severe exacerbation and time to first URI. Secondary outcomes included peak severity and area under the curve for exacerbation symptoms. A pre-specified sub-group analysis was conducted to determine whether effects of the intervention on co-primary outcomes were modified by baseline vitamin D status. This trial is registered with ClinicalTrials.gov (NCT00977873).

Results 122 participants were allocated to the intervention arm of the trial, and 118 to the control arm. Vitamin D supplementation did not influence time to first exacerbation (HR 0.86, 95% CI 0.60–1.24, p = 0.42) or time to first URI (HR 0.95, 95% CI 0.69–1.31, p = 0.75) in the study population as a whole, but it did reduce peak severity (p = 0.042) and area under the curve (p = 0.032) for exacerbation symptoms. Pre-specified sub-group analysis revealed that vitamin D supplementation was protective against moderate/severe exacerbation among the 148 participants with baseline serum 25-hydroxyvitamin D (25[OH]D) concentration < 50 nmol/L (aHR 0.57, 95% CI 0.35 to 0.92, p = 0.021), but not among the 92 participants with baseline serum 25(OH) D ≥ 50 nmol/L (aHR 1.45, 95% CI 0.81 to 2.62, p = 0.21; P for interaction = 0.021). Baseline vitamin D status did not modify the effect of vitamin D supplementation on risk of URI (P for interaction = 0.41).

Conclusions Vitamin D supplementation protected against moderate/severe exacerbation, but not upper respiratory infection, in COPD patients with baseline 25(OH)D < 50 nmol/L. It also modestly reduced peak severity and area under the curve for exacerbation symptom scores, irrespective of baseline vitamin D status.

S105 THE EFFECTS OF REAL-WORLD EXPOSURES TO DIESEL TRAFFIC EMISSIONS ON CARDIO-RESPIRATORY OUTCOMES IN COPD : 'OXFORD STREET 2'

[1]R Sinharay, [2]B Barratt, [3]J Gong, [1]C Goward, [1]JP Rocha, [2]F Kelly, [3]J Zhang, [4]P Cullinan, [1]KF Chung. *[1]Royal Brompton and Harefield NHS Foundation Trust, Imperial College, London, UK; [2]Kings College Environmental Research Group, London, UK; [3]Duke Global Health Initiative, Durham, United States; [4]Royal Brompton and Harefield NHS Foundation Trust, London, UK*

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Introduction and objectives We studied the changes in lung function and cardiovascular responses in healthy volunteers and patients with COPD exposed to the high pollution levels in a busy London street.

Methods Using a cross-over design, 37 healthy volunteers and 37 COPD patients (walked along Oxford Street (diesel only traffic) and, on a separate occasion, in Hyde Park (low or little traffic), London for two hours. Cardio-respiratory measurements were performed at baseline, and during and after each exposure, alongside personal particulate and gaseous exposure measurements.

Findings Compared to Hyde Park, mean exposures on Oxford Street had higher levels of black carbon (10.4 μm/m^3 vs. 1.2 μm/m^3, p < 0.001) and ultrafine particle counts (25472/cm^3 vs 5709/cm^3, p < 0.001).

In comparison with Hyde Park the healthy subjects had a mean fall in FEV_1 from baseline of6.05% (p = 0.01) 6 h and a fall of 4.17% (p = 0.01) 24 h after arrival in Oxford St. There was no associated drop in FVC. Arterial stiffness measured by pulse wave velocity (PWV) increased 24 h after arriving on Oxford Street.

In volunteers with COPD, there was a mean fall in FEV_1 of 4% (p = 0.01) with an associated drop in FVC of 3.4% (p = 0.02) one hour after the start of exposure on Oxford Street, compared to Hyde Park. Measurement of impulse oscillometry in volunteers with COPD demonstrated increased airway resistance at 5 Hz of 0.05 kPa/l/s (p = 0.01) four hours and at 20 Hz of 0.02 (p = 0.04) 24 h after exposure began on Oxford Street. PWV increased by 0.8 m/s and 0.5 m/s three hours and six hours after exposure started on Oxford street respectively.

