

Examination of Development Sites – ACT 7

Mrs J Trimmer

DS30/Matter 3

Residents have been objecting to the pollution created by the businesses on this site for many years. Recently due to the representations of the local residents group – Stop Horn Lane Pollution and the Local MP, Angie Bray, the Environment Agency has increased its monitoring of the Waste Disposal Site, Gowing & Pursey, and achieved a reduction in pollutants. None the less pollution levels still remain in excess of national and EU limits as set out on the www.londonair.org.uk web site maintained by King's College and replicated on the Ealing council site. Copies are attached. In addition the figures for 2012 reflect a reduction of approximately 36% (according to the Environment Agency) due to a publicly funded program of road coatings which has now ceased, so I would anticipate the figures for 2013 will show an increase.

I am concerned about the health effects of this pollution. A summary of the health consequences is contained in Annex A to Defra's publication “Air Pollution: Action in a changing climate” attached. In addition, the pollution from waste disposal sites seems of particular concern – see attached article produced by the environmental research group at Kings College, London. I also attach print outs from the NHS Local health website (www.localhealth.org.uk) which show that Chronic Obstructive Pulmonary Disease and Heart disease hospital admissions are high in the area and well above national averages. In fact I believe that the proposals for this site should now be considered by the new Health and Wellbeing Board set up under the new Health and Social Care Act 2012 as I note they have a remit to consider the wider social, environmental and economic factors that impact on health and well being in all policy and decision making.

It seems to me that the current proposals to relocated the builders yard from the South Side of the railway, allow additional complementary uses and possibly also additional bus interchange facilities can only increase the level of pollution, much of which is generated by the heavy and dusty vehicles using local roads to access the site.

Attachments:

1. Print outs of pollution monitoring statistics from London Air web site.
2. Defra publication.
3. Article on Particulate Matter from Waste Transfer Station Activity.
4. Print outs from Local Health web site.

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Air Quality Statistics:

The table below shows whether pollution levels recorded at the site you have selected remained within the Government's Air Quality Strategy Objectives in 2005. For further basic statistics click on the button below, or for more precise statistics, use the Statistics Calculator.

Pollutant	Objective	Was it achieved?	Value
PM10 Particulate	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	205
PM10 (redundant method)	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	230

** Note that results are excluded where analysers have not returned valid data for at least 90% of the year, unless a short-term objective was exceeded.*

** Note - all results from TEOM PM10 analysers from 2004 onward are now converted to reference equivalence using the volatile correction method. Results using the old 'TEOM*1.3' method are also shown for comparison where relevant. More information.*

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Pollutant	Objective	Was it achieved?	Value
PM10 Particulate	40 ug/m3 as an annual mean	NO	60
PM10 Particulate	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	182
PM10 (redundant method)	40 ug/m3 as an annual mean	NO	74
PM10 (redundant method)	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	224

** Note that results are excluded where analysts have not returned valid data for at least 90% of the year, unless a short-term objective was exceeded.*

** Note - all results from TEOM PM10 analysers from 2004 onward are now converted to reference equivalence using the volatile correction method. Results using the old 'TEOM*1.3' method are also shown for comparison where relevant. More information.*

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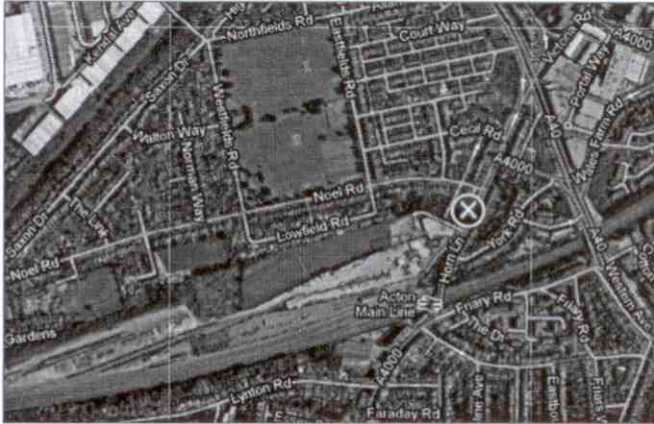
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Pollutant	Objective	Was it achieved?	Value
PM10 Particulate	40 ug/m3 as an annual mean	NO	43
PM10 Particulate	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	108
PM10 (redundant method)	40 ug/m3 as an annual mean	NO	53
PM10 (redundant method)	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	173

* Note that results are excluded where analysers have not returned valid data for at least 90% of the year, unless a short-term objective was exceeded.

* Note - all results from TEOM PM10 analysers from 2004 onward are now converted to reference equivalence using the volatile correction method. Results using the old 'TEOM*1.3' method are also shown for comparison where relevant. More information.

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The table below shows whether pollution levels recorded at the site you have selected remained within the Government's Air Quality Strategy Objectives in 2008. For further basic statistics click on the button below, or for more precise statistics, use the Statistics Calculator.

Pollutant	Objective	Was it achieved?	Value
PM10 Particulate	40 ug/m3 as an annual mean	NO	41
PM10 Particulate	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	96
PM10 (redundant method)	40 ug/m3 as an annual mean	NO	49
PM10 (redundant method)	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	137

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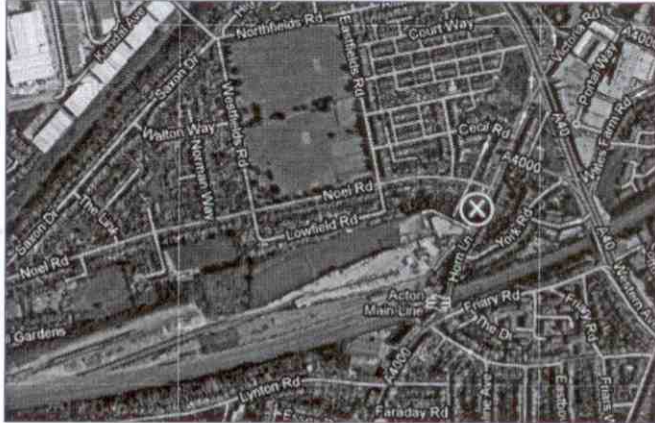
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Pollutant	Objective	Was it achieved?	Value
PM10 Particulate	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	73
PM10 (redundant method)	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	110

** Note that results are excluded where analysers have not returned valid data for at least 90% of the year, unless a short-term objective was exceeded.*

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Pollutant	Objective	Was it achieved?	Value
PM10 Particulate	40 ug/m3 as an annual mean	NO	42
PM10 Particulate	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	93
PM10 (redundant method)	40 ug/m3 as an annual mean	NO	47
PM10 (redundant method)	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	124

* Note that results are excluded where analysers have not returned valid data for at least 90% of the year, unless a short-term objective was exceeded.

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The table below shows whether pollution levels recorded at the site you have selected remained within the Government's Air Quality Strategy Objectives in 2011. For further basic statistics click on the button below, or for more precise statistics, use the Statistics Calculator.

Pollutant	Objective	Was it achieved?	Value
PM10 Particulate	40 ug/m3 as an annual mean	YES	36
PM10 Particulate	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	61
PM10 (redundant method)	40 ug/m3 as an annual mean	NO	40
PM10 (redundant method)	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	88

** Note that results are excluded where analysers have not returned valid data for at least 90% of the year, unless a short-term objective was exceeded.*

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The table below shows whether pollution levels recorded at the site you have selected remained within the Government's Air Quality Strategy Objectives in 2012. For further basic statistics click on the button below, or for more precise statistics, use the Statistics Calculator.

Pollutant	Objective	Was it achieved?	Value
PM10 Particulate	40 ug/m3 as an annual mean	YES	34
PM10 Particulate	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	53
PM10 (redundant method)	40 ug/m3 as an annual mean	YES	38
PM10 (redundant method)	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	89
Nitrogen Dioxide	200 ug/m3 as a 1 hour mean, not to be exceeded more than 18 times a year	YES	2
Nitrogen Dioxide	40 ug/m3 as an annual mean	NO	53

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The table below shows whether the pollution levels recorded at the site you have selected remained within the Government's Air Quality Strategy Objectives in 2013 (to date). For further basic statistics click on the button below, or for more precise statistics, use the Statistics Calculator.

Pollutant	Objective	Was it achieved?	Value
PM10 Particulate	40 ug/m3 as an annual mean	NO	41
PM10 Particulate	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	YES	35
PM10 (redundant method)	40 ug/m3 as an annual mean	NO	44
PM10 (redundant method)	50 ug/m3 as a 24 hour mean, not to be exceeded more than 35 times a year	NO	37
Nitrogen Dioxide	200 ug/m3 as a 1 hour mean, not to be exceeded more than 18 times a year	YES	0
Nitrogen Dioxide	40 ug/m3 as an annual mean	NO	58

* Note that these results cover from the start of the year to now, and are still subject to change until the end of the year.

* Note - all results from TEOM PM10 analysers from 2004 onward are now converted to reference equivalence using the volatile correction method. Results using the old 'TEOM*1.3' method are also shown for comparison where relevant. More information.

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Particulate Matter Oxidative Potential from Waste Transfer Station Activity

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BACKGROUND: Adverse cardiorespiratory health is associated with exposure to ambient particulate matter (PM). The highest PM concentrations in London occur in proximity to waste transfer stations (WTS), sites that experience high numbers of dust-laden, heavy-duty diesel vehicles transporting industrial and household waste.

OBJECTIVE: Our goal was to quantify the contribution of WTS emissions to ambient PM mass concentrations and oxidative potential.

METHODS: PM with a diameter < 10 μm (PM_{10}) samples were collected daily close to a WTS. PM_{10} mass concentrations measurements were source apportioned to estimate local versus background sources. PM oxidative potential was assessed using the extent of antioxidant depletion from a respiratory tract lining fluid model. Total trace metal and bioavailable iron concentrations were measured to determine their contribution to PM oxidative potential.

RESULTS: Elevated diurnal PM_{10} mass concentrations were observed on all days with WTS activity (Monday–Saturday). Variable PM oxidative potential, bioavailable iron, and total metal concentrations were observed on these days. The contribution of WTS emissions to PM at the sampling site, as predicted by microscale wind direction measurements, was correlated with ascorbate ($r = 0.80$; $p = 0.030$) and glutathione depletion ($r = 0.76$; $p = 0.046$). Increased PM oxidative potential was associated with aluminum, lead, and iron content.

CONCLUSIONS: PM arising from WTS activity has elevated trace metal concentrations and, as a consequence, increased oxidative potential. PM released by WTS activity should be considered a potential health risk to the nearby residential community.

