnosis is provided in the Supplemental Material, Table S2.

3.2. Primary model results

Estimates from the primary models of the association between maximum temperature at lags 0 through 6 and all outcomes, evaluated for a change in maximum temperature from 27 °C to 32 °C (representing a change from the 25th to the 75th percentile of maximum temperature during the study period), are shown in Figure 1. For all internal causes, heat illness, fluid and electrolyte imbalances, and selected renal conditions (RENAL and NEPH), for all ages combined, this degree of change in maximum temperature was associated with significant increases in ED visits (Supplemental Material, Table S3). RRs were generally highest for lag 0 with RRs of 1.012 (95% confidence interval (CI) 1.008-1.016) for all internal causes, 4.535 (95% CI 4.112-5.002) for heat illness, and 1.125 (95% CI 1.102-1.149) for fluid and electrolyte imbalances at lag 0; and an RR of 1.068 (95% CI 1.053-1.083) for all renal disease at lag 1. Statistical evidence of non-linear effects of maximum temperature at lag 0 was observed for heat illness, fluid and electrolyte imbalances, and nephritis and nephrotic syndrome, with effects increasing most rapidly at the highest temperatures (Supplemental Material, Figure S3). There were no consistent trends in the strengths of the associations between maximum temperature at lag 0 and ED visits for these outcomes when examined by 5-year time period (Supplemental Material, Figure S4), although there was a suggestion of a slight decrease in RRs for some outcomes across the study period.

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When considered by age group, RRs for these conditions could not be estimated for some age groups due to low daily counts. For all internal causes combined, RRs at lag 0 for a change in maximum temperature of 27 °C to 32 °C were highest for the ≥65 year age group, followed by the 0-4 year age group; RRs for the 5-18 year age group were small, imprecise, and not statistically significant. There was some evidence of “temporal displacement” (in which a reduction in the susceptible population due to short-term effects of high temperature results in protective associations at longer lags) in the two youngest age groups (0-4 years and 5-18 years), with statistically significant protective RRs observed at lags of 5-6 days. Unlike the all internal causes outcome group, RRs were highest in the 19-64 year age group for fluid and electrolyte imbalances (lag 0) and renal diseases (lag 1). Statistically significant associations between maximum temperature and ED visits among children were observed for all internal causes (lags 0 and 1, 0-4 year age group), fluid and electrolyte disorders (lags 0 and 1, 5-18 year age group), and heat-related illness (lags 0 through 4, 5-18 year age group), but associations for heat-related illness and renal diseases could not be fully assessed among children due to low ED visit counts.

Observed associations between maximum temperature and selected cardiovascular conditions, evaluated for a change in maximum temperature from 27 °C to 32 °C, are shown in Figure 1 and Supplemental Material, Table S4. For all ages combined, statistically significant protective RRs were observed for the all circulatory outcome group at lags of 1-3 days, for hypertension at lags of 0-1 days, and for CHF at lags of 0-6 days. When considered by age group, RRs for cardiovascular conditions were predominantly close to null or less than 1 for the 65+ year age group. RRs for IHD and stroke were generally greater than 1 in the 19-64 year age group, with statistically significant RRs of 1.028 (95% CI 1.002-1.054) at lag 4 for IHD and 1.039 (95% CI 1.001-1.078) at lag 1 for stroke. Associations with cardiovascular outcomes among children could be assessed only for the all circulatory disease and dysrhythmia outcome groups, for which confidence intervals were wide due to low daily counts.

Estimated associations between maximum temperature and respiratory conditions, evaluated for a change in maximum temperature from 27 °C to 32 °C, are shown in Figure 1 and Supplemental Material, Table S5. RRs were predominantly less than 1 for the all-respiratory disease outcome group, particularly in the youngest age groups (0-4 years and 5-18 years). No statistically significant RRs were observed for this temperature contrast for pneumonia. For COPD, RRs were close to 1 or protective, with age-specific effects estimated only in the adult age groups. In contrast, for asthma/wheeze, statistically significant RRs of approximately 1.02 were observed for all ages combined at lags 2-4, with strongest associations observed among 5-18 year olds (RR of 1.059 (95% CI 1.030-1.088) at lag 2).

