P205 BURDEN OF ANXIETY AND DEPRESSION IN THE DIFFICULT ASTHMA CLINIC AND RELATIONSHIP TO OUTCOME

¹JSS Canine, ²I Bryce, ²G MacDonald, ¹D Cowan, ¹M Brewis. ¹Glasgow Royal Infirmary, Glasgow, UK; ²Stobhill Ambulatory Care Hospital, Glasgow, UK

10.1136/thoraxjnl-2017-210983.347

National Review of Asthma Deaths (NRAD)¹ stated that “there is a well-recognised link between asthma and psychosocial problems; the prevalences of anxiety, depression and panic disorder are much higher in people with asthma than in matched controls and are associated with poor outcomes.” We aim to evaluate the prevalence of psychological illness within our difficult asthma clinic and its impact on asthma outcomes using data from the BTS Difficult Asthma Registry.

Methods Retrospective analysis of data submitted to the BTS Difficult Asthma Registry from July 2009 to June 2016. Outcomes were compared in those with and without anxiety or depression as defined by Hospital Anxiety and Depression (HAD) scores for each of $\geq 11/21$. Categories for comparison included Asthma Control Questionnaire (ACQ) and Asthma Quality of Life Questionnaire (AQLQ) scores, Forced expiratory volume in one second (FEV₁), Fraction of exhaled nitric oxide (FeNO), Unplanned GP/A+E attendances, Hospital Admissions, and need for oral prednisolone. Simple statistical tests such as Mann-Whitney and unpaired t tests were used to analyse the data.

Results The database included 198 individuals (68 male and 130 female), of whom 69 (35%) had anxiety and 49 (25%) had depression. There were no statistically significant differences between each group for FEV₁, FeNO and peripheral eosinophil count. Anxiety was associated with higher ACQ and lower AQLQ scores than non-anxiety (4 vs 2.8 and 2.9 vs 3.8 respectively, both $p < 0.0001$) and increased number of steroid boosts per year (6 vs 5, $p = 0.013$). Depression was associated with higher ACQ and lower AQLQ scores than non-depression ($p < 0.0001$) and increased number of steroid boosts per year (6 vs 5, $p = 0.021$).

Conclusions There is a high prevalence of anxiety and depression in the difficult asthma clinic, and both co-morbidities are associated with a greater symptom burden and an increased number of steroid boosts per year not explained by objective measures of asthma control. Further research is required to evaluate the impact of clinical psychology in this setting.

REFERENCE

1. NRAD – <https://www.rcplondon.ac.uk/projects/outputs/why-asthma-still-kills>

P206 A STUDY TO INVESTIGATE THE MECHANISMS UNDERLYING CIRCADIAN RHYTHM IN ASTHMA

HJ Durrington, K Krakowiak, D Singh, D Ray. University of Manchester, Manchester, UK

10.1136/thoraxjnl-2017-210983.348

Background Time of day is critical in the pathogenesis of asthma, and has been realised for centuries. Symptoms of asthma are worse around 4 am, when airway restriction is at its highest. Many asthma treatments are taken in the morning or evening, however there is increasing data that steroids are more efficacious if taken mid-afternoon. Investigating the biological timing of asthma is crucial to better understand the

pathogenesis of asthma, this may lead to the discovery of new drug targets, and the identification of dynamic biomarkers that change by time of day and would be useful in a future chronotherapeutics study.

Aims • Define new biochemical pathways involved in the circadian variation in asthma

• Determine a circadian biomarker in asthma

Method We recruited 10 atopic, moderately severe asthmatics and 10 healthy volunteers to complete 4 study visits, including an overnight stay. Blood, induced sputum and breath were sampled at intervals throughout the day and night, and physiological measurements made. PBMCs were harvested from peripheral blood at 4 am and 4 pm and plated in 6 groups, control, +LPS/anti-CD3/anti CD28, +P38 i, +LPS/anti-CD3/anti CD28 +P381, +Dexamethasone, +LPS/anti-CD3/anti CD28 +dexamethasone for 2 hours. Conditioned medium was collected and frozen at -80°C , cell lysates were prepared for RNA extraction, and protein purification.