KEY WORDS: antioxidants, iron, meteorology, oxidative potential, respiratory tract lining fluid, source apportionment, waste transfer sites. *Environ Health Perspect* 118:493–498 (2010). doi:10.1289/ehp.0901303 [Online 17 December 2009]

Waste transfer stations (WTSs) represent a hybrid of traffic and industrial microenvironments. These sites experience high volumes of heavy-duty diesel trucks and are often situated in densely populated urban centers (Gil and Kellerman 1993). These facilities serve as the link in the waste management system between the waste collection program and final disposal: Street waste collection vehicles discharge loads, avoiding uneconomic travel to distant landfill sites. Although this approach effectively decreases the overall air quality burden the waste management system poses on an urban center, communities in proximity to WTSs suffer from enhanced traffic flow and increased particulate matter (PM) levels arising from vehicular tailpipe/nontailpipe emissions and dust releases from laden trucks (Fuller and Baker 2001; Restrepo et al. 2004). Studies conducted in New York City (Maciejczyk et al. 2004; Restrepo et al. 2004) and London (Fuller and Baker 2008) have reported that the highest PM concentrations in these urban centers occur at sampling sites influenced by WTS-related emissions.

Concerned citizens inhabiting urban communities with WTSs across the United States have raised questions to the WTS working group of the National Environmental Justice

Advisory Committee about the impact of these facilities on the surrounding communities; degraded health and environmental conditions were highlighted, as well as wider negative impacts on the community. The consequence of the environmental disamenities of these facilities has resulted in disproportionate clustering in low-income areas in the United States [National Environmental Justice Advisory Council] (NEJAC) 2000]. Similar studies in the United Kingdom have identified a disproportionately high number of industrial sites, including WTSs in socio-economically deprived regions (Higgs and Langford 2009; Walker et al. 2005).

The relationship between extended air pollutant exposure and increased prevalence of asthma symptoms has been well documented (Romieu et al. 1995; Schwartz et al. 1993; Studnicka et al. 1997; Sunyer et al. 1997). In New York City, asthma hospitalization rates were stratified by the geographic location within each of the city's boroughs. Children living in the Bronx, specifically the South Bronx, suffered from hospitalization rates for asthma 70% and 700% greater than the average for New York City and New York state, respectively (New York City Department of Health 1999). Sources

contributing to adverse air quality in South Bronx include industrial facilities and vehicular traffic (Jackson 1995; Maantay 2007). The 19 WTSs housed in this low-income and densely inhabited community area also contributed significantly to increased PM exposures, with children experiencing elevated exposures due to the proximity of these facilities to schools, playgrounds, and residential buildings (Maciejczyk et al. 2004). To date, it is not clear whether such health effects are simply due to residents experiencing elevated PM concentrations or whether the toxicity of PM derived from WTS-related activities is elevated above that of urban PM.

Increasing interest has developed in assessing which PM physical and chemical characteristics specifically contribute to their observed toxicity. Recent literature has suggested that transition metals (Stohs and Bagchi 1995), organic species (polycyclic aromatic hydrocarbons and quinones) (Squadrito et al. 2001), and endotoxin (Thorne 2000) are associated with increased PM toxicity. The capacity of PM to induce toxicity (in particular, to elicit a respiratory and/or systemic inflammatory response) has been proposed to be a function of its oxidative potential (Li et al. 2002; Nel et al. 2001)—that is, the ability of PM to generate reactive oxygen species directly or indirectly to engender oxidative injury to the lung (Pourazar et al. 2005; Prahalad et al. 1999). We therefore evaluated PM oxidative potential to provide a simple aggregate measure of the particulate oxidative burden in the ambient airshed influenced by WTS emissions.

The objective of this study was to examine the pattern of elevated PM concentrations at a WTS located adjacent to a densely populated community in London. We used PM source apportionment techniques and microscale

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meteorologic information to identify pollutant episodes attributed to specific local sources, and quantified PM oxidative potential derived from WTS activity emissions.

Materials and Methods

Sampling location. The Bexley 4 sampling site, located in Erith, London Borough of Bexley, was selected for investigation because it is situated at the entrance of a WTS (Figure 1). This curbside site is also situated close to a metal reclamation yard and is near high-density residential buildings. This London Air Quality Network (LAQN) site has consistently breached the European Union Limit Value/U.K. Air Quality Strategy Objective for PM₁₀ (PM with an aerodynamic diameter < 10 μm), which allows 35 daily means per year > 50 μg/m³ [assessed as Tapered Element Oscillating Microbalance (TEOM) × 1.3]. Given the PM₁₀ exceedances above regulatory limits, this sampling site was declared an air quality management area in 2001: PM₁₀ concentrations were > 50 μg/m³ on 77 days and 116 days in 2001 and 2006, respectively (Fuller and Baker 2008).

PM sampling. PM₁₀ samples were collected daily on Teflon filters using the

automated cartridge collection unit installed on the TEOM. Sampling was conducted between 5 June 2002 and 30 August 2002. To increase filter mass loading, filters were exposed repeatedly between 2 and 5 times, but only on the same day of the week as previous collections (i.e., all Monday sampling occurred on the same filter). Filters were archived at -20°C in air-tight desiccation chambers until processed for oxidative screening and metal analysis.

Chemicals and Chelex water preparation. All chemicals used were obtained from Sigma Chemical Company Ltd. (Poole, U.K.), Fluka (Dorset, U.K.), or Laboratory Supplies (Poole, U.K.) and were of analytical grade or better quality. Ultrapure Chelex 100 resin-treated water was employed throughout to eliminate possible background metal contamination.

Sample preparation. Filters were extracted in high-pressure liquid chromatography-grade methanol by vortexing for 10 min before sonication using an MSE Soniprep 150 (23kHz) generator (Measuring & Scientific Equipment, London, UK) with a titanium probe at an amplitude of 5 μm for 30 sec. The methanol extract was dried under nitrogen and then resuspended to 150 μg/mL in Chelex

resin-treated water containing 5% methanol. Sample masses were determined from averaged TEOM measurements over the multiday exposure period.

Assessment of oxidative potential. We assessed the oxidative burden associated with collected PM by measuring antioxidant depletion using a synthetic human respiratory tract lining fluid (RTLFL) model as described by Mudway et al. (2004). The acellular RTLFL model provides a highly repeatable and rapid method of quantifying PM oxidative potential (Ayres et al. 2008). Assessment of oxidation by this assay is limited to intrinsic and non-latent redox components of PM: transition metal and quinone-dependent reactions.

Particle-free and known particle controls were run in parallel with filter samples to ensure interexperiment standardization. A residual oil fly ash sample, supplied by the U.S. Environmental Protection Agency (EPA), served as a positive control. A model carbon black particulate sample (M120) was selected for the negative PM control.

To demonstrate the association between PM₁₀ transition metal content with oxidative potential, we performed metal chelation experiments. PM samples (50 μg/mL) in the composite 200-μM antioxidant RTLFL solution were incubated for 4 hr (37°C) in the presence of diethylenetriamine-pentaacetic acid (DTPA), a strong copper [Cu(II)] and iron [Fe(III)] chelator (Buettner and Jurkiewicz 1996). Experiments were performed on pooled Monday–Friday PM suspensions and separately on Saturday and Sunday samples because of the limited sample availability. Antioxidant concentrations were determined after incubations.

Determination of total and reduced bioavailable Fe pool. The total soluble ferrous ion pool in PM suspensions was determined using the chromogenic chelator bathophenanthroline disulfonate (BPS). BPS binds Fe(II) to form a complex that absorbs strongly at a wavelength of 535 nm, permitting the quantification of the soluble Fe(II) (Nilsson et al. 2002). The total soluble Fe pool concentration was quantified by incubating PM suspensions (100 μg/mL) at room temperature for 30 min in the presence of ascorbate (AA; 10 mM) and BPS (50 mM); AA reduces Fe(III) to Fe(II), allowing formation of Fe(II)–BPS complex, which was then measured. A standard curve (0–50 μM) of ferrous ammonium sulfate was used to determine sample Fe concentrations. Fe(II) concentrations were quantified similarly to total Fe; however, AA was not included in the incubation solution. Fe(III) was determined by subtracting the measured Fe(II) from total soluble Fe concentrations.

Total metal screening. EMC Environmental Ltd. (Cumbria, UK) measured total metal concentrations in the PM. Samples were digested

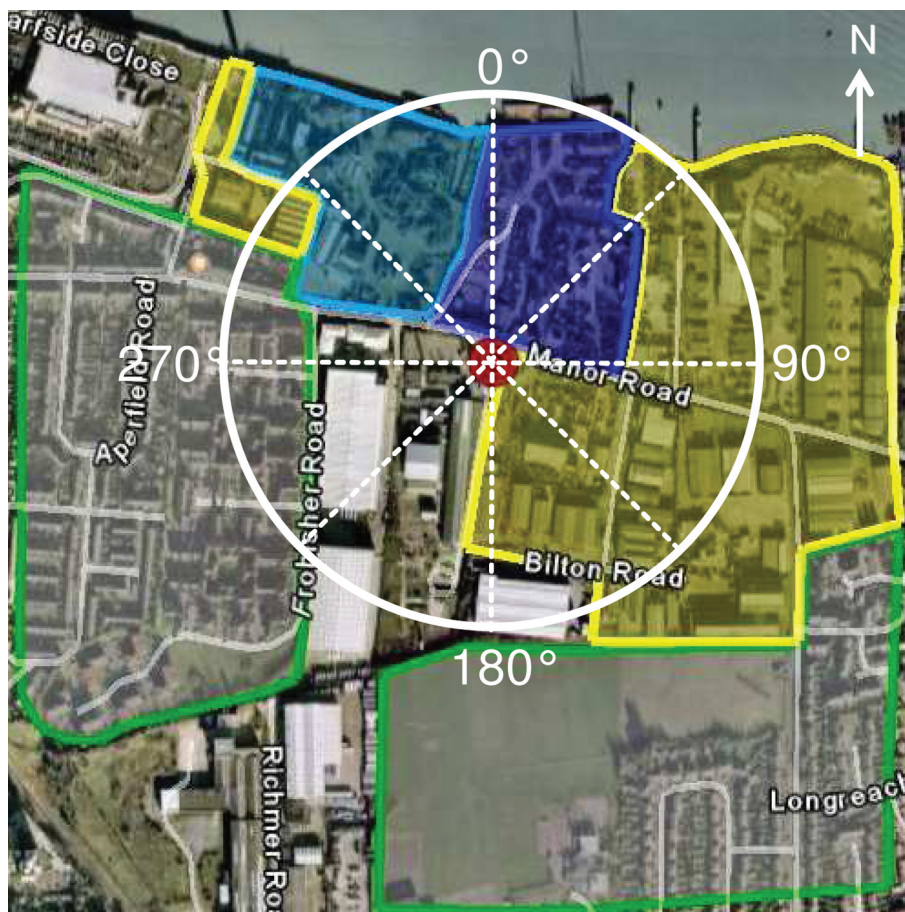


Figure 1. Aerial view of the WTS (red marker) located in Erith, London Borough of Bexley. The blue, yellow, and green shaded areas indicate the WTS, other industrial grounds, and residential communities, respectively. Radius of the circle corresponds to 300 m.

in nitric acid/peroxide and analyzed by inductively coupled plasma mass spectrometry (ICP-MS) by an EMC in-house procedure based on U.S. EPA Compendium Method IO 3.1 and IO 3.5.

