LETTERS TO THE EDITOR

Acute exposure to biomass smoke causes oxygen desaturation in adult women

Long-term exposure to biomass fuel smoke indoors is causally associated with chronic obstructive pulmonary disease (COPD)¹ but the effect of acute exposures has not been studied. Should acute effects occur, this might increase the risk of an exacerbation of pre-existing lung disease. We studied acute changes in lung function and oxygen saturation in individuals exposed regularly to biomass smoke in Nepal.

Spirometry (EasyOne spirometer using American Thoracic Society (ATS) criteria) and oxygen saturation data were collected from 26, randomly selected, life-long non-smoking women (mean±SD age 38.0±11.7 years) before and after cooking (morning or evening). Smoke concentrations were measured using a DustTrak (TSI, Shoreview, Minnesota, USA) during cooking events and expressed as PM_{2.5} (particulate matter of <2.5 µm in diameter).²

Geometric mean (range) PM_{2.5} exposures were 2742 (1951–3853) µg/m³ for a mean ±SD period of 164±34 min.

Spirometry results are presented in table 1. Taking the best values, group mean forced expiratory volume in 1 s (FEV₁) was 1.95 litres (77% predicted), group mean forced vital capacity (FVC) was 2.44 litres (84% predicted) and group mean FEF_{25–75} (forced expiratory flow between 25% and 75% of FVC) 2.19 l/s (60% predicted). Three (12.5%) women had COPD (2 severe and 1 moderate, GOLD (Global Initiative on Obstructive Lung Disease classification), 8 (33%) had a restrictive defect ranging from mild to severe and 9 (37.5%) had small airways disease. When rested after cooking FEV₁ and FEF_{25–75} fell but not significantly (FEV₁: morning p=0.264 and evening p=0.421; FEF_{25–75} morning p=0.142 and evening p=1.000) when re-tested after cooking.

All subjects had normal SaO₂ (arterial oxygen saturation) values before cooking but values fell significantly after cooking (mean difference before and after cooking in the morning, –1.56%, 95% CI –0.94 to 2.18 (p<0.001) and in the evening, –1.53%, 95% CI –0.76 to 2.30 (p=0.001)). Heart rate fell significantly after the morning cooking

period (–5.4±9.7, 95% CI 10.3 to 0.6, p=0.029) but not during the evening (–0.7±8.5, 95% CI 5.2 to 3.9, p=0.751). The changes were independent of fuel type and were more likely to be seen in those with abnormal lung function especially if the defect was severe.

This study suggests that women in low and middle income countries regularly exposed to biomass smoke who have chronic lung disease might be more at risk of hypoxia if they have marginal gas exchange when exposed acutely to biomass smoke. Consequently, while biomass smoke exposure contributes to the development of COPD, once established in an individual continued smoke exposure may further impair gas exchange, putting individuals at risk of cardiopulmonary decompensation.

These changes in SaO₂ did not relate to acute changes in lung function during cooking suggesting that the acute changes in SaO₂ were due to ventilation–perfusion imbalance. This could be due to inhalation of fine particles causing small airway narrowing³ or could affect pulmonary capillary blood flow either by a direct toxic effect from the particles or through autonomic nervous system stimulation.

Table 1 Best values for lung function and change in oxygen saturation during cooking (ΔSaO₂) for each individual—all individuals were women

