International Journal of Current Research in Medical Sciences
ISSN: 2454-5716
(A Peer Reviewed, Indexed and Open Access Journal)
www.ijcrims.com

Original Research Article

Volume 7, Issue 6 -2021

DOI: http://dx.doi.org/10.22192/ijcrms.2021.07.06.005

Chronic Biomass Smoke Exposure Elicits Platelet-Driven Leukocyte Activation and Enhanced Circulating Leukocyte–Platelet Aggregate Formation in Rural Indian Women

Madhuchanda Banerjee*
Post Graduate Department of Zoology, Midnapore College (Autonomous), P.O. Midnapore, Dist. Paschim Medinipur, West Bengal, INDIA, PIN-721 101
E-mail: *madhuchanda.banerjee@gmail.com*
*Corresponding Author

Abstract

Household combustion of traditional biomass fuels remains a major source of sustained particulate exposure in low-resource settings, particularly for women engaged in daily cooking. This proceedings paper presents an adapted account of a study that examined whether long-term biomass smoke exposure is accompanied by measurable thrombo-inflammatory alterations in circulating blood cells. A total of 320 rural women from eastern India were evaluated, including 165 long-term biomass users and 155 women cooking with liquefied petroleum gas. Indoor particulate concentrations, routine hematological indices, platelet activation, leukocyte integrin expression, and circulating leukocyte–platelet aggregates were assessed, primarily by whole-blood flow cytometry. Kitchens using biomass showed markedly higher PM10 and PM2.5 levels than LPG kitchens. Compared with controls, biomass-exposed women demonstrated increased white blood cell, neutrophil, monocyte, eosinophil, and platelet counts; elevated CD11b and CD18 expression on polymorphonuclear leukocytes and monocytes; higher proportions of CD62P-positive platelets; and substantially greater frequencies of platelet-associated leukocyte aggregates. The strongest independent associations after adjustment for tobacco chewing and environmental tobacco smoke were observed for CD11b upregulation on polymorphonuclear leukocytes and monocytes and for platelet CD62P expression. Taken together, the findings support the interpretation that chronic biomass smoke exposure is linked to systemic platelet and leukocyte activation, with potential implications for thrombosis-related cardiovascular risk in exposed women.

Keywords: biomass smoke; household air pollution; platelet activation; CD11b/CD18; CD62P; leukocyte–platelet aggregates; flow cytometry

Introduction

Household air pollution from biomass combustion remains one of the most persistent environmental health challenges in developing regions. In rural Indian households, the use of wood, dung, crop residue, and related unprocessed solid fuels often occurs in kitchens with poor ventilation and without smoke exhaust, creating repeated exposure to high particulate loads during daily cooking.[1-2] Women, who usually spend the most time near the combustion source, therefore experience the greatest cumulative exposure burden.[5-10]

Biomass smoke contains very high concentrations of fine particulate matter (PM). In a typical Indian household, the concentration of PM10 (PM 10 mm in diameter) in the kitchen varies between 500 and

2000 mg/m3 during the hours of cooking,[2] which is several-fold higher than the air quality standard recommended by the US Environmental Protection Agency.[3] Besides morbidity, indoor air pollution from biomass burning in developing countries is believed to be responsible for an estimated 2.2-2.5million premature deaths every year.[4] Over 600 000 deaths/year occur in India alone that can be attributed to biomass fuel use.[5] Although the pulmonary consequences of biomass smoke are well recognized, its systemic vascular and hematologic effects have received comparatively less attention. Airborne particulate exposure has been linked to platelet reactivity, altered blood rheology, endothelial perturbation, and inflammatory signaling, all of which can contribute to cardiovascular risk. Within this context, the interaction between activated platelets and leukocytes is particularly important. Platelet P-selectin facilitates early adhesion, while leukocyte CD11b/CD18 supports firmer cell-cell interactions and promotes inflammatory and procoagulant responses.[11-15]

The present work focuses on finding out the association between chronic exposure to biomass smoke with a circulating thrombo-inflammatory phenotype in rural women. Specifically, the study evaluated platelet activation, expression of CD11b/CD18 on polymorphonuclear leukocytes and monocytes, and the abundance of circulating leukocyte–platelet aggregates in comparison with women using LPG as a cleaner domestic fuel.

Materials and Methods

This study enrolled 320 women between 21 and 60 years of age from villages in West Bengal, India, during community health camps conducted between August 2003 and July 2004. The exposed group comprised 165 women who had cooked for at least five years with biomass fuels such as wood, cow dung, bamboo, jute stick, paddy husk, hay, and dried leaves, typically for 3–5 hours each day. The comparison group included 155 women using liquefied petroleum gas. Eligibility criteria required active engagement in household cooking, non-smoking status, and body mass index within the specified study range.

The inclusion criteria were: actively engaged in daily household cooking for the past 5 years or more, age not less than 20 and 60 years, non-smoker, and body mass index greater than15 and less than 40 kg/m^2.

Women who were pregnant, breastfeeding, using oral contraceptives, or had major current or prior illnesses such as tuberculosis, asthma, cardiovascular disease, or malignancy were excluded. The protocol received ethics approval from the Chittaranjan National Cancer Institute. The characteristics of the study population and controls are summarized in Table 1.