Semicontinuous measurements. TEOM PM₁₀, nitrogen oxides (NO_x), and meteorologic [wind speed, wind direction (WD), temperature] measurements were made with 15-min resolution at the Bexley 4 sampling site. NO_x was measured with a Monitor Labs (Englewood, CO, USA) 9841B analyzer, and concentrations were scaled using test results from a nitric oxide calibration gas and a zero air source. Both the NO_x and TEOM instruments were subject to United Kingdom Accreditation Service–accredited biannual audits to ensure traceability to national metrology standards. TEOM PM₁₀ and NO_x measurements were also undertaken to the same standards at eight additional LAQN background and suburban sites: Bexley 1, Bexley 2, Greenwich 4, Hounslow 2, North Kensington 1, Richmond 2, Thurrock 1, and Tower Hamlets 1 (LAQN 2009).

PM₁₀ source apportionment. PM₁₀ concentrations were fractionated into source components over the campaign period: the total regional PM₁₀ background concentration and that local to the Bexley 4 sampling site. This total PM₁₀ background was further subdivided into background primary and background natural and secondary fractions using NO_x measurements also collected at these background/suburban sites. Background PM₁₀ associated with background NO_x categorized the primary contribution. The remaining background PM₁₀, not related to NO_x, comprised the natural and secondary PM₁₀ fraction. Further details regarding the methodology employed have been described previously (Fuller and Green 2006; Fuller et al. 2002). Total background PM₁₀ estimates were then used to quantify the PM₁₀ fraction measured at Bexley 4 related to local activities. Unfortunately, NO_x measurements at Bexley 4 were not available during the campaign to further apportion local PM₁₀. As a result, local PM₁₀ concentrations at the sampling site represent PM₁₀-associated primary (local tailpipe emissions, vehicular-induced particulate resuspension, and tire and brake wear) and nonprimary (industrial activity) sources.

Statistical analysis. All statistical analysis was performed using SPSS statistical program for Windows, version 15 (SPSS Inc., Chicago, IL, USA). Data are presented as the mean ± 1 SD. Data variability within groups of equal sample sizes was quantified using one-way ANOVA post hoc multiple testing with the Student Newman–Kuels test. Pearson correlations were used to assess the strength of correlations, which were considered significant for *p*-values < 0.05.

Results

PM source apportionment. The local PM₁₀ fraction exhibited a clear diurnal trend from Monday to Saturday, achieving maximum concentrations during working hours. Local PM₁₀ concentrations 15-min averaged on Monday–Friday, Saturday, and Sunday were 22.5, 18.2, and 4.8 μg/m³, respectively (Figure 2A). Locally generated PM₁₀ was highly sensitive to WD: Wind originating with a bearing of 280–150°, as measured at the site, was exposed predominantly to emissions from industrial facilities (mainly the WTS) and the roadway (Figure 2B). During periods of industrial (WTS) emission exposure, the local PM₁₀ fraction increased significantly (35.7 ± 9.4 μg/m³ average) compared with residential emission exposure (9.4 ± 5.2 μg/m³ average).

PM oxidative potential. We observed significantly greater PM oxidative potential on weekdays per unit mass, with a decline in PM reactivity on Saturday and the lowest activity observed in the Sunday sample (Figure 3). Although PM oxidative potential was highest on weekdays, considerable variation existed within Monday–Friday samples. AA losses ranged from 71 to 89%, and glutathione (GSH) losses ranged from 1 to 26%.

Metal analysis. Filters were assessed for the following metal concentrations, which were then standardized by the PM mass collected on each filter: aluminum (Al), arsenic (As), cadmium (Cd), Cu, Fe, lead (Pb), magnesium (Mg), platinum (Pt), tungsten (W), and zinc (Zn) (Table 1). Of these, Pt, W, and As were below the limit of detection. Cd and Cu were present at very low levels; all quantities were near the instrument detection limits (< 0.05 μg per microgram PM) after subtraction of background filter concentration and thus were considered unsuitable for inclusion in the correlation analysis.

Fe and Pb concentrations followed the pattern of industrial activity: higher concentrations on Monday–Friday, intermediate concentrations on Saturday, and further decreases on Sunday. Similar to antioxidant concentrations, significant interweekday variability was observed for each of these metal species.

Bioavailable Fe. Metal species associated with PM exist in various forms, each with differing abilities to drive reactions. In the lung, as it is likely that only bioavailable (soluble and surface-associated) metals govern PM oxidative potential, it was of interest to quantify the bioavailable fraction of Fe. Because of the limited

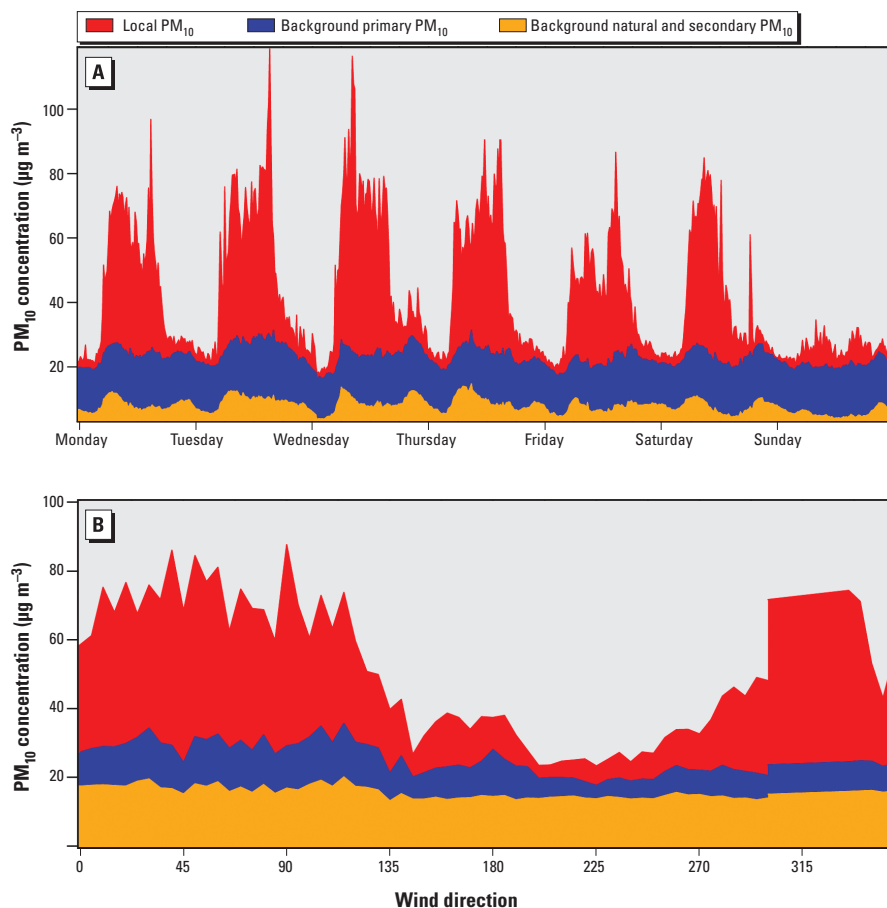


Figure 2. Source-apportioned PM₁₀ fractions 15 min for 5 June–30 August 2002 averaged by (A) day of the week and (B) 5° WD bins at the sampling site.

availability of sample material, this analysis was possible only on four (Monday, Wednesday, Thursday, and Saturday) of the seven filter samples (Figure 4). AA loss from the RTLF was partially explained by its oxidation during the reduction of Fe(III) to Fe(II) (Davies 1992; Miller and Aust 1989). Thus, samples with high Fe(II) metal content were expected to be the most reactive; measured weekday samples were more active compared with the Saturday samples across the sampling period.

Metal chelation analysis. Transition metal-induced PM oxidative potential was confirmed by metal chelation experiments. In all cases, PM₁₀ incubation with an excess of DTPA completely blocked PM-dependent AA and GSH oxidation (Table 2). The relationship between the extent of antioxidant depletion and the concentration of metals on the PM filters was examined using the Pearson correlation. A significant association was apparent between the extent of AA and GSH from the composite antioxidant solution after incubation with model and ambient PM ($r = 0.78$, $p = 0.039$, $n = 7$). Moreover, ICP-MS analysis revealed significant associations between the degree of AA loss and total Pb ($r = 0.88$, $p = 0.009$) and Fe ($r = 0.80$, $p = 0.032$) metal concentrations. In contrast, the loss of GSH was not related to Fe but rather to Al ($r = 0.76$, $p = 0.048$) and Pb ($r = 0.89$, $p = 0.008$) concentrations. Notably,

the association between the antioxidant loss and Pb reflected an underlying association between this trace metal with Fe ($r = 0.87$, $p = 0.011$) and Al ($r = 0.84$, $p = 0.019$).

WD and correlation analysis. Weekday WTS emissions all exhibited an equivalent diurnal profile and maintained an approximately constant magnitude. Thus, the resulting PM oxidative potential was not expected to fluctuate from Monday to Friday. However, the observed variability in PM oxidative potential (comprising antioxidant depletion, the bioavailable Fe pool, and total metal concentrations measurements) suggested that regardless of proximity, the sampling site was not continually exposed to WTS emissions. This was likely due to oscillations in WD. As a result, exposure scenarios were defined for each day of the week during the sampling period to account for the percentage of time that the sampled PM₁₀ was influenced by fugitive WTS emissions. Fifteen-minute averaged WD measurements were considered for each individual day included in the bulk filter exposures. WD measurements between 280° and 150° indicated that sampled PM₁₀ was influenced by industrial sources, whereas remaining bearings were classified as residential-derived PM₁₀ (Figure 5). This WD grouping method results in three cases, each presenting different emission scenarios (Table 3): *a*) Monday–Wednesday, weekday

filters when the sampled emissions were primarily emitted from the WTS; *b*) Saturday, a weekend sample influenced predominantly by the local WTS; and *c*) Sunday, a weekend sample that represented a filter primarily exposed to residential emissions. The remaining sampling days served as mixed residential and WTS emission sampling cases.

The days of the week predominantly influenced by WTS emissions corresponded to a greater percentage loss of AA ($r = 0.80$, $p = 0.030$) and GSH ($r = 0.76$, $p = 0.046$) concentrations relative to 4-hr particle-free controls (Figure 6). Days of the week (Monday–Wednesday) with bulk filter exposures that experienced the greatest influence of WTS emissions corresponded with the highest levels of antioxidant loss. Locally generated PM₁₀ concentrations were positively associated with the frequency with which WTS emissions were sampled during each bulk filter exposure.