ID	Age (years)	Absolute values				Oxygen saturation in the morning (%)		Oxygen saturation in the evening (%)		Mean PM _{2.5} (µg/m ³)*	Time exposed (min)	Classification
		FEV ₁	FVC	FEV ₁ /FVC %	FEF _{25–75}	BMC–AMC	ΔSaO ₂	BEC–AEC	ΔSaO ₂			
1	58	1.53	2.00	77	1.26	98–96	–2	–	–	804	194	SAD
2	32	2.02	2.53	82	1.96	98–97	–1	98–94	–4	12198	192	SAD
3	49	2.00	2.45	82	2.19	98–97	–1	98–98	0	2615	204	SAD
4	20	2.37	2.86	86	2.61	99–98	–1	–	–	7078	103	SAD
5	48	1.88	2.25	84	2.33	–	–	98–97	–1	7078	103	SAD
6	26	2.33	2.82	83	2.37	97–96	–1	–	–	3380	151	SAD
7	20	2.23	2.30	97	3.84	–	–	98–97	–1	3380	151	Moderate restriction
8	32	2.20	3.04	73	1.58	98–97	–1	–	–	15280	150	SAD
9	65	0.62	1.63	52	0.26	–	–	98–98	0	15280	150	Severe COPD
10	35	2.09	2.42	86	2.88	97–93	–4	98–96	–2	4838	235	Mild restriction
11	36	1.99	2.40	83	2.15	98–98	0	98–96	–2	15116	201	Mild restriction
12	35	2.57	2.92	89	3.56	96–96	0	98–98	0	2756	183	Normal
13	34	2.46	3.26	82	2.35	–	–	–	–	2756	183	SAD
14	35	1.87	2.26	83	2.02	98–96	–2	98–98	0	808	190	Mild restriction
15	52	2.09	2.33	86	2.28	98–97	–1	97–96	–1	6357	135	Normal
16	55	0.60	1.07	56	0.36	96–91	–5	96–91	–5	1467	160	Severe COPD
17	41	1.81	2.28	91	2.19	97–96	–1	97–94	–3	291	169	Mild restriction
18	28	1.48	1.82	93	1.98	96–94	–2	96–93	–3	3364	154	Severe restriction
19	19	1.73	2.24	80	1.38	98–97	–1	–	–	1135	171	Moderate restriction
20	24	2.90	3.33	87	5.34	–	–	98–97	–1	1135	171	Normal
21	37	2.69	3.45	78	2.30	98–96	–2	97–96	–1	8261	162	SAD
22	36	2.64	3.06	86	3.40	98–97	–1	97–97	0	4523	108	Normal
23	45	1.34	1.93	69	0.78	98–96	–1	97–95	–2	675	162	Moderate COPD
24	50	1.35	1.79	80	1.19	–	–	–	–	675	162	Mild restriction
Mean±SD	38±12.5	1.95±0.59	2.44±0.58	81.04±10.27	2.19±1.12	–	–1.56±1.25	–	–1.53±1.50	4741±4879	166±31.96	

Normal values were taken as 10% below Caucasian values.

Two subjects were excluded because of poor quality blows.

*Cumulative exposure during the cooking period in the morning and evening.

AEC, after evening cooking; AMC, after morning cooking; BEC, before evening cooking; BMC, before morning cooking; COPD, chronic obstructive pulmonary disease; FEV₁, mean forced expiratory volume in 1 s; FEF_{25–75}, forced expiratory flow between 25% and 75% of FVC; FVC, forced vital capacity; SAD, small airways disease.

In summary, these findings suggest that acute exposure to biomass smoke in women with small airways disease or restrictive lung disease is associated with blood deoxygenation, suggesting that in these individuals continued exposures may increase the risk of disease exacerbation.

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Simulated driving performance coupled with driver behaviour can predict the risk of sleepiness-related car accidents

Obstructive sleep apnoea syndrome (OSAS) is associated with the risk of sleepiness-related car accidents and worse driving simulator performances. Nevertheless, driving simulator studies failed to predict the real-world accident risk. Two-thirds of patients with OSAS will never experience a car crash, so that restricting their driving licence because of an OSAS diagnosis is not feasible.¹

Our previous studies validated a 30 min monotonous driving scenario (STISIM 300 Driving Simulator, Systems Technology, Hawthorne, California, USA) versus subjective and objective measures of sleepiness in healthy volunteers undergoing a sleep deprivation challenge and in patients with OSAS. Our test disclosed that primary vehicle control parameters (crashes, lane position variability) were the strongest predictors of sleepiness and, potentially, of crash risk.^{2,3} We reviewed the driving simulator and sleepiness (maintenance of wakefulness test (MWT) and Epworth sleepiness scale (ESS)) data of

43 men (mean age 53±9 years) with severe OSAS (mean apnoea-hypopnoea index of 55±16/h) together with their responses to a questionnaire on driving history. Clinical features, self-reported behaviour and driving simulation data are reported in table 1, together with statistical comparisons (Mann–Whitney or χ^2 tests). Twenty patients (47%) had crashed in the previous 5 years and frequently considered sleepiness the main cause of their accident. They were rated sleepier on the ESS ($p=0.038$), and tended to crash sooner in the driving simulation ($p=0.05$), without any other difference. Therefore, the identification of patients at risk based only on laboratory measures is not feasible.

