respiratory tract infections. Understanding the immunological effects of different particle types will help guide public health interventions.

P122 LOCAL SOURCES RATHER THAN INTERACTIONS WITH OXIDISING CO-POLLUTANT GASES DETERMINE THE GEOGRAPHICAL AND SEASONAL VARIATION IN PARTICULATE MATTER OXIDATIVE POTENTIAL

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Background Reactions of ambient particulate matter (PM) with atmospheric radicals and oxidising gases such as nitrogen dioxide (NO₂) and ozone (O₃) may alter their toxicity – often referred to as particle ageing. As concentrations of O₃ dominate in the warmer months, with NO₂ episodes more common in the winter, we investigated whether seasonal differences in the PM oxidative potential (the capacity of inhaled PM to cause damaging oxidation reactions at the air-lung interface) could be attributed to their interaction with these gases.

Methods Daily PM₁₀ and PM_{2.5} filters from 4 sites in Southern England were obtained during summer (21st July – 23rd August) and winter (6th Jan–11th Feb) campaigns in 2012: two sites in London, roadside (Marylebone Road) and urban background (North Kensington) locations, plus two rural sites to the West (Harwell) and East (Detling) of the city. Ascorbate and glutathione-dependent oxidative potential (OP^{AA} and OP^{GSH}, expressed per unit mass of PM) were then determined following incubation in a synthetic respiratory tract lining fluid (4 hours at 37°C, pH 7.0) using a novel on filter methodology.

Results In terms of the OP^{AA} metric only PM_{2.5} measured at the urban background location demonstrated a seasonal increase ($p < 0.05$) during the summer, with equivalent seasonal OPs across the remaining 3 sites and no differences apparent in PM₁₀.

In contrast, PM₁₀ and PM_{2.5} OP^{GSH} was significantly ($P < 0.001$) lower in the summer period at the urban background site. Summer increases in OP^{GSH} were observed at the rural sites, with evidence that summer PM_{2.5} OP^{GSH} was increased at the roadside site. This heterogeneous pattern of seasonal contrasts could not be simplistically related to the differences in period, or daily NO₂ or O₃ concentrations.

Conclusions No consistent seasonal pattern in PM₁₀ or PM_{2.5} OP was observed across the four sites and the associations between the measured particulate OPs with O₃ and NO₂ at these locations did not fit with the particle ageing hypothesis. Rather high OP days were often found to be associated with wind speed and direction, suggesting that the differences were driven by a combination of long and short range sources.

P123 WHAT CAUSES OCCUPATIONAL ASTHMA IN CLEANERS?

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Domestic cleaners show a consistently raised risk of developing asthma in population-based epidemiological studies. The main risks appear to be the use of bleach and sprayed cleaning agents. These risks are now found in other occupations, such as health-care due to the requirement for disinfection of clinical areas. Since 2000, there has been 26 cleaners and healthcare workers with occupational asthma (OA) reported to Shield (West Midlands registry for OA) thought to be due to cleaning agents. The majority of these were using chlorine-releasing substances containing dichloro-isocyanurate for disinfection purposes. We have carried out 8 specific inhalation challenge tests to chlorine-releasing tablets mixed with cold water but none of these have elicited an asthmatic reaction despite the workers previously showing work-related changes on serial peak flows when exposed. In a

Abstract P123 Figure 1 Specific inhalation challenge testing showed no asthmatic response to hartabs or urine alone. The chlorine urine mixture provoked a dual asthmatic reaction and sustained drop in FEV1 of up to 34% from base line. The grey box denotes the exposure.

separate worker, a positive reaction was seen when challenged to a washing up liquid containing triclosan (another chlorine based substance). Due to the negative nature of the majority of tests, we considered the possibility that the chlorine itself may not be the provoking factor, but that chlorine-releasing formulations have the potential to react with amines from urine or bodily fluids when they are being used to disinfect clinical areas.

We report a 48 year-old health-care assistant for the elderly exposed to Haztabs (a chlorine-based cleaner) at work who had clear evidence of occupational asthma from serial peak expiratory flow records (Oasys score 3.44, ABC score 40 L/min/hr, 5 positive timepoints), but negative tests to Haztab exposure when mixed with cold water. Adding urine (5%) to the Haztab mixture resulted in a dual immediate and late asthmatic reaction (Figure 1). She showed mild airway hyper-reactivity pre challenge which was not repeated post challenge due to a continued low FEV1 of 39% predicted.

Conclusions The SIC results suggested that chloramines were the cause of her asthma, similar to the agents causing occupational asthma in workers exposed to indoor swimming pool vapours/mists.

P124 AN INVESTIGATION INTO COMORBIDITY ACCUMULATION IN ASTHMA PATIENTS WITH SYSTEMIC STEROID EXPOSURE

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Introduction Much of the economic cost associated with severe asthma is related to the treatment of comorbidities, several of which arise from systemic steroid exposure related to asthma management. However these comorbidities may not progress equally across age-groups and genders following systemic steroid exposure.