When considering only days when the WD carried emissions predominantly originating from the WTS area to the sampling site, the percentage of antioxidant depletion was higher on weekdays (Monday–Wednesday) than on Saturday. Weekday local PM₁₀ fraction concentrations during working hours remained approximately constant. Moreover, weekday levels were not statistically different from concentrations measured on Saturdays. However, PM metal composition did vary significantly between weekday and Saturday samples. The lower Saturday metal concentrations were likely responsible for the decreased PM oxidative potential. Comparison of Saturday versus Sunday PM (weekend periods sampling industrial and residential emissions,

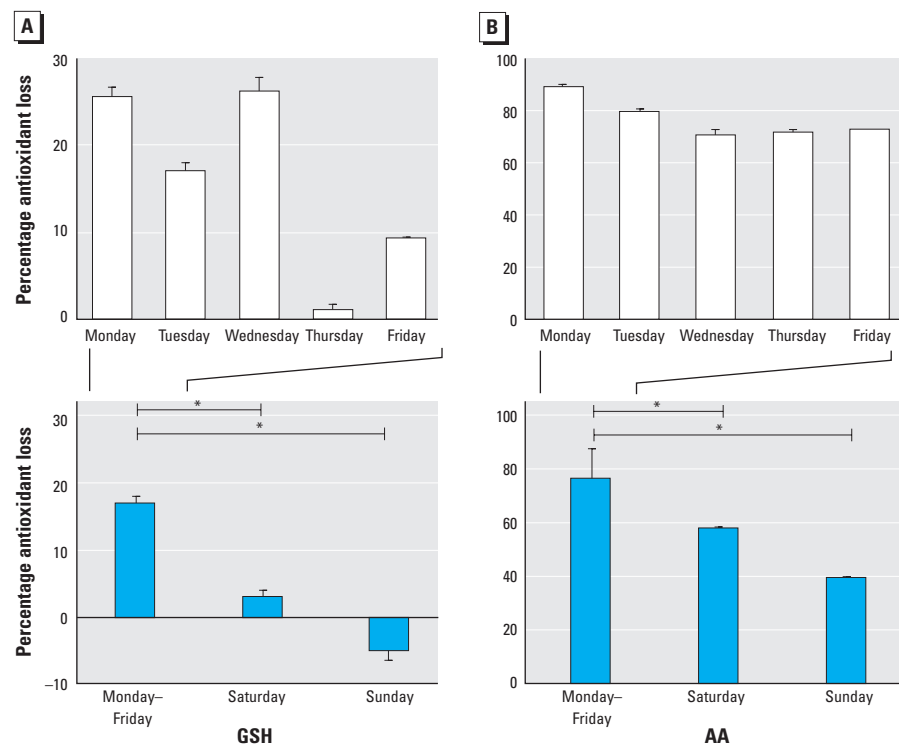


Figure 3. Percentage loss of GSH (A) and AA (B) relative to the 4-hr particle-free control concentration in antioxidant solutions incubated for 4 hr with PM (50 µg/mL) extracted from Teflon filters collected daily (Monday–Sunday). All filter extracts were run in triplicate. Values are mean ± SD.

* $p < 0.05$ for difference between filter reactivity.

Table 1. Individual filter metal concentrations. All metal concentrations were standardized by the total PM mass collected on the filter (mg PM₁₀).

Filter	Fe	Pb	Al	Mg	Zn
Monday	25.9	2.32	5.22	2.35	0.94
Tuesday	10.5	1.35	2.10	1.15	0.65
Wednesday	13.9	1.59	3.61	1.91	0.94
Thursday	16.2	1.02	2.29	0.77	0.00
Friday	5.04	1.20	1.73	1.11	0.38
Saturday	9.14	1.14	1.91	0.83	0.30
Sunday	0.00	0.29	1.91	1.65	0.64

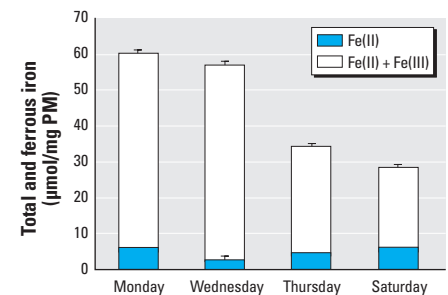


Figure 4. Mean total and ferrous bioavailable Fe concentrations in PM₁₀ with the associated SD of sample replicates ($n = 3$).

respectively) revealed that antioxidant losses were higher on Saturday, which was potentially attributed to a change in WD and a decrease in locally produced PM₁₀.

Discussion

Elevated PM concentrations have been reported in proximity to WTS (Fuller and Baker 2008; Maciejczyk et al. 2004; Restrepo et al. 2004), but no study has yet examined whether there is a basis for believing PM from these industrial emissions may have enhanced toxicity, based on their chemical composition. Questions have also been voiced by concerned residents in these areas regarding the environmental disamenities resulting from the poor air quality including degraded health and limited economic growth (NEJAC 2000). To investigate the raised health concerns and to examine whether there was any basis for believing PM from such facilities might display enhanced toxicity, we examined both the composition and oxidative properties of PM generated by WTS activity. Incorporation of PM oxidative potential into a receptor model source apportionment analysis is a novel aspect of this study.

A weekly pattern of local PM₁₀ concentrations was observed at the sampling site: Elevated concentrations were found on weekdays and Saturdays, with much lower concentrations on Sundays. The PM₁₀ mass concentration difference between weekday and Sunday samples was expected, because the WTS was closed on Sundays. This general trend also extended to oxidative potential measurements. Intercomparison of weekday oxidative potential levels revealed variation regardless of the continual WTS operation during these days. The extent that industrial emissions influenced sampled PM₁₀ concentration, as predicted by WD, was used to explain this weekday variation. Moreover, elevated PM oxidative potential was established when WTS emissions predominately contributed to PM sampled at this site. Additionally, the measured PM oxidative potential was likely attributable to the compositional profile of the local PM₁₀ fractions, given its high sensitivity to WD.

The oxidative potential of PM derived from WTS was compared with the PM total metal and bioavailable Fe content. Correlations between total transition metals and antioxidant depletion produced significant associations between metal species and GSH (Al, Pb) and AA (Fe, Pb) depletion. Moreover, the concentration of Pb was strongly associated with both Fe and Al, suggesting a common source(s). Total Fe and Pb exhibited a significant difference between weekday, Saturday, and Sunday concentrations following the pattern of locally produced PM₁₀ at the sampling site.

Cu was not identified as a predictor of PM oxidative potential, unlike results from

previous studies (Künzli et al. 2006; Mudway et al. 2004). Differences in the traffic profile are likely accountable for this discrepancy: Cu is a marker of light-duty vehicle brake wear, whereas barium and antimony are characteristic of brake wear from heavy-duty vehicles (Sternbeck et al. 2002). The vehicle fleet servicing the WTS comprised predominantly the latter type, unlike the traffic profiles of the previously mentioned studies.

Bioavailable Fe (BPS-mobilized Fe, as opposed to total Fe), is the key determinant of the PM-associated Fe pool that would be available *in vivo* to drive damaging oxidations (Aust et al. 2002; Duggan 2007). These BPS measurements of soluble Fe also provide information regarding oxidation state. This is useful, as PM oxidative potential is partially explained by the reduction of Fe(III) to Fe(II), and therefore samples with high Fe(III)

Table 2. Details of DTPA metal chelation incubations with PM₁₀ samples.

Filter	Percent AA loss		Percent GSH loss	
	(-DTPA)	(+DTPA)	(-DTPA)	(+DTPA)
Monday–Friday	61.3 ± 1.25	1.56 ± 1.56	19.6 ± 6.20	ND
Saturday	50.9 ± 1.26	0.38 ± 1.40	1.10 ± 5.43	ND
Sunday	44.3 ± 2.33	3.08 ± 3.61	12.7 ± 1.64	ND

ND, not detected. (-DTPA) and (+DTPA) represent incubations without and with DTPA, respectively. The percentage of antioxidant losses are expressed relative to the zero time point particle-free control, as incubations with DTPA eliminated the background auto-oxidation seen in the particle-free controls.

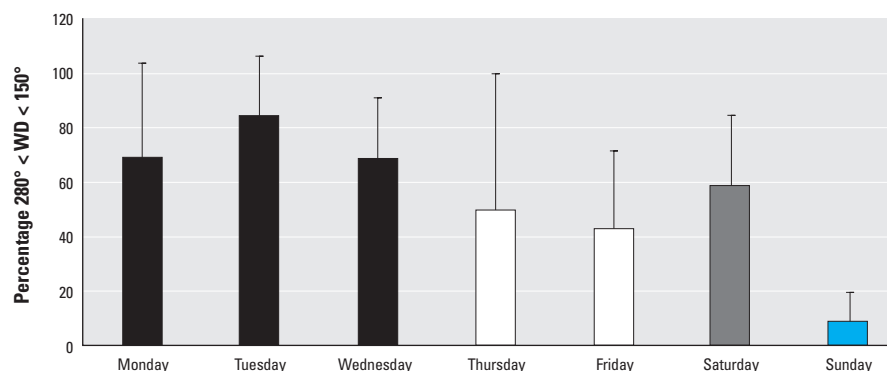


Figure 5. Percentage (mean ± SD) of daily averaged 15-min resolution WD measurements with a bearing of 280–150°, corresponding to the frequency at which the sampled air originated predominantly from over the industrial facilities. Bar color indicates the four emission scenarios during the sampling campaign: black, weekday samples when emissions were primarily emitted from the WTS; gray, weekend sample influenced predominantly by the local WTS; blue, weekend sample exposed primarily to residential emissions; white, remaining samples representing mixed residential and WTS emission cases.

Table 3. Summary of PM₁₀ toxicity analysis grouped by WD.^a

Case	Filter	Source	% AA loss	% GSH loss	PM ₁₀ (mg)	Fe	Al	Pb
1	Mon–Wed	Industrial	79.8 ± 1.2	23.0 ± 2.2	2.71 ± 0.75	16.80 ± 6.61	3.65 ± 1.27	1.50 ± 0.51
2	Sat	Industrial	57.6 ± 1.2	3.0 ± 2.4	3.14	9.14	1.91	1.14
3	Sun	Residential	39.5 ± 0.6	-5.0 ± 1.1	1.06	0.00	1.91	0.29

^aMean weekday concentrations are presented ± 1 SD. Metal concentrations are standardized to the PM mass on the filter [μg (mg/PM₁₀)].

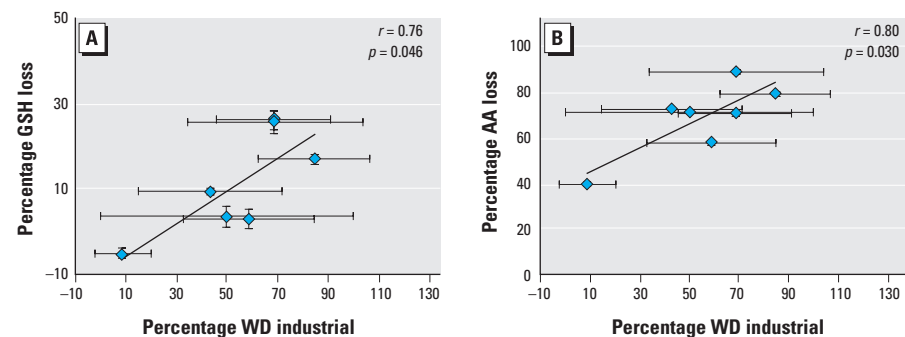


Figure 6. Correlation between the percentage of GSH (A) and AA (B) relative to the 4-hr particle-free control concentration and the frequency sampled air was transported over industrial facilities (percent WD industrial given by a WD with a bearing of 280°–150°) (n = 7). Values shown are mean ± SD.

metal detail are expected to be most reactive. Low-molecular-weight antioxidants found in the RTLF model and lung are able to rapidly reduce Fe(III) complexes. Upon ferric ion reduction, AA is simultaneously oxidized. In addition, the superoxide formed in these reactions will further drive AA and GSH oxidation. Moreover, selection of BPS as a chelator has biological relevance: The stability constant of BPS for Fe(III) is 1×10^{22} M (Nilsson et al. 2002), which is lower than that for transferrin (1×10^{28} M) and albumin (Halliwell and Gutteridge 1999; Halliwell et al. 1999), providing a measure of the Fe pool likely to be associated with low molecular weight ligands *in vivo* and hence its catalytic activity.