Abstract S105 Table 1 Mean changes in, FVC, at IOS 5Hz andHz, FeNO and PWV after exposures began in Oxford Street (OS) and Hyde Park (HP)

	Healthy				COPD			
	OS	HP	Δd	p	OS	HP	Δd	p
Spirometry (difference%)								
FEV_1 1 h	0.78	3.46	-2.68	0.09	**-2.30**	**1.70**	**-4.00**	**0.01** *
FEV_1 2 h	0.49	2.38	-1.89	0.17	-1.43	-0.38	-1.05	0.39
FEV_1 6 h	**1.84**	**7.89**	**-6.05**	**0.01** *	1.68	2.08	-0.41	0.79
FEV_1 24h	**0.05**	**4.22**	**-4.17**	**0.01** *	1.85	0.92	0.93	0.65
FVC 1 h	0.00	1.81	-1.81	0.28	**-1.84**	**1.57**	**-3.41**	**0.02** *
FVC 2 h	-1.00	1.16	-2.16	0.32	-3.22	-0.11	-3.11	0.09
FVC 6 h	0.22	3.57	-3.35	0.17	1.38	1.60	-0.22	0.90
FVC 24h	-1.60	-2.84	1.24	0.68	1.60	2.54	-0.95	0.73
IOS 5hz (difference kPa/l/s)								
IOS 5hz 4 h	-0.01	-0.01	0.00	0.78	**0.03**	**-0.02**	**0.05**	**0.01** *
IOS 20hz 4 h	-0.01	-0.02	0.01	0.48	-0.01	-0.04	0.03	0.09
IOS 20hz (difference kPa/l/s)								
IOS 20hz 4 h	-0.01	-0.01	0.00	0.80	-0.72	-0.03	-0.68	0.35
IOS 20hz 24h	-0.01	-0.01	0.00	0.73	**0.00**	**-0.03**	**0.02**	**0.04** *
FeNO (difference ppb)								
3h	-5	-4	-1	0.75	-2	-4	2	0.71
5h	-4	-2	-2	0.37	-7	-7	-1	0.89
24h	-4	-1	-3	0.26	2	-6	8	0.09
PWV (difference m/s)								
3 h	-0.1	-0.2	0.2	0.47	**0.1**	**-0.6**	**0.8**	**0.03** *
6h	0.3	0.0	0.3	0.32	**0.2**	**-0.3**	**0.5**	**0.03** *
24 h	**0.6**	**-0.4**	**1.0**	**0.04** *	0.3	-0.4	0.7	0.32

* p < 0.05
Δ d Mean difference of difference between each exposure site

There were no changes in FeNO in either group between the two sites.

Preliminary multivariate analysis has so far found no associations with individual particulate measurements.

Conclusions These findings show that airways obstruction occurred in both the healthy volunteers and COPD patients exposed to ambient levels of diesel pollution on a busy London Street. The associated vascular dysfunction was more prominent in COPD patients. Further analyses of markers of inflammation in the collected samples are now needed to ascertain the mechanistic cause of the pathophysiological findings.

S106 REVIEW OF EUROPEAN COPD AUDIT DATA: FACTORS AFFECTING LENGTH OF STAY

[1]M Ruparel, [2]JL López-Campos, [3]F Pozo-Rodriguez, [3]A Castro-Acosta, [4]S Hartl, [5]CM Roberts. *[1]Royal Free Hospital, London, UK; [2]Unidad Medico-Quirurgica de Enfermedades Respiratorias/Instituto de Biomedicina de Sevilla (IBiS), Hospital Universitario Virgen Del Rocio/CIBER de Enfermedades Respiratorias (CIBERES), Seville, Spain; [3]Centre for Biomedical Research on Respiratory Diseases (CIBERES). Instituto de Salud Carlos III, Madrid, Spain; [4]Ludwig Boltzmann Institute of COPD and Respiratory Epidemiology, Otto Wagner Hospital, Vienna, Austria; [5]Barts and the London School of Medicine and Dentistry, Queen Mary, University of London, London, UK*

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