For diabetes, statistically significant associations at lag 0 were observed for all ages combined (RR of 1.034 (95% CI 1.014-1.054) and in both adult age groups, with strongest associations in the ≥65 year age group (RR of 1.050 (95% CI 1.008-1.095)). For gastrointestinal infections, statistically significant RRs greater than 1 were observed for all ages combined at lag 0 (RR=1.047, 95% CI 1.014-1.082), for ages 0-4 years at lag 0 and for ages 5-18 years at lags 0,1 and 6. Statistically significant RRs for gastrointestinal infections
that were less than 1 were observed for the 19-64 year age group at a lags of 3 and 4 days and for the ≥65 year age group at a lag of 6 days.

3.3. Secondary analysis results

Observed associations from models in which outcome groups were defined based on the presence of the selected codes in any of the recorded diagnoses differed from those observed with the primary models in some cases. In particular, statistically significant positive associations with lag 0 maximum temperature were observed for the all circulatory outcome group, hypertension, and COPD compared to close to 1 and/or protective associations when only primary diagnoses were considered (Figure 2). In contrast, associations with diabetes and renal disorders were slightly weaker when considering all diagnoses versus only primary diagnoses (Figure 2). Associations with fluid and electrolyte disorders and asthma/wheeze were also generally weaker when considering all diagnoses compared to only the primary diagnosis; exceptions to this were among children aged 0-4 years for fluid and electrolyte disorders and among 19-64 year olds for asthma/wheeze (Figure 3).

Results of models that used maximum apparent temperature as the exposure metric were generally similar to results of models that used maximum temperature as the exposure metric. RRs for a change in maximum apparent temperature from 28 °C to 33 °C were within 5% of RRs for a change in maximum temperature from 27 °C to 32 °C, except for heat-related illness (for which maximum apparent temperature RRs were 5.5-18% higher) and dysrhythmia in the 0-4 year age group (for which maximum apparent temperature RRs were 5.3-5.9% higher) (Supplemental Material, Table S6). In more cases, there was a difference in statistical significance of the RRs between models using the different exposure metrics. In approximately 60% of those cases, the p-value for a maximum apparent temperature change from 28 °C to 33 °C was statistically significant while the p-value for a maximum temperature change from 27 °C to 32 °C was not.

3.4. Sensitivity analysis results

Models that controlled for minimum dew-point temperature rather than maximum dew-point temperature, and models that controlled for average wind speed, barometric pressure, or the presence of precipitation yielded very similar results to the primary models that did not control for these variables. Overall, controlling for air pollutants did not substantially change the model results or conclusions. Models that controlled for ozone gave slightly attenuated RRs for some outcomes in some age groups (e.g., for all internal causes among ages overall and the 0-4 and 5-18 year age groups at lag 0, and for fluid and electrolyte imbalances among ages overall and for the 5-18 and 19-64 year age groups at lag 0) but gave slightly higher RRs for other outcomes and age groups. In most cases, however, control for air pollutants changed RRs for maximum temperature by less than 10%. Changes in maximum temperature RRs by 10% or more when controlling for pollutants were only found for selected outcome-age group-pollutant control combinations (details shown in Supplemental Material, Table S7). In all of these cases, the RR for maximum temperature was stronger with control for the pollutant compared with the primary model.
4. Discussion

We observed statistically significant associations between daily warm-season maximum temperature and ED visits in Atlanta in our primary models for several outcomes, including all internal causes, heat-related illness, fluid and electrolyte disorders, renal diseases, asthma/wheeze, diabetes and gastrointestinal infections. While the rate ratios for a change in maximum temperature from the 25th percentile to the 75th percentile were sometimes highest for those aged 65 years and older (e.g., for all internal causes, and diabetes), rate ratios were highest for some conditions among younger adults aged 19-64 years (e.g., for renal diseases and fluid and electrolyte disorders) or children (e.g., for gastrointestinal infections and asthma/wheeze). Of particular note, we observed statistically significant positive associations between daily maximum temperature and emergency department visits for several conditions among children (for all internal causes and GI infections among children aged 0-4 and for fluid and electrolyte disorders, heat-related illness, asthma/wheeze and gastrointestinal infections among children aged 5-18 years). However, we did not have sufficient power (due to low daily ED visit counts) to assess associations with some diseases in some age groups, notably for renal diseases in the pediatric age groups and for heat-related illness in all age groups except the 5-18 year age group.