Bioavailable Fe content was considered for a subset of exposed samples. Weekday (Monday and Wednesday) samples were found to have the highest ferric ion content. This trend of elevated weekday Fe(III) concentrations also corresponded with the highest PM oxidative potential measurements reflective of WTS contributions to the sampling site rather than residential emissions. As the contribution of WTS emissions to sampling site declined, so too did the PM₁₀ bioavailable iron pool. Because only four samples were available for this type of analysis, it was not appropriate to attempt soluble ferric ion content correlation analysis with WTS emission exposure ($280^\circ < \text{WD} < 150^\circ$) and antioxidant depletion.

Organic PM content, including polycyclic aromatic hydrocarbons, quinones, and endotoxin, were not measured in the present study, although they can also trigger the generation of reactive oxygen species, once inhaled, through metabolism and the induction of inflammation (Ayres et al. 2008). Of these components, only particulate quinone content would have influenced the measured PM oxidative potential in the acellular RTLF model because of their capacity to redox cycle (Squadrito et al. 2001). Thus, the measured PM oxidative potential in the RTLF model is restricted to the intrinsic redox active components, catalytic metals, and quinones and should not be viewed as reflecting the total activity of the sample through its interaction with the complete biological matrix. However, the metal chelator experiments demonstrated that quinone-driven oxidative activity of WTS-derived PM was low (Table 2). No significant antioxidant depletion was measured when PM₁₀ samples were incubated with DTPA. This demonstrates that PM catalytic metal content explained much of the intrinsic capacity of these samples to catalyze damaging oxidation reactions.

Conclusion

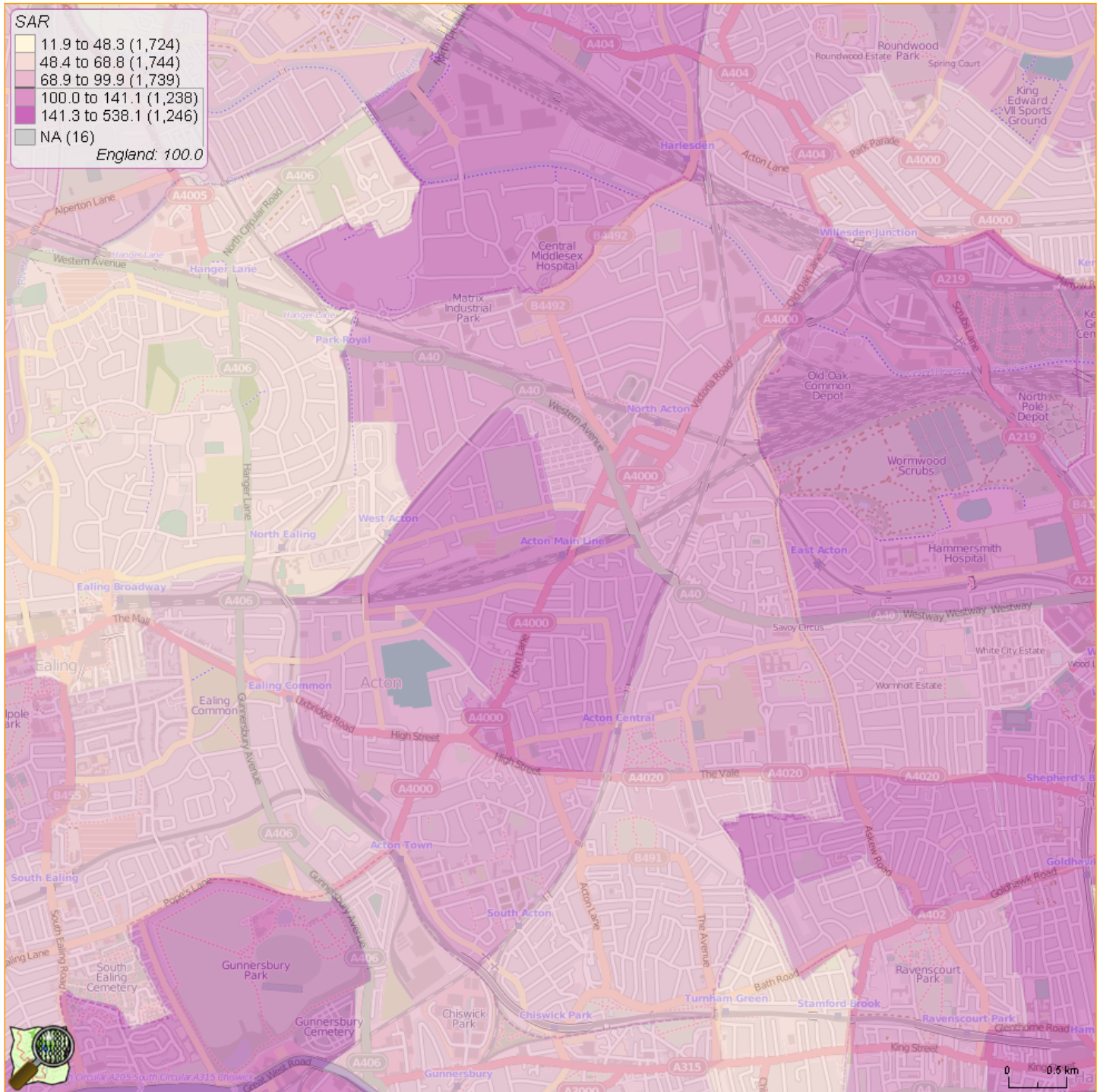
PM₁₀ exceedances at the sampling site were the result of WTS emissions linked to site activity. Detailed PM chemical characterization indicated that these high PM₁₀ mass concentrations contained elevated transition metal levels. Concentrations of total Fe, Al, and Pb were especially high; WTS soluble Fe emissions existed predominantly in the form of Fe(III). Assessment of the WTS PM oxidative potential revealed that it was associated with increased transition metal concentrations. Thus, PM₁₀ generated by WTSs may be a potential health risk to neighboring communities. The resident population is exposed to a high PM mass concentration on a recurrent basis, and particulate emitted by these industrial emissions may have a greater capacity to drive damaging oxidation reactions in the lung compared with other PM sources in urban environments.

REFERENCES

- Aust SD, Ball JC, Hu A, Lighty JS, Smith KR, Straccia AM, et al. 2002. Particle characteristics responsible for effects on human lung epithelial cells. *Res Rep Health Eff Inst* 110:1–65.
- Ayres JG, Borm P, Cassee FR, Castranova V, Donaldson K, Ghio A, et al. 2008. Evaluating the toxicity of airborne particulate matter and nanoparticles by measuring oxidative stress potential—a workshop report and consensus statement. *Inhal Toxicol* 20(1):75–99.
- Buettner GR, Jurkiewicz BA. 1996. Catalytic metals, ascorbate and free radicals: combinations to avoid. *Radiat Res* 145(5):532–541.
- Davies MB. 1992. Reactions of L-ascorbic acid with transition metal complexes. *Polyhedron* 11(3):285–321.
- Duggan ST. 2007. Oxidative activity as an indicator for the health effects of environmental particulate matter (PM) [PhD thesis]. London, UK:King's College London.
- Fuller GW, Baker T. 2001. The Manor Road Air Pollution Study 2001. London:King's College London.
- Fuller GW, Baker TD. 2008. PM₁₀ Source Apportionment at Bexley 4, Manor Road, Erith. London:King's College London.
- Fuller GW, Carslaw DC, Lodge HW. 2002. An empirical approach for the prediction of daily mean PM₁₀ concentrations. *Atmos Environ* 36(9):1431–1441.
- Fuller GW, Green D. 2006. Evidence for increasing concentrations of primary PM₁₀ in London. *Atmos Environ* 40(32):6134–6145.
- Gil Y, Kellerman A. 1993. A multicriteria model for the location of solid waste transfer stations: the case of Ashdod, Israel. *GeoJournal* 29(4):377–384.
- Halliwell B, Gutteridge JMC. 1999. *Free Radicals in Biology and Medicine*. Oxford, UK:Oxford University Press.
- Halliwell B, Zhao K, Whiteman M. 1999. Nitric oxide and peroxynitrite. The ugly, the uglier and the not so good: a personal view of recent controversies. *Free Radic Res* 31(6):651–669.
- Higgs G, Langford M. 2009. GIScience, environmental justice, and estimating populations at risk: the case of landfills in Wales. *Appl Geogr* 29(1):63–76.
- Jackson K. 1995. *The Encyclopedia of New York City*. New Haven, CT:Yale University Press.
- Künzli N, Mudway IS, Götschi T, Shi T, Kelly FJ, Cook S, et al. 2006. Comparison of oxidative properties, light absorbance, and total and elemental mass concentration of ambient PM_{2.5} collected at 20 European sites. *Environ Health Perspect* 114:684–690.
- LAQN (London Air Quality Network). 2009. London Air Quality Network. Available: <http://www.londonair.org.uk> [accessed 31 March 2009].
- Li N, Kim S, Wang M, Froines J, Sioutas C, Nel A. 2002. Use of a stratified oxidative stress model to study the biological effects of ambient concentrated and diesel exhaust particulate matter. *Inhal Toxicol* 14(5):459–486.
- Maantay J. 2007. Asthma and air pollution in the Bronx: methodological and data considerations in using GIS for environmental justice and health research. *Health Place* 13(1):32–56.
- Maciejczyk PB, Offenberg JH, Clemente J, Blaustein M, Thurston GD, Chi Chen L. 2004. Ambient pollutant concentrations measured by a mobile laboratory in South Bronx, NY. *Atmos Environ* 38(31):5283–5294.
- Miller DM, Aust SD. 1989. Studies of ascorbate-dependent, iron-catalyzed lipid peroxidation. *Arch Biochem Biophys* 271(1):113–119.
- Mudway IS, Stenfors N, Duggan ST, Roxborough H, Zielinski H, Marklund SL, et al. 2004. An *in vitro* and *in vivo* investigation of the effects of diesel exhaust on human airway lining fluid antioxidants. *Arch Biochem Biophys* 423(1):200–212.
- NEJAC (National Environmental Justice Advisory Council). 2000. *A Regulatory Strategy for Siting and Operating Waste Transfer Stations*. New York:National Environmental Justice Advisory Council.
- Nel AE, Diaz-Sanchez D, Li N. 2001. The role of particulate pollutants in pulmonary inflammation and asthma: evidence for the involvement of organic chemicals and oxidative stress. *Curr Opin Pulm Med* 7(1):20–26.
- New York City Department of Health. 1999. *Asthma Facts*. New York:New York City Department of Health.
- Nilsson UA, Bassen M, Sävman K, Kjellmer I. 2002. A simple and rapid method for the determination of “free” iron in biological fluids. *Free Radic Res* 36(6):677–684.
- Pourazar J, Mudway IS, Samet JM, Helleday R, Blomberg A, Wilson SJ, et al. 2005. Diesel exhaust activates redox-sensitive transcription factors and kinases in human airways. *Am J Physiol Lung Cell Mol Physiol* 289(5):L724–L730.
- Prahalad AK, Soukup JM, Inmon J, Willis R, Ghio AJ, Becker S, et al. 1999. Ambient air particles: effects on cellular oxidant radical generation in relation to particulate elemental chemistry. *Toxicol Appl Pharmacol* 158(2):81–91.
- Restrepo C, Zimmerman R, Thurston G, Clemente J, Gorczynski J, Zhong M, et al. 2004. A comparison of ground-level air quality data with New York State Department of Environmental Conservation monitoring stations data in South Bronx, New York. *Atmos Environ* 38(31):5295–5304.
- Romieu I, Meneses F, Sienra-Monge JJJ, Huerta J, Velasco SR, White MC, et al. 1995. Effects of urban air pollutants on emergency visits for childhood asthma in Mexico City. *Am J Epidemiol* 141(6):546–553.
- Schwartz J, Slater D, Larson TV, Pierson WE, Koenig JD. 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. *Am Rev Respir Dis* 147(4):826–831.
- Squadrito GL, Cueto R, Dellinger B, Pryor WA. 2001. Quinoid redox cycling as a mechanism for sustained free radical generation by inhaled airborne particulate matter. *Free Radic Biol Med* 31(9):1132–1138.
- Sternbeck J, Sjödin A, Andréasson K. 2002. Metal emissions from road traffic and the influence of resuspension: results from two tunnel studies. *Atmos Environ* 36(30):4735–4744.
- Stohs SJ, Bagchi D. 1995. Oxidative mechanisms in the toxicity of metal ions. *Free Radic Biol Med* 18(2):321–336.
- Studnicka M, Hackl E, Pischinger J, Fangmeyer C, Haschke N, Kühn J, et al. 1997. Traffic-related NO₂ and the prevalence of asthma and respiratory symptoms in seven year olds. *Eur Respir J* 10(10):2275–2278.
- Sunyer J, Spix C, Quenel P, Ponce-de-Leon A, Ponka A, Barumandzadeh T, et al. 1997. Urban air pollution and emergency admissions for asthma in four European cities: the APHEA project. *Thorax* 52(9):760–765.
- Thorne PS. 2000. Inhalation toxicology models of endotoxin- and bioaerosol-induced inflammation. *Toxicology* 152(1–3):13–23.
- Walker G, Mitchell G, Fairburn J, Smith G. 2005. Industrial pollution and social deprivation: evidence and complexity in evaluating and responding to environmental inequality. *Local Environ* 10(4):361–377.