Aim To examine the relationship between age-groups and gender in relation to prevalence of steroid induced comorbidity and the associated costs.

Methods Data for a cohort of 808 patients with severe asthma (SA) matched by age and sex with a cohort of 3,975 patients with a diagnosis of mild asthma and 2,412 non-asthma patients with a diagnosis of rhinitis were extracted from the Optimum Patient Care Research Database (OPCRD). Consultation data were used to identify comorbidities and conditional logistic regression analysis provided odds ratios (95% CIs) for comorbidity risk between cohorts by age-group and gender. Prescription data for individual comorbidities was coded using Prescription Cost Analysis data from Northern Ireland and regressed upon cohort, age and gender.

Results Results presented below focus on a comparison between the non-asthma and severe asthma cohorts. Compared to older patients, younger patients in the SA cohort show significantly higher odds ratios relative to the non-asthma cohort for many comorbidities (Table 1). Apart from chronic kidney disease, hypercholesterolemia, osteoporosis and osteopenia, males with SA have higher odds ratios for all other comorbidities listed in Table 1 relative to non-asthma patients than females with SA. Upon examination of costs for individual comorbidities, males cost more than females for type II diabetes, hypertension, psychiatric disorders, while the opposite was observed for osteoporosis and osteopenia. Significant linear and non-linear increasing relationships were observed across age for costs related to type II diabetes, hypertension, hypercholesterolemia, dyspeptic disorders, cardiovascular disease, osteoporosis and osteopenia. Furthermore cost differentials for age and gender exhibited varying relationships across morbidities.

Conclusions The odds of having many steroid-induced comorbidities are higher among younger persons; differences between genders are also evident. While patients' with SA cost significantly more than patients without steroid exposure or with mild steroid exposure, the distribution of the cost across age and gender varies across comorbidities.

Abstract P124 Table 1 Summary of odds ratios for comparison of comorbidity risk between severe asthma and rhinitis cohorts

	Severe Asthma Cohort vs Rhinitis (Non-Asthma) Cohort – Odds Ratio (95% Confidence Intervals)					
	Gender		Age-group			
	Male	Female	≤45 yr	46–60 yr	61–70 yr	>70 yr
Type 2 Diabetes	1.0 (0.6–1.6)	2.7 (1.8–4.1)***	4.2 (1.1–18.0)**	4.0 (2.2–7.2)***	1.7 (0.9–3.2)	0.9 (0.5–1.6)
Obesity	1.8 (1.3–2.4)***	2.8 (2.2–3.5)***	3.7 (2.4–5.6)***	3.3 (2.4–4.6)***	1.8 (1.3–2.6)***	1.8 (1.3–2.6)***
Osteoporosis†	49.2 (11.2–217)***	4.6 (3.2–6.8)***	18.3 (6.4–52.8)***		6.2 (3.0–12.6)***	5.2 (4.0–6.9)***
Osteopenia†	‡	4.5 (2.8–7.3)***	50.4 (12.0–210)***		2.7 (1.2–6.3)***	3.1 (1.5–6.6)***
Hypertension	1.9 (1.4–2.6)***	1.7 (1.3–2.2)***	4.1 (1.9–8.9)***	2.4 (1.6–2.4)***	1.7 (1.2–2.5)***	1.2 (0.9–1.7)
Chronic Kidney Disease†	3.9 (2.4–6.4)***	1.9 (1.3–2.7)***	7.7 (3.6–16.8)***		3.2 (1.7–5.9)***	1.4 (1.0–2.1)*
Dyspeptic Disorders	5.5 (4.0–7.6)***	7.8 (5.9–10.3)***	9.4 (5.8–15.2)***	9.8 (6.5–14.7)***	6.2 (3.9–9.8)***	4.0 (3.3–4.8)***
Psychiatric Disorders	1.6 (1.2–2.2)***	2.0 (1.6–2.6)***	2.6 (1.8–3.9)***	2.1 (1.6–2.9)***	1.8 (1.2–2.5)***	1.3 (0.9–2.0)
Sleep Disorders	1.2 (0.4–3.4)	3.5 (1.9–6.7)***	12.9 (1.6–103)**	8.1 (2.1–30.3)***	2.3 (0.7–7.5)	0.6 (0.2–1.8)
Hypercholesterolemia	2.1 (1.4–3.1)***	1.4 (1.0–1.9)*	‡	2.2 (1.3–3.8)***	1.7 (1.1–2.8)**	1.2 (0.8–1.8)

†Age groups '≤45 yr' and '46–60 yr' were combined

‡Too few cases of comorbidity for analysis

* P<0.1, ** P<0.05, ***P<0.01