Our results are in general agreement with several previous studies that have shown increasing ambient temperature to be associated with increases in hospital admissions or ED visits for internal or all natural causes, fluid and electrolyte disorders, heat-related illness and renal disease (Basu et al., 2012; Choudhary and Vaidyanathan, 2014; Fletcher et al., 2012; Green et al., 2010; Gronlund et al., 2014; Kovats et al., 2004; Li et al., 2012; Linares and Diaz, 2008; Lippmann et al., 2013; Ostro et al., 2010; Saha et al., 2015; Wang and Lin, 2014). Similar to our findings, several previous studies have also found associations between high ambient temperature and diabetes ED visits or hospital admissions, with strongest effects observed in the elderly if analyses were stratified by age (Basu et al., 2012; Green et al., 2010; Ostro et al., 2010; Wang and Lin, 2014); and several previous studies have found associations between high ambient temperature and gastrointestinal infections, with effects particularly observed in children if analyses were stratified by age (Basu et al., 2012; Green et al., 2010; Lam, 2007; Pudpong and Hajat, 2011).

We observed no significant association with the overall respiratory disease outcome group, although some positive rate ratios at lags of 0-1 were observed among those aged 65 years and older. Several previous studies have found associations between high ambient temperature and measures of overall respiratory morbidity, with effects often observed primarily or only among the elderly (Anderson et al., 2013; Green et al., 2010; Gronlund et al., 2014; Kovats et al., 2004; Lin et al., 2009; Linares and Diaz, 2008; Michelozzi et al., 2009; Ostro et al., 2010; Pudpong and Hajat, 2011; Wichmann et al., 2011). However, other studies have not found associations between high ambient temperatures and respiratory morbidity (Basu et al., 2012; Son et al., 2014). Some previous studies have observed associations between high temperatures and pneumonia morbidity (Green et al., 2010; Ostro et al., 2010), but we did not. We observed an association between high ambient temperatures and asthma, particularly in the 5-18 year age group. Some previous studies have also found associations between high temperature and asthma hospital admissions (Lin et al., 2009) or
ED visits for childhood asthma (Xu et al., 2013), while other studies have found no association with asthma ED visits (Wang and Lin, 2014), or associations only in some months of the year (Buckley and Richardson, 2012).

We observed no associations or negative associations between high ambient temperatures and cardiovascular conditions in analyses that considered only the primary diagnosis. Some previous studies have observed positive associations between high temperatures and hospital admissions or ED visits for cardiovascular disease outcomes (Basu et al., 2012; Green et al., 2010; Koken et al., 2003; Lin et al., 2009; Ostro et al., 2010; Schwartz et al., 2004; Son et al., 2014). Other studies have found no association or negative associations between high temperatures and ED visits or hospital admissions for cardiovascular disease outcomes (Basu et al., 2012; Grønlund et al., 2014; Koken et al., 2003; Kovats et al., 2004; Linares and Diaz, 2008; Michelozzi et al., 2009; Wang and Lin, 2014; Wichmann et al., 2011; Wichmann et al., 2012).

Most previous studies of acute associations between heat and morbidity (either measured using hospital admission or ED visit counts) have considered only the primary diagnosis. This approach has the advantage of optimizing specificity of disease classification. However, results of these analyses may be affected by coding practices that determine the order in which the diagnoses are listed. A lack of an association with an outcome based on the primary diagnosis only may not indicate that heat does not have an impact on that outcome. To assess this issue, we also considered analyses in which outcomes were defined based on the selected ICD-9 codes being present in any of the listed diagnoses rather than as the primary diagnosis only. These analyses showed some differences in observed associations between the two approaches. Differences were particularly striking for cardiovascular outcomes, for which significant positive associations were observed only when all diagnoses were considered. A study of California heat waves took a similar approach, including the first 9 diagnoses in defining specific outcomes among ED visits and hospital admissions, and also observed a significant association between heat wave periods and ED visits for cardiovascular diseases (Knowlton et al., 2009). It may be that heat causes exacerbation of cardiovascular diseases but people with cardiovascular diseases tend to be given other primary diagnoses on hot days (such as diagnoses of fluid and electrolyte or renal disorders). The potential importance of issues of how visits are coded for hospital admissions was previously pointed out by Kilbourne (Kilbourne, 1999), and differences in estimates of excess diagnosis-specific hospital admissions associated with the 1995 Chicago heat wave were noted between analyses considering only primary vs. all diagnoses (Semenza et al., 1999), with higher estimates of percent excess cardiovascular disease admissions when all diagnoses were considered. In addition, discrepancies have previously been noted between effects of heat in studies of mortality and morbidity, particularly in relation to cardiovascular disease, with weaker heat associations being observed in morbidity studies (Kovats et al., 2004; Linares and Diaz, 2008; Michelozzi et al., 2009; Turner et al., 2012); our results support the suggestion of Kilborne (Kilbourne, 1999) that coding issues may be part of the explanation. Another explanation that has been offered for weak apparent associations between temperature and cardiovascular disease morbidity is that people might die quickly of cardiovascular conditions as a result of heat exposure, before they can come to medical attention (Kovats et al., 2004; Linares and Diaz, 2008).
A major contribution of our study is examination of ambient temperature effects on ED visits across all ages, including children. The significant associations that we observed between high ambient temperatures and several outcomes in children support evidence for these associations from previous studies, including associations with various measures of morbidity for gastrointestinal infections (Basu et al., 2012; Checkley et al., 2000; Chou et al., 2010; Green et al., 2010; Hashizume et al., 2007; Lam, 2007; Pudpong and Hajat, 2011), asthma (Xu et al., 2013), heat-related illness (Knowlton et al., 2009; Lippmann et al., 2013), fluid and electrolyte imbalances (Basu et al., 2012; Green et al., 2010; Knowlton et al., 2009), and overall visits (Kovats et al., 2004).