REC reference 14/NW/1352.

Results There is a high amplitude circadian change in FEV₁ in asthmatics compared to healthy controls. The nadir is at 4 am (figure 1). There was also an increase in sputum and serum eosinophils (a key effector cell in asthma) at 4 am compared to 4 pm in asthmatics (figure 1).

On-going workflow includes

- Transcript measurement from PBMCs using a combination of RNA-Sequencing and nanostring technology (for clock genes; inflammatory mediators; Signalling regulators; MAP kinase family members)
- Bioplex screen for expression and activation of additional MAPkinase components, and IkBa, from protein extracts and conditioned media
- Lipidomic analyses of serial, matched serum samples will allow analysis of the ceramide/sphingolipid pathway
- Breathomics analysis for volatile organic compounds in serial, matched breath samples.

Discussion We have demonstrated a significant diurnal effect on asthma lung physiology and eosinophil profiles. Further downstream analysis of serum, breath and sputum is underway.

P207 AMBIENT AIR POLLUTION AND ADMISSIONS TO HOSPITAL WITH EXACERBATIONS OF ASTHMA

F Thompson, A Hujan, M Richardson, G Woltmann, S Siddiqui, S Gonen. University of Leicester, Leicester, UK

10.1136/thoraxjnl-2017-210983.349

Background Air pollution has been linked to increased morbidity and mortality associated with a number of chronic health conditions including asthma. We hypothesised that levels of ambient air pollution would be related to the number of hospital admissions with exacerbations of asthma.

Methods Data on asthma admissions to a large acute NHS trust in the East Midlands were extracted using discharge diagnosis codes over a five-year period from April 2011 to March 2016. Ambient air pollution levels during this period were obtained from the website of the Department for Environment, Food and Rural Affairs. These data were based on a single monitoring station located in the centre of the City which recorded hourly readings of ozone, nitrogen oxides and

Abstract P206 Figure 1

small particulate matter (PM_{2.5}). Daily mean data were utilised in the analysis. The effect of ambient air pollution on daily admissions for asthma was assessed using a generalised linear model in R version 3.3.1. Independent variables in the model were time, Ozone, NO, NO_x and PM_{2.5}. Fourier terms to capture any long-term trend or seasonality were also included in the model. Delayed exposure effects were investigated by fitting separate constrained lag models.

Results During the period from April 2011 to March 2016 there were 4204 admissions due to asthma exacerbations (71% female, mean age 48 years). Admission numbers increased progressively from 726 in 2011/12 to 1043 in 2015/16. Admissions were most frequent in the month of December (2.9 per day) and least frequent in August (1.5 per day). None of Ozone, NO, NO_x and PM_{2.5} or their lagged terms were found to be independent predictors of daily admissions for asthma.

Conclusion Ambient air pollution measured at a single fixed monitoring station within a city is not able to predict the frequency of admissions due to asthma. Further research on the health effects of air pollution should make use of more detailed assessments of city-wide exposure, for instance by utilising satellite imaging and geospatial mapping.

P208

EFFECTS OF PERSONAL AIR POLLUTION EXPOSURE ON ASTHMA SYMPTOMS, LUNG FUNCTION AND AIRWAY INFLAMMATION

L Chambers, J Finch, K Edwards, A Jeanjean, R Leigh, S Gonem. *University of Leicester, Leicester, UK*

10.1136/thoraxjnl-2017-210983.350

Introduction and objectives There is evidence that exposure to traffic-related air pollution may impact adversely on asthma and other respiratory conditions. We aimed to test the hypothesis that day-to-day variability in personal air pollution exposure has clinically important effects on asthma symptoms, lung function and airway inflammation.

Methods We recruited 31 patients with a clinical diagnosis of asthma from Leicester and the surrounding area. Participants were provided with a personal air pollution monitor (Cairclip NO₂/O₃; Cairpol, La Roche Blanche, France) which measures total oxidants in the ambient air and stores 15 min average readings. The Cairclip was attached to a belt or handbag when outdoors, and kept in close proximity to participants when indoors. We also measured exposure using modelled daily average ambient levels of NO₂ and particulate matter

Abstract P208 Figure 1

(PM_{2.5} and PM₁₀) based upon satellite imaging data, as well as directly measured ozone, NO₂ and PM_{2.5} levels from a monitoring station in central Leicester.