Ward (2011) Estimates - see How To... page for more details

Emergency hospital admissions for chronic obstructive pulmonary disease, standardised admission ratio, 2006/7 - 2010/11 - source: PHOs (now part of Public Health England), produced from Hospital Episodes Statistics (HES). Copyright © 2012 The NHS Information Centre for Health and Social Care. All rights reserved.

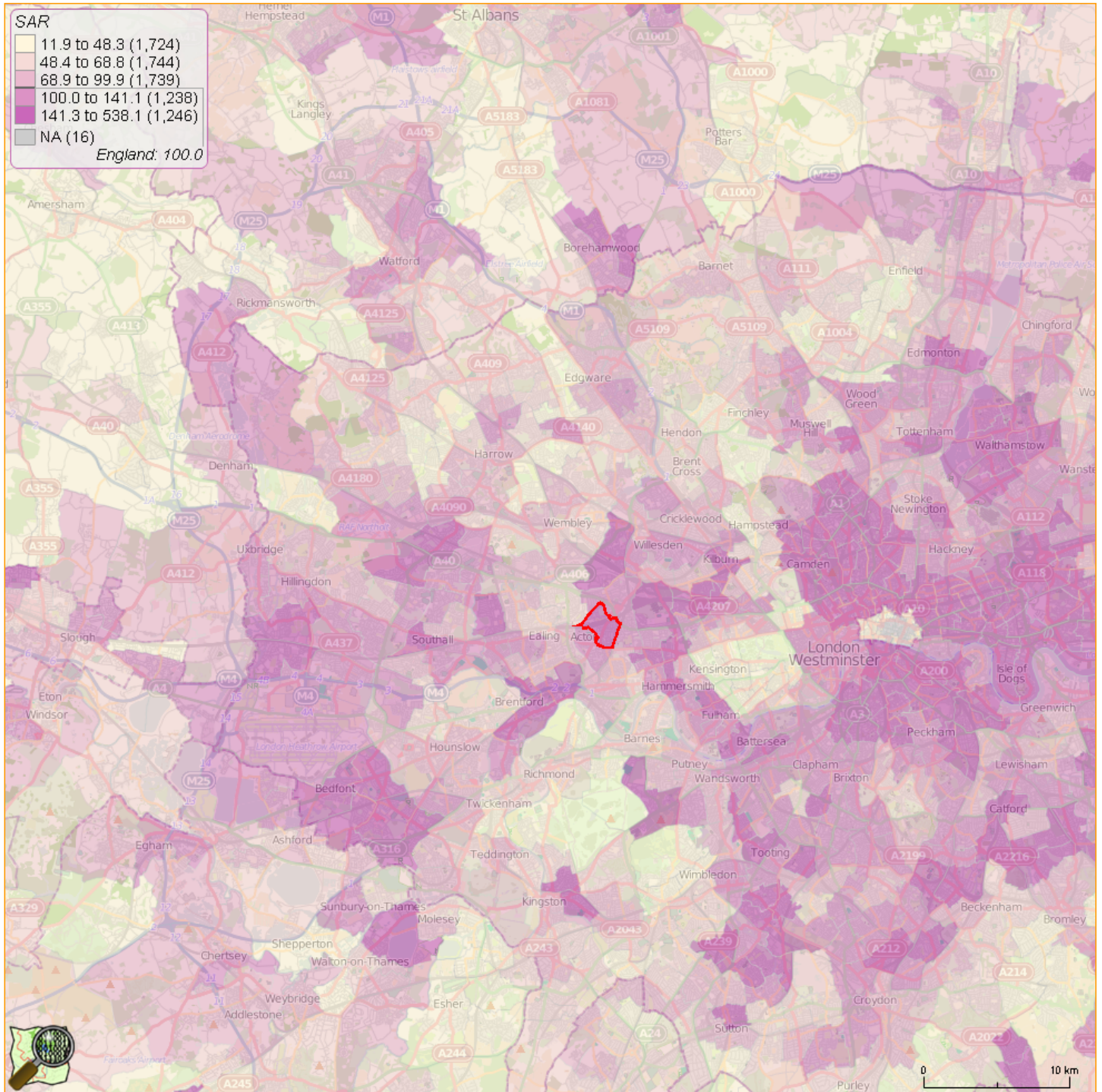


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Emergency Admissions, Chronic Obstructive Pulmonary Disease (COPD), Standardised admission ratio, 2006/7-2010/11. Numerator = Observed COPD admissions; defined by a primary diagnosis (ICD10) code of J40*, J41*, J42*, J43* or J44* Emergency admissions with an admission method of 21 - 28 and patient classification 'ordinary' (1 or 2), which are completed during the time period stated above (episode status = 3, episorder = 1). A patient's area of residence is defined by the Middle Super Output Area Code (MSOA) as supplied with the HES dataset. Denominator = Expected admissions during the time period (Adjusted) England age specific admission rates were calculated from all admission episodes with valid MSOA and age coding. Unadjusted expected admissions within each MSOA are calculated by applying age-specific admission rates for England in 2006/7-2010/11 to each MSOA's population. These have been adjusted to compensate for the effect of missing age codes within the National data; this methodology assumes that the age distribution within the missing data is the same as that at the National level.

Ward (2011) Estimates - see How To... page for more details

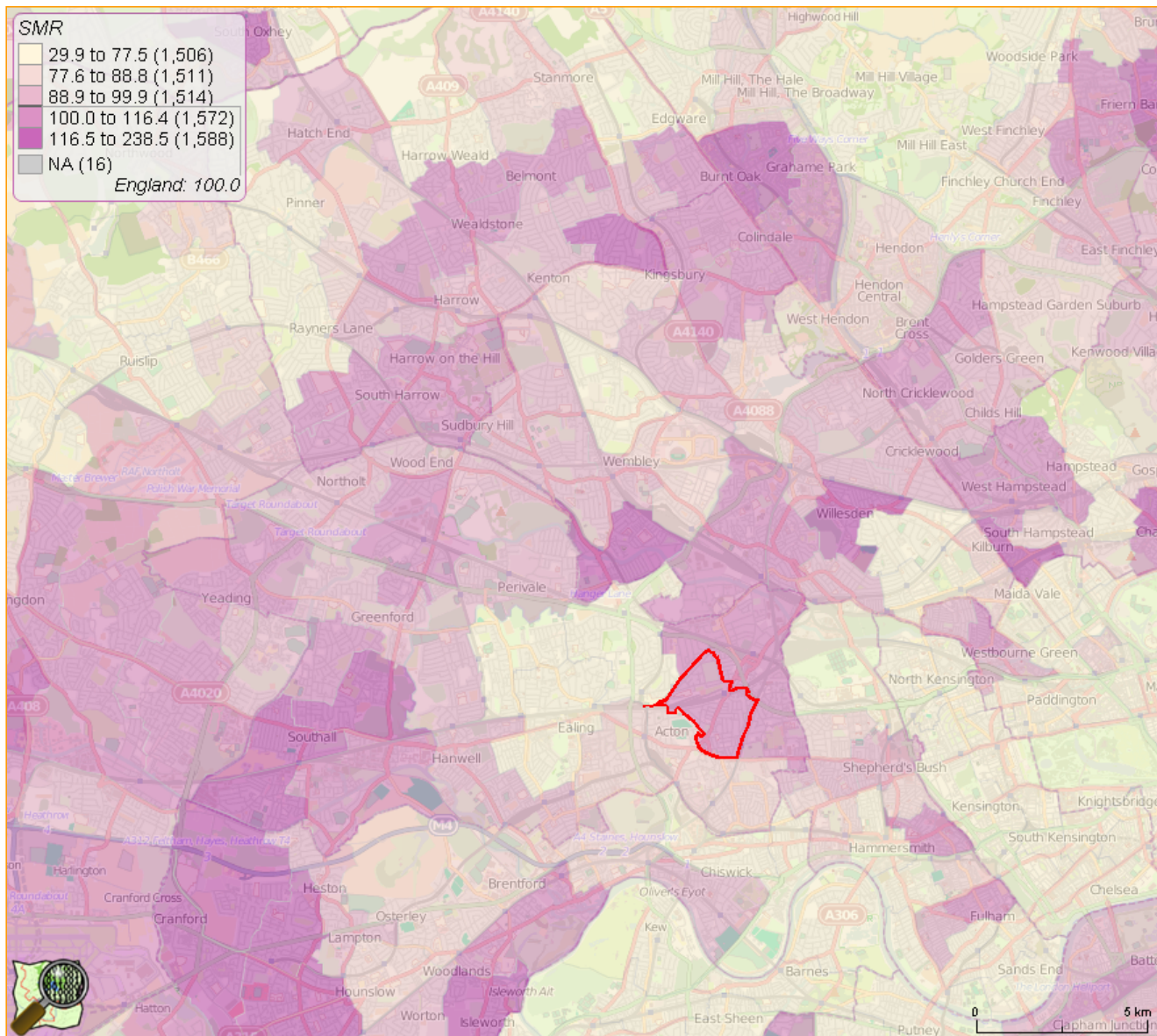
Emergency hospital admissions for chronic obstructive pulmonary disease, standardised admission ratio, 2006/7 - 2010/11 - source: PHOs (now part of Public Health England), produced from Hospital Episodes Statistics (HES). Copyright © 2012 The NHS Information Centre for Health and Social Care. All rights reserved.