Table 1. Demographic and socio-economic characteristics of the participants

Variable	Control (n=155)	Biomass user (n=165)	P value*
Median age in year (range)	39 (23–57)	37 (21–60)	>0.05
Body mass index (kg/m²)	22.7	22.4	>0.05
Median years of schooling (range)	8 (2–17)	8 (0–15)	>0.05
Marital status (%)			
Married	91	93	>0.05
Widow	4	3	
Unmarried	5	4	
Median cooking years (range)	20 (5–36)	22 (5–41)	>0.05
Median hours of cooking per day (range)	3.4 (2.0–4.5)	3.8 (2.5–5.5)	>0.05
Homes with separate kitchen (%)	67	61	
Smoking habit (%)			
Current smoker	0	0	>0.05
Ex-smoker	0	0	
Never-smoker	100	100	
Smoker in family (%)	52	68	<0.05
Tobacco/betel quid chewing habit (%)	17	27	<0.05
Food habit (%)			
Vegetarian	5	8	>0.05
Mixed	95	92	>0.05
Mean number of members in family (range)	5 (2–7)	5 (2–9)	>0.05
Average family income per month (US$)	38	29	<0.05

**Statistically analysed by χ^2-test.*

Kitchen air quality was assessed in a subset of households with a portable laser photometer configured to measure PM10 and PM2.5. Monitoring was performed over three consecutive days for eight hours per day and covered both cooking and non-cooking periods. The instrument was positioned within the breathing zone of the cook to approximate personal exposure during meal preparation.

Blood was collected in the morning on the third monitoring day, processed rapidly, and analyzed for hematological indices and cellular activation markers. Total and differential leukocyte counts and platelet counts were obtained using standard microscopy-based methods[16-17]. Whole-blood flow cytometry was then used to quantify CD11b and CD18 expression on polymorphonuclear leukocytes and monocytes, platelet CD41 expression, and platelet CD62P as an indicator of

platelet activation. Leukocyte–platelet aggregates were identified by the co-expression of leukocyte marker CD11b with platelet-associated CD41 or CD62P.[18-19] Approximately 10,000 events were acquired per assay. Statistical comparisons were performed using the chi-square test and Student's t-test, while logistic regression was used to examine associations after accounting for tobacco chewing and environmental tobacco smoke.

Results

The exposure contrast between the two groups was pronounced. During cooking periods, biomass-using kitchens showed mean PM10 and PM2.5 concentrations of 625 and 312 µg/m3, respectively, whereas LPG kitchens showed corresponding values of 169 and 77 µg/m3. Even outside cooking hours, biomass-using kitchens maintained substantially higher particulate levels. These findings confirmed a markedly heavier inhalation burden among biomass users.

Group characteristics were broadly similar with respect to age, body mass index, schooling, marital status, cooking history, smoking status, and family size. However, women using biomass had greater exposure to passive smoking, a higher prevalence of tobacco or betel quid chewing, and lower household income. Health complaints and spirometric impairment were also more frequent in the biomass-exposed group (Table 2).

Table 2. Prevalence (%) of general health problems among LPG and biomass users

Parameter	LPG user (n=155)	Biomass user (n=165)	P value in χ^2-test
Upper respiratory symptoms	8.4	17.0	<0.05
Lower respiratory symptoms	5.2	12.7	<0.05
Lung function impairment	20.0	49.7	<0.001
Hemoglobin <10 g/dL	4.5	10.3	<0.05
Diarrhea and dysentery	7.7	26.7	<0.001
Headache	16.1	43.0	<0.001
Eye irritation or tears while cooking	4.5	17.6	<0.001
Chest pain or chest tightness	2.5	15.1	<0.001
Hypertension	3.2	6.0	<0.05

Routine hematology revealed significantly higher white blood cell, neutrophil, monocyte, eosinophil, and platelet counts in biomass users, while hemoglobin, erythrocyte count, and lymphocyte count did not differ significantly (Table 3). Flow-cytometric analysis further showed clear activation-related changes. Mean fluorescence intensity of CD11b increased by 68% on polymorphonuclear leukocytes and by 50% on monocytes in biomass users. CD18 expression rose by 48% on polymorphonuclear leukocytes and by 62% on monocytes. Platelet CD41 did not change significantly, but CD62P-positive platelets increased sharply in the biomass group, indicating enhanced platelet activation (Table 4).

Table 3. Hematological changes

Parameters	Control (n=155)	Biomass user (n=165)	% Change over control
Hemoglobin (g/dL)	12.8 ± 0.2	12.5 ± 0.2	−2
RBC (×10⁶/mL)	4.2 ± 0.3	4.0 ± 0.2	−5
WBC (×10³/mL)	7.5 ± 0.4	8.9 ± 0.4*	19
Platelet (×10⁵/mL)	2.3 ± 0.3	3.9 ± 0.3*	70
Neutrophil (×10³/mL)	4.1 ± 0.2	5.3 ± 0.2*	29
Eosinophil (×10³/mL)	0.3 ± 0.02	0