Participants were provided with an electronic symptom diary and peak expiratory flow metre (Micro Diary; Carefusion, Basingstoke, UK) and a portable exhaled nitric oxide monitor (NOBreath; Bedfont, Maidstone, UK), and made daily measurements using these for 12 weeks. Relationships between exposure and outcome variables were analysed using cross-correlation.

Results At a group level there were no consistent relationships between personal air pollution exposure and clinical outcomes. However, a number of individuals (n=6) manifested strong correlations between exposure and outcome variables, particularly those residing within inner-city Leicester. Figure 1 shows overlaid time series of modelled ambient NO₂ levels and exhaled nitric oxide measurements in Participant 13 (Panel A). Results of the cross-correlation analysis are shown in Panel B, indicating a strong positive correlation centred on a lag of zero days.

Conclusions Air pollution exposure appears to have a significant effect on symptoms, lung function and airway inflammation in selected patients with asthma. These individuals may have a combination of increased susceptibility and higher than average levels of exposure. Future studies should focus on understanding the genetic and epigenetic mechanisms underlying susceptibility to air pollution, as well as developing strategies to mitigate the effects of exposure in susceptible individuals.

Sleep and breathing

P209 CHARACTERISTICS OF EAST LONDON CHILDREN WITH SEVERE OBESITY REQUIRING NON-INVASIVE VENTILATION FOR SLEEP DISORDERED BREATHING

SMN Brown, J Rae, A Franklin, E Mapazire, J Bettencourt. Royal London Children's Hospital, Barts Health NHS Trust, London, UK

10.1136/thoraxjnl-2017-210983.351

Introduction and objectives Childhood obesity is an increasing problem leading to significant health concerns. Children are referred to our Tier 3 obesity service if they have BMI ≥ 3.5 standard deviations (SD) or BMI ≥ 3 SD with comorbidities. There are currently 120 children under the care of the service; 22 have severe sleep disordered breathing (SDB) requiring non-invasive ventilation (NIV) overnight. We sought to characterise the demographics of those with sleep disordered breathing, the extent of associated comorbidities and to assess adherence to NIV.

Methods A retrospective review of all data from patients under the care of the obesity service with SDB requiring NIV was undertaken. Adherence to NIV was assessed by patient and parent/guardian verbal report.

Results The median onset of obesity was <5 years of age. 45% of patients reported poor adherence to NIV. The majority of children had obstructive sleep apnoea (OSA) requiring CPAP. 45% of patients had also undergone adenotonsillectomy. Those requiring BiPAP support had evidence of hypoventilation. Further patient demographics and comorbidities are presented in Table 1. There were no patients with pulmonary hypertension although one had cardiomyopathy.

Conclusion There is a high level of associated comorbidities in our patient cohort – highlighting the severe medical complications already present in these young patients. This is important to note for respiratory paediatricians who may be looking after these children for SDB. Obesity services are fragmented throughout the UK and not all children will have access to an obesity service. Vitamin D deficiency is independently associated with obesity and our Results highlight the importance of screening for this. In addition a high level of non-adherence to NIV was reported, although probably underestimated. Although only 9% were known to mental health services, severe obesity is associated with mental health issues which are currently not adequately addressed by our service. NICE guidance states that Tier 3 obesity services should include a psychologist and social worker. Most services do not fulfil these requirements through lack of funding. We aim to recruit a psychologist to support this group of patients in weight loss management and NIV support.