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Emergency Admissions, Chronic Obstructive Pulmonary Disease (COPD), Standardised admission ratio, 2006/7-2010/11. Numerator = Observed COPD admissions; defined by a primary diagnosis (ICD10) code of J40*, J41*, J42*, J43* or J44* Emergency admissions with an admission method of 21 - 28 and patient classification 'ordinary' (1 or 2), which are completed during the time period stated above (episode status = 3, episorder = 1). A patient's area of residence is defined by the Middle Super Output Area Code (MSOA) as supplied with the HES dataset. Denominator = Expected admissions during the time period (Adjusted) England age specific admission rates were calculated from all admission episodes with valid MSOA and age coding. Unadjusted expected admissions within each MSOA are calculated by applying age-specific admission rates for England in 2006/7-2010/11 to each MSOA's population. These have been adjusted to compensate for the effect of missing age codes within the National data; this methodology assumes that the age distribution within the missing data is the same as that at the National level.

Ward (2011) Estimates - see How To... page for more details

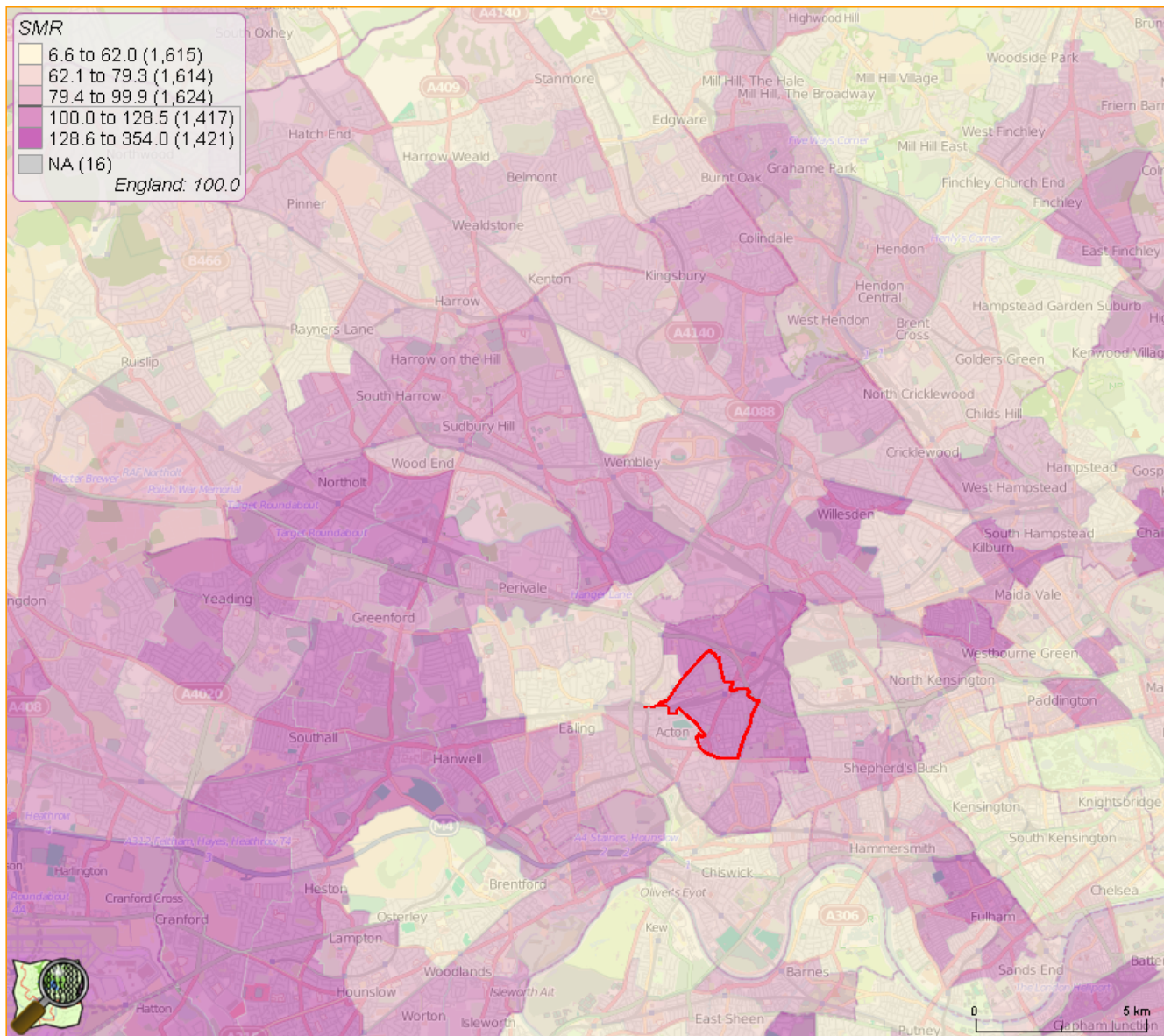


Deaths from coronary heart disease, all ages, standardised mortality ratio, 2006-2010

Source: PHOs (now part of Public Health England), produced from ONS data © Copyright 2011

Standardised Mortality Ratio (SMR), using England as the comparison area. Mortality data by sex and five-year age group were extracted from annual files supplied to the public health observatories (PHOs) (now part of Public Health England) by the Office for National Statistics (ONS). These were deaths registered in the calendar years 2006-10. Deaths were allocated to the deceased's lower layer super output area (LSOA) of usual residence using the August 2011 version of the National Statistics Postcode Directory. Deaths included where underlying cause of death = ICD10 I20-I25. Population data are experimental mid-year population estimates for LSOAs, by sex and single year of age, supplied by ONS to PHOs (now part of Public Health England). Counts by single year of age from 2006-10 were aggregated into five-year age groups. LSOA deaths and population estimates were aggregated to higher geographies using standard geographical lookup tables obtained from ONS Geography. Expected deaths calculated by applying age-specific death rates for England in 2006-10 to each area's population. SMRs calculated by dividing the observed total deaths in the area by the expected deaths and multiplying by 100.

Ward (2011) Estimates - see How To... page for more details

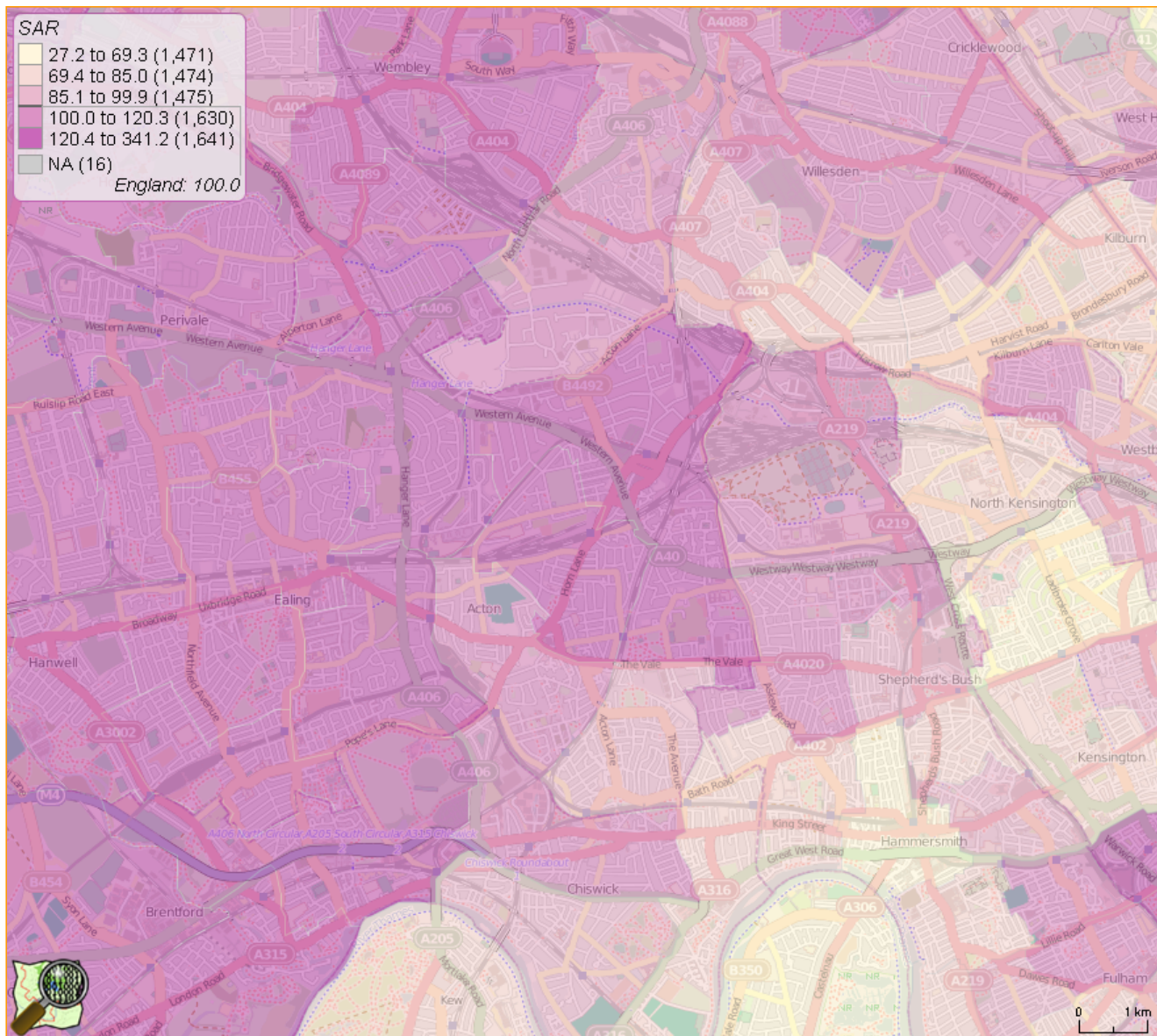


Deaths from coronary heart disease, under 75 years, standardised mortality ratio, 2006-2010

Source: PHOs (now part of Public Health England), produced from ONS data © Copyright 2011

Standardised Mortality Ratio (SMR), using England as the comparison area. Mortality data by sex and five-year age group were extracted from annual files supplied to the public health observatories (PHOs) (now part of Public Health England) by the Office for National Statistics (ONS). These were deaths registered in the calendar years 2006-10. Deaths were allocated to the deceased's lower layer super output area (LSOA) of usual residence using the August 2011 version of the National Statistics Postcode Directory. Deaths included where underlying cause of death = ICD10 I20-I25. Population data are experimental mid-year population estimates for LSOAs, by sex and single year of age, supplied by ONS to PHOs (now part of Public Health England). Counts by single year of age from 2006-10 were aggregated into five-year age groups. LSOA deaths and population estimates were aggregated to higher geographies using standard geographical lookup tables obtained from ONS Geography. Expected deaths calculated by applying age-specific death rates for England in 2006-10 to each area's population. SMRs calculated by dividing the observed total deaths in the area by the expected deaths and multiplying by 100.