Because very different approaches to modeling and quantifying heat effects have been used in previous studies, it is difficult to compare the magnitudes of our observed effects with those in other studies. We modeled temperature effects using linear, quadratic and cubic terms for maximum temperature and estimated rate ratios for a change in maximum temperature from the 25th percentile (27 °C) to the 75th percentile (32 °C) among warm-season temperatures based on all three temperature terms. We also assessed evidence for nonlinearity in the relationship between maximum temperature and the log of the daily ED visit count by examining the contribution of the quadratic and cubic terms to model fit. This approach differs from most previous studies, which have either restricted the analysis to the warm or summer season and assumed linearity, identified a threshold temperature and assumed linearity above the threshold, or tested for non-linearity without finding evidence of non-linear relationships (Turner et al., 2012). Our approach is somewhat similar to, although simpler than, the approach used in some previous studies (Gronlund et al., 2014; Xu et al., 2013) which calculated effects for specified temperature changes based on natural cubic splines for temperature with 3 or 5 degrees of freedom. Using our approach, we found evidence of non-linearity for some outcomes within the warm season, including fluid and electrolyte disorders, heat-related illness and nephritis/nephrotic syndrome. For these conditions, rate ratios relative to 27 °C increased more rapidly at higher temperatures, indicating that the RRs that we present are lower than those that would be observed if we considered temperature contrasts of a similar magnitude at higher temperatures.

In our primary models we did not control for air pollutants. As Buckley, et al. pointed out, consideration of potential causal relationships between ambient temperature, air pollutants and the outcomes of interest in studies of acute ambient temperature health effects generally does not support controlling for air pollutants purely as potential confounders themselves (Buckley et al., 2014), because ambient temperature likely influences air pollutant levels, rather than air pollutant levels influencing temperature. However, inclusion of air pollutants in the model could control for confounding by some other unmeasured factors that might be associated with air pollution. In our analyses, including terms for air pollutants in the models generally did not substantially change the rate ratios for the relationship between ambient temperature and the outcomes that we considered, and when it did, maximum temperature RRs were increased with control for the pollutant rather than decreased.

The length of the time series in our study is a substantial strength of the study. A limitation of this study design is the fact that associations with high temperatures cannot be assessed for age group-outcome combinations with low daily ED visit counts, for which the public
health impact would be expected to be small. In addition, measurement error, residual confounding due to factors we were not able to consider, or other types of model misspecification could have impacted our results. However, we did not find evidence for substantial residual confounding due to the factors that we considered in sensitivity analyses. Finally, further examination of the effects of high temperatures on health outcomes, such as consideration of the effects of sustained high temperatures, is also important but is beyond the scope of the current analysis.