Abstract P209 Table 1 Demographics and comorbidities

PATIENT DEMOGRAPHICS		COMORBIDITIES	
Gender (female)	63	Hypertension	23
Age at referral (years)	9.7 (5–13.4)	Lipid abnormalities	45
Current age (years)	14.3 (10.6–16.3)	Type 2 diabetes	14
Age at commencing NIV (years)	11.6 (7–14.8)	Insulin resistance	50
CPAP	86	Steatohepatitis	18
BiPAP	14	Vitamin D deficiency	92
Adenotonsillectomy	45	Asthma	27
Chromosomal abnormality	23	Pulmonary hypertension	0
Child protection register	9	CAMHS	9

Results are presented as%. For ages, results are median (interquartile range)
CAMHS – Child and Adolescent Mental Health Services

P210 PERI-OPERATIVE TREATMENT OF SLEEP-DISORDERED BREATHING AND OUTCOMES IN BARIATRIC PATIENTS

¹JH Meurgey, ²R Brown, ²A Woloszy, ²J Steier. ¹King's College London, London, UK; ²Guy's and St. Thomas' NHS Foundation Trust, London, UK

10.1136/thoraxjnl-2017-210983.352

Background Obstructive sleep apnoea (OSA) is common in bariatric patients undergoing sedation during surgery. However, its contribution to peri-operative respiratory

complications and mortality has not been established. We sought to pre-operatively identify OSA in bariatric patients and record peri-operative complications following bariatric surgery.

Methods Data were collected and analysed from June 2014 to March 2017 for 410 bariatric surgery patients referred to the sleep laboratory for pre-operative screening and treatment of OSA. The STOP-BANG questionnaire, Epworth Sleepiness Scale (ESS) and nocturnal pulse oximetry were recorded and treatment was allocated with continuous positive airway pressure (CPAP). Peri-operative complications and mortality were the primary outcome measures for patients receiving CPAP treatment for OSA, with patients not requiring CPAP used as control. The mean follow-up time for all patients was 433 days; 732 days for the patients who had undergone bariatric surgery. The two groups were compared with Chi-square test and unpaired two-tailed t-test.

Results Significant OSA was present in 70% of the screened patients, 40% of patients involved in the study received CPAP treatment. Patients receiving CPAP treatment [49.5 (11.3) years old, 61% female, 50.3 (8.5) kg/m²] were older, had a lower female percentage and had a higher BMI than those not receiving CPAP [44.9 (12.0), 81% female, 46.6 (7.7)]. No significant differences, including hospital stay or rate of complications, were observed between patients on CPAP and those not on CPAP. Out of 53 patients who had undergone bariatric surgery at the cut-off date, only 1 respiratory complication had occurred.

Conclusion Bariatric patients who are screened pre-operatively for OSA and treated per guidelines have no increased risk of respiratory complications compared to patients without OSA.

Abstract P210 Table 1 Comparison of patients with CPAP vs those without CPAP. BMI, body mass index; HADS, hospital anxiety and depression scale; ODI, oxygen desaturation index; SpO₂, peripheral capillary oxygen saturation.

	CPAP	NON-CPAP	P-VALUE
Age (years)	49.5 (11.3); N = 155	44.9 (12.0); N = 241	<0.001
Male vs. Female ratio	0.63:1; N = 155	0.24:1; N = 244	<0.001
Weight (kg)	142.9 (29.8); N = 155	129.1 (24.5); N = 244	<0.001
BMI (kg.m ⁻²)	50.3 (8.5); N = 155	46.6 (7.7); N = 244	<0.001
Length of stay (days)	2.42 (1.0); N = 14	2.57 (1.3); N = 19	0.65 ^b
HADS (score out of 42)	16.1 (7.0); N = 10	20.5 (7.5); N = 14	0.153
Mean heart rate (min ⁻¹)	75.8 (10.6); N = 152	71.4 (8.4); N = 239	<0.001
Pulse rise (h ⁻¹)	41.8 (20.7); N = 150	28.9 (15.0); N = 240	<0.001
Mean spo ₂ (%)	92.7 (3.8); N = 153	95.1 (3.2); N = 244	<0.001
Nadir spo ₂ (%)	69.0 (11.3); N = 150	80.4 (5.7); N = 240	<0.001
4% ODI (h ⁻¹)	29.2 (19.7); N = 153	6.4 (6.5); N = 244	<0.001
3% ODI (h ⁻¹)	36.4 (21.1); N = 153	9.5 (8.0); N = 244	<0.001
<90% spo ₂ (% of sleep study)	16.5 (17.8); N = 153	2.4 (6.8); N = 244	<0.001