Ward (2011) Estimates - see How To... page for more details



Elective hospital admissions for coronary heart disease, standardised admission ratio, 2006/7 - 2010/11

Source: PHOs (now part of Public Health England), produced from Hospital Episodes Statistics (HES). Copyright © 2012 The NHS Information Centre for Health and Social Care. All rights reserved.

Elective Admissions, Coronary Heart Disease, Standardised admission ratio, 2006/7-2010/11. Numerator = Observed Coronary Heart Disease admissions; defined by a primary diagnosis (ICD10) code of I20*, I21*, I22*, I23*, I24* or I25* Elective admissions with an admission method of 11 - 13 and patient classification 'ordinary' (1 or 2), which are completed during the time period stated above (episode status = 3, episode order = 1). A patient's area of residence is defined by the Middle Super Output Area Code (MSOA) as supplied with the HES dataset. Denominator = Expected admissions during the time period (Adjusted) England age specific admission rates were calculated from all admission episodes with valid MSOA and age coding. Unadjusted expected admissions within each MSOA are calculated by applying age-specific admission rates for England in 2006/7-2010/11 to each MSOA's population. These have been adjusted to compensate for the effect of missing age codes within the National data; this methodology assumes that the age distribution within the missing data is the same as that at the National level.

Report - Wards: Acton Central Ward, Ealing

Hospital admissions - specific causes

Hospital Admissions - disease specific causes, numbers, 2006/7 to 2010/11

Indicator	Selection	Local Authorities Lower Tier (Ealing)	Local Authorities Upper Tier (Ealing)	England
Emergency hospital admissions for coronary heart disease	215	5,561	5,561	733,521
Elective hospital admissions for coronary heart disease	154	5,006	5,006	597,899
Emergency hospital admissions for stroke	73	1,729	1,729	343,972
Emergency hospital admissions for myocardial infarction	51	1,458	1,458	318,883
Emergency hospital admissions for COPD	143	2,484	2,484	530,197

Source: PHOs (now part of Public Health England), NHS IC © Copyright 2012

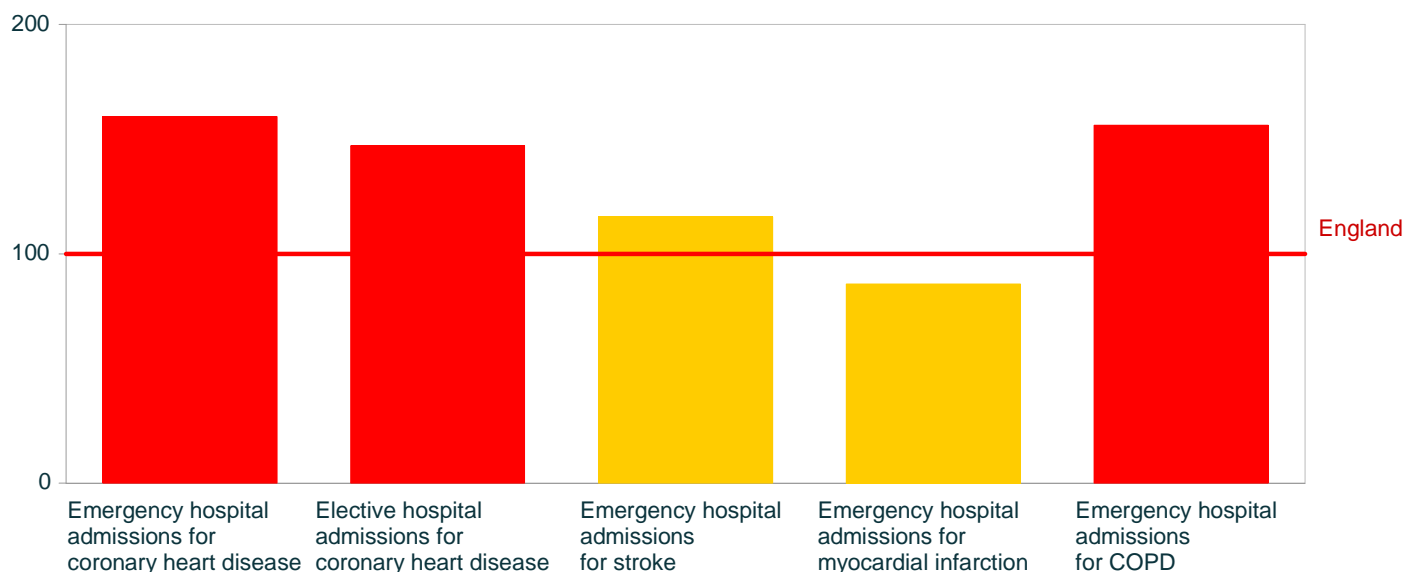
Hospital Admissions - disease specific causes, Standardised Admission Ratios (SARs), 2006/7 to 2010/11

Indicator	Selection	Local Authorities Lower Tier (Ealing)	Local Authorities Upper Tier (Ealing)	England
Emergency hospital admissions for coronary heart disease	159.8	166	166	100
Elective hospital admissions for coronary heart disease	147.1	184.6	184.6	100
Emergency hospital admissions for stroke	116.1	114	114	100
Emergency hospital admissions for myocardial infarction	86.8	100.9	100.9	100
Emergency hospital admissions for COPD	156	107.3	107.3	100

Source: PHOs (now part of Public Health England), NHS IC © Copyright 2012

Hospital admissions - disease specific causes, SARs, 2006/7 to 2010/11, Selection (comparing to England average)

■ Significantly better than England
 ■ Not significantly different
 ■ Significantly worse than England



Source: PHOs (now part of Public Health England), NHS IC © Copyright 2012

Report - Wards: East Acton Ward, Ealing

Hospital admissions - specific causes

Hospital Admissions - disease specific causes, numbers, 2006/7 to 2010/11

Indicator	Selection	Local Authorities Lower Tier (Ealing)	Local Authorities Upper Tier (Ealing)	England
Emergency hospital admissions for coronary heart disease	227	5,561	5,561	733,521
Elective hospital admissions for coronary heart disease	164	5,006	5,006	597,899
Emergency hospital admissions for stroke	76	1,729	1,729	343,972
Emergency hospital admissions for myocardial infarction	69	1,458	1,458	318,883
Emergency hospital admissions for COPD	133	2,484	2,484	530,197

Source: PHOs (now part of Public Health England), NHS IC © Copyright 2012

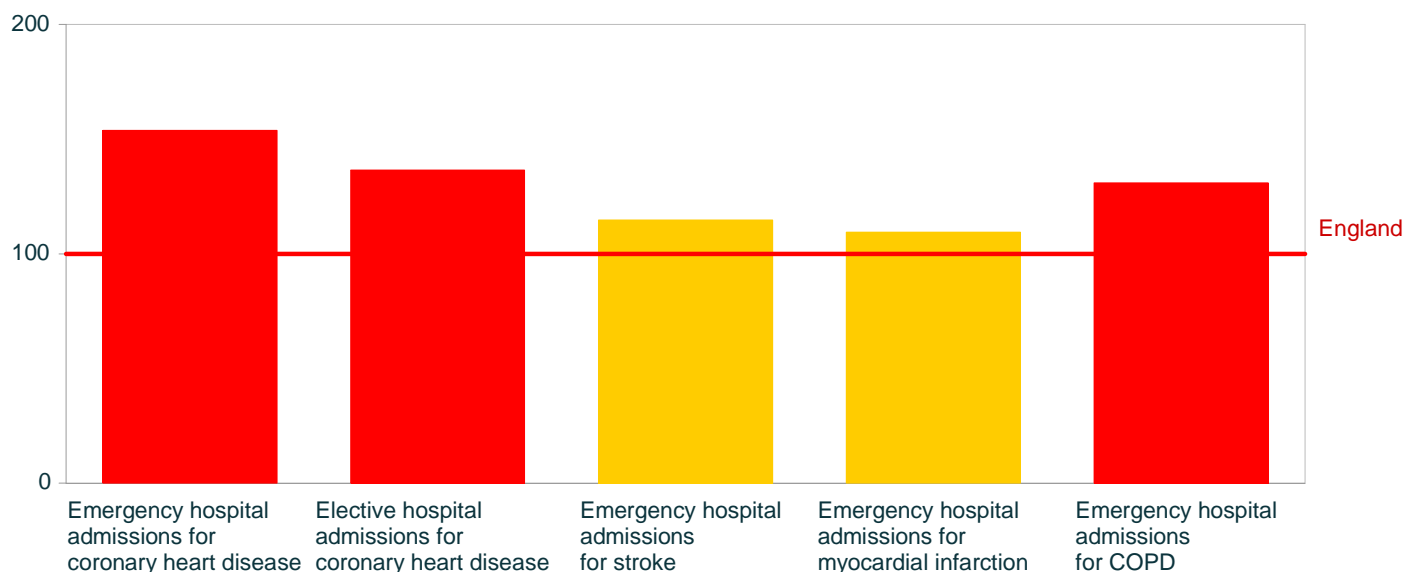
Hospital Admissions - disease specific causes, Standardised Admission Ratios (SARs), 2006/7 to 2010/11

Indicator	Selection	Local Authorities Lower Tier (Ealing)	Local Authorities Upper Tier (Ealing)	England
Emergency hospital admissions for coronary heart disease	153.8	166	166	100
Elective hospital admissions for coronary heart disease	136.5	184.6	184.6	100
Emergency hospital admissions for stroke	114.8	114	114	100
Emergency hospital admissions for myocardial infarction	109.4	100.9	100.9	100
Emergency hospital admissions for COPD	130.9	107.3	107.3	100

Source: PHOs (now part of Public Health England), NHS IC © Copyright 2012

Hospital admissions - disease specific causes, SARs, 2006/7 to 2010/11, Selection (comparing to England average)

■ Significantly better than England
 ■ Not significantly different
 ■ Significantly worse than England



Source: PHOs (now part of Public Health England), NHS IC © Copyright 2012