Atlanta is a large, growing metropolitan area with mild winters, hot and humid summers, and a high air conditioning prevalence (>94% according to the 2011 American Housing Survey (Commerce, 2011)). These characteristics may lead to the health effects of high ambient temperatures being different in Atlanta than in other locations. Ostro et al. found that increased air conditioning prevalence led to reductions in the magnitudes of the association between temperature and hospital admissions for several outcomes (Ostro et al., 2010). In addition, there is some evidence that associations between high ambient temperature and various health outcomes may be somewhat weaker in southern regions of the United States than in northern regions of the United States, possibly due to population adaptation to high temperatures (Anderson and Bell, 2009; Saha et al., 2015), which could include air conditioning use or other types of adaptations. The high prevalence of air conditioning in Atlanta, for example, may partially explain the lack of observed associations between maximum temperature and some outcomes in our analysis, such as congestive heart failure, if people with these conditions follow recommendations to stay in air conditioned environments on very warm days.

5. Conclusions

We observed associations between high warm-season ambient temperatures and ED visits for several conditions. Associations were observed in all age groups and the strength of association for particular conditions varied by age group. Our findings have importance for public health planning in Atlanta, for which comprehensive estimates of heat effects on morbidity across all ages were not previously available, and may be applicable to other cities with similar climate and environmental conditions. Because they include age-group specific effect estimates across all age groups, these heat effect estimates may allow more accurate estimation of expected burdens on EDs in Atlanta due to high ambient temperatures and better health-impact projections for climate change scenarios. They also provide additional evidence to help guide interventions to prevent health effects from heat, and suggest that interventions will be most effective if they account for heat health impacts across ages, including younger age groups.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>ED</td>
<td>emergency department</td>
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<tr>
<td>RR</td>
<td>rate ratio</td>
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<tr>
<td>INTERN</td>
<td>All internal causes</td>
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<td>HEAT</td>
<td>Heat illness</td>
</tr>
<tr>
<td>FLEL</td>
<td>Fluid and electrolyte imbalance</td>
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<td>All renal disease</td>
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<td>Nephritis and nephrotic syndrome</td>
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</tr>
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</tr>
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<td>IHD</td>
<td>Ischemic heart disease</td>
</tr>
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<td>DYS</td>
<td>Dysrhythmia</td>
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<tr>
<td>CHF</td>
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</tr>
<tr>
<td>PNEU</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>COPD</td>
<td>Chronic Obstructive Pulmonary Disease</td>
</tr>
<tr>
<td>ASW</td>
<td>Asthma/wheeze</td>
</tr>
<tr>
<td>DIA</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>GI</td>
<td>Intestinal infection</td>
</tr>
</tbody>
</table>

References


### Highlights

- Warm-season maximum temperature was associated with ED visits for several outcomes
- Associations strongest among ages 65+ yrs for all internal causes and diabetes
- Associations strongest among ages 19-64 yrs for fluid/electrolyte/renal diagnoses
- Associations strongest among ages 5-18 yrs for asthma/wheeze, intestinal infections
Figure 1.
Estimated rate ratios for ED visits for outcome groups, defined based on the primary diagnosis, in relation to a change from a maximum temperature of 27 °C to 32 °C (approximately 1 IQR), by age category and lag, Atlanta, warm season (May-September), 1993-2012. Abbreviations: INTERN: All internal causes; HEAT: Heat illness; FLEL: Fluid and electrolyte imbalance; RENAL: All renal disease; NEPH: Nephritis and nephrotic syndrome; CIRC: All circulatory system disease; HT: Hypertension; IHD: Ischemic heart disease; DYS: Dysrhythmia; CHF: Congestive heart failure; STK: Ischemic stroke; RESP: All respiratory system disease; PNEU: Pneumonia; COPD: Chronic obstructive pulmonary disease; ASW: Asthma/wheeze; DIA: Diabetes mellitus; GI: Intestinal infection
Figure 2.
Estimated rate ratios for ED visits for outcome groups, defined based on the primary diagnosis only (PRIMARY) or on any listed diagnosis (ANY), in relation to a change from a maximum temperature of 27 °C to 32 °C (approximately 1 IQR) at lag 0, for cardiovascular outcomes, diabetes, COPD and renal disease, ages overall, Atlanta, warm season (May-September), 1993-2012. CIRC: All circulatory system disease; HT: Hypertension; IHD: Ischemic heart disease; DYS: Dysrhythmia; CHF: Congestive heart failure; STK: Ischemic stroke; DIA: Diabetes mellitus; COPD: Chronic obstructive pulmonary disease; RENAL: All renal disease
Figure 3.
Estimated rate ratios for ED visits for outcome groups, defined based on the primary diagnosis only (PRIMARY) or on any listed diagnosis (ANY), in relation to a change from a maximum temperature of 27 °C to 32 °C (approximately 1 IQR) at lag 0, for fluid and electrolyte disorders and asthma/wheeze, by age category, Atlanta, warm season (May-September), 1993-2012
### Table 1
Descriptive Statistics for daily meteorological measures, Atlanta, Warm Season (May-September), 1993-2012