Twenty-eight (65%) patients continued to drive while sleepy ('risky behaviour'), without differing significantly from patients that stopped driving ('safe behaviour'). Within the 'risky behaviour' subgroup, the 14 (50%) patients with a crash history were sleepier according to the ESS ($p=0.019$) without reaching statistical significance on the MWT ($p=0.062$), crashed more frequently ($p=0.036$) and sooner ($p=0.007$) at the driving simulation, and showed worse tracking performance ($p=0.017$) than patients without self-reported crash history. Conversely, there were no differences between subjects with (40%) and without (60%) a crash history in the subgroup of 15 patients that used to stop driving while sleepy.

Previous driving simulator studies were controversial, showing either the presence^{3,4} or the absence⁵ of association between driving simulator performance and accident risk. These conflicting results could reflect the confounding effect of the unexplored

Table 1 Clinical features, self reported behaviour and driving simulation data of patients with and without crash history and in the subgroups with safe and risky behaviour

Clinical features	All patients			Safe behaviour			Risky behaviour		
	No crash	Crash	p Value	No crash	Crash	p Value	No crash	Crash	p Value
Age	53.9±7.4	52.6±9.9	NS	54.3±5.9	46.3±11.6	NS	53.6±8.5	55.3±8.1	NS
BMI	31.9±5.0	31.9±4.5	NS	34.7±6.0	33.8±5.7	NS	30.2±3.5	31.1±3.9	NS
AHI	51.8±15.1	57.8±17.5	NS	52.2±17.2	57.9±17.1	NS	51.7±14.4	57.7±18.3	NS
ESS score	9.2±4.0	12.0±4.2	0.038	11.5±4.2	12.3±3.8	NS	7.7±3.3	11.8±4.5	0.019
Mean sleep latency on MWT (min)	23.4±14.3	16.1±12.7	NS	17.1±13.3	14.6±13.2	NS	27.4±13.9	16.7±13.0	[0.062]
Smoking (%)	17.4	36.8	NS	22.2	40.0	NS	14.3	35.7	NS
Habitual coffee drinking (%)	69.6	63.2	NS	77.8	100.0	NS	64.3	50.0	NS
Habitual alcohol drinking (%)	47.8	42.1	NS	55.6	40.0	NS	42.9	42.9	NS
Self-reported behaviour									
Driving >20000 km/year (%)	60.9	50.0	NS	44.4	50.0	NS	71.4	50.0	NS
Driving at night (%)	47.8	75.0	NS	33.3	66.7	NS	57.1	64.3	NS
Sleepy while driving at least monthly (%)	43.5	35.0	NS	66.7	50.0	NS	28.6	28.6	NS
Stopping driving while sleepy (%)	39.1	30.0	NS	100.0	100.0	NS	0.0	0	NS
Sleep attacks while driving at least monthly (%)	26.1	35.0	NS	33.3	50.0	NS	21.4	28.6	NS
Near-miss accidents in the last 5 years (%)	17.4	35.0	NS	0.0	16.7	NS	28.6	42.9	NS
Sleepiness-related accident (%)	0.0	70.0	<0.0001	0.0	66.7	0.004	0.0	71.4	0.0001
Mean driving simulator parameters									
Crashes (n)	0.5±1.0	2.7±5.9	NS	0.9±1.5	6.3±9.8	NS	0.2±0.3	1.2±2.4	0.036
Time to first crash (min)	27.1±5.1	22.3±8.6	0.050	14.2±7.1	22.3±10.8	NS	28.9±1.8	22.2±8.0	0.007
Lane position variability (m)	0.4±0.1	0.6±0.3	NS	0.5±0.1	0.7±0.5	NS	0.4±0.1	0.6±0.2	0.017

Continuous data are presented as mean±SD.

AHI, apnoea–hypopnoea index; BMI, body mass index; ESS, Epworth sleepiness scale score; MWT, maintenance of wakefulness test; NS, not significant.