<table>
<thead>
<tr>
<th>Meteorological measure</th>
<th># Days</th>
<th>Mean</th>
<th>SD</th>
<th>Min.</th>
<th>25th percentile</th>
<th>50th percentile</th>
<th>75th percentile</th>
<th>Max.</th>
<th>1</th>
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<tbody>
<tr>
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<td>3060</td>
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<td>13.3</td>
<td>27.2</td>
<td>30.0</td>
<td>32.0</td>
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<tr>
<td>Max apparent temperature, °C</td>
<td>3060</td>
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<td>14.2</td>
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<td>33.0</td>
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<tr>
<td>Max dew-point temperature, °C</td>
<td>3060</td>
<td>19.6</td>
<td>3.6</td>
<td>2.2</td>
<td>17.8</td>
<td>20.6</td>
<td>22.2</td>
<td>28.0</td>
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<tr>
<td>Min dew-point temperature, °C</td>
<td>3060</td>
<td>15.5</td>
<td>4.4</td>
<td>−3.3</td>
<td>12.8</td>
<td>16.7</td>
<td>18.9</td>
<td>22.8</td>
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</tr>
<tr>
<td>Mean barometric pressure (millibars)</td>
<td>2754</td>
<td>980.1</td>
<td>3.5</td>
<td>964.5</td>
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<td>990.1</td>
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<td>Average daily wind speed, m/s</td>
<td>3060</td>
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<td>1.2</td>
<td>0.0</td>
<td>2.4</td>
<td>3.1</td>
<td>3.8</td>
<td>11.0</td>
<td></td>
</tr>
<tr>
<td>Total daily precipitation, mm</td>
<td>3060</td>
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<td>16.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>1.0</td>
<td>290.0</td>
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</tbody>
</table>

1 Apparent temperature was calculated hourly using the formula $AT = -1.3 + 0.92\times T + 2.2\times VP$, where $T$ is the temperature in degrees Celsius and $VP$ is the vapor pressure in kilopascals, which was calculated using the formula $VP = 0.6112\times e^{17.67\times Td/(Td+243.5)}$ in which $Td$ is the dew-point temperature (Bolton, 1980; Steadman, 1984). Maximum apparent temperature for each day was the maximum of the hourly values for a given day.
Table 2

Spearman correlations between daily meteorological measures, Atlanta, Warm Season (May-September), 1993-2012 [TMX=maximum temperature (°C), ATMX=maximum apparent temperature (°C), TDMX=maximum dew-point temperature (°C), TDMN=minimum dew-point temperature (°C), BPGT=mean barometric pressure (millibars), AVWSP=average wind speed (m/s), TOTPRE=total daily precipitation (mm)].

<table>
<thead>
<tr>
<th></th>
<th>TMX</th>
<th>ATMX</th>
<th>TDMX</th>
<th>TDMN</th>
<th>BPGT</th>
<th>AVWSP</th>
<th>TOTPRE</th>
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<td>ATMX</td>
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<td>1.00</td>
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<td>TDMX</td>
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<td>0.66</td>
<td>1.00</td>
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<td>0.57</td>
<td>0.88</td>
<td>1.00</td>
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<tr>
<td>BPGT</td>
<td>−0.01</td>
<td>−0.03</td>
<td>−0.12</td>
<td>−0.03</td>
<td>1.00</td>
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<tr>
<td>AVWSP</td>
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<td>−0.20</td>
<td>−0.08</td>
<td>−0.14</td>
<td>−0.18</td>
<td>1.00</td>
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</tr>
<tr>
<td>TOTPRE</td>
<td>−0.18</td>
<td>−0.07</td>
<td>0.32</td>
<td>0.28</td>
<td>−0.17</td>
<td>0.03</td>
<td>1.00</td>
</tr>
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</table>

1 Apparent temperature was calculated hourly using the formula AT = −1.3+0.92*T+2.2VP, where T is the temperature in degrees Celsius and VP is the vapor pressure in kilopascals, which was calculated using the formula VP=0.6112*e^{17.67*Td/(Td+243.5)} in which Td is the dew-point temperature (Bolton, 1980; Steadman, 1984). Maximum apparent temperature for each day was the maximum of the hourly values for a given day.
Table 3

Descriptive statistics for number of ED visits based on primary ICD-9 code, Atlanta, 1993-2012, Warm season (May-September)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Abbreviation</th>
<th>ICD9 Code(s)</th>
<th># Days</th>
<th>Total # Visits</th>
<th>Mean #/Day</th>
<th>SD #/Day</th>
<th>Mean #/Day, 0-4 yrs</th>
<th>Mean #/Day, 5-18 yrs</th>
<th>Mean #/Day, 19-64 yrs</th>
<th>Mean #/Day, 65+ yrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>All ED visits</td>
<td>EDALL</td>
<td>All</td>
<td>30</td>
<td>9,856</td>
<td>3.22</td>
<td>1.45</td>
<td>392</td>
<td>196</td>
<td>359</td>
<td></td>
</tr>
<tr>
<td>All internal causes</td>
<td>INTER</td>
<td>001-799</td>
<td>30</td>
<td>6,994</td>
<td>2.28</td>
<td>1.14</td>
<td>292</td>
<td>142</td>
<td>285</td>
<td></td>
</tr>
<tr>
<td>Heat illness</td>
<td>HEAT</td>
<td>992</td>
<td>30</td>
<td>8,594</td>
<td>2.8</td>
<td>4.3</td>
<td>0.0</td>
<td>0.6</td>
<td>2.0</td>
<td>0.2</td>
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<tr>
<td>Fluid and electrolyte imbalance</td>
<td>FLEL</td>
<td>276</td>
<td>30</td>
<td>66,369</td>
<td>21.7</td>
<td>12.8</td>
<td>2.1</td>
<td>2.0</td>
<td>11.1</td>
<td>6.5</td>
</tr>
<tr>
<td>All renal disease</td>
<td>RENAL</td>
<td>580-593</td>
<td>30</td>
<td>140,678</td>
<td>46.0</td>
<td>27.8</td>
<td>0.5</td>
<td>2.3</td>
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<tr>
<td>Nephritis and nephrotic syndrome</td>
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<td>580-589</td>
<td>30</td>
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<td>3.7</td>
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<td>All circulatory system disease</td>
<td>CIRC</td>
<td>390-459</td>
<td>30</td>
<td>396,353</td>
<td>129.5</td>
<td>72.5</td>
<td>0.7</td>
<td>1.3</td>
<td>72.3</td>
<td>55.2</td>
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<tr>
<td>Hypertension</td>
<td>HT</td>
<td>401-405</td>
<td>30</td>
<td>84,206</td>
<td>27.5</td>
<td>19.0</td>
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<td>6.8</td>
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<tr>
<td>Ischemic heart disease</td>
<td>IHD</td>
<td>410-414</td>
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<td>66,522</td>
<td>21.7</td>
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<td>Dysrhythmia</td>
<td>DYS</td>
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<td>Congestive heart failure</td>
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<td>RESP</td>
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<td>30</td>
<td>900,540</td>
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<td>161.0</td>
<td>74.0</td>
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<td>PNEU</td>
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<td>COPD</td>
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<td>Outcome</td>
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<td>ICD9 Code(s)</td>
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<td>Mean #/Day, 19-64 yrs</td>
<td>Mean #/Day, 65+ yrs</td>
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<td>Asthma/wheeze</td>
<td>ASW&lt;sup&gt;a&lt;/sup&gt;</td>
<td>493 or 7896.09/0.07</td>
<td>30 60</td>
<td>177.0 20</td>
<td>57.8</td>
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<td>13.7</td>
<td>16.8</td>
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</tr>
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<td>Diabetes mellitus</td>
<td>DIA&lt;sup&gt;b&lt;/sup&gt;</td>
<td>250 or 249</td>
<td>30 60</td>
<td>70.07 6</td>
<td>22.9</td>
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<td>5.0</td>
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<td>30 60</td>
<td>30.61 0</td>
<td>10.0</td>
<td>6.4</td>
<td>3.2</td>
<td>1.7</td>
<td>3.8</td>
<td>1.3</td>
</tr>
</tbody>
</table>

<sup>a</sup>ASW = 493 or 7896.09 (before 10/1/1998) or 786.07 (on or after 10/1/1998)

<sup>b</sup>DIA = 250 or 249 (on or after 10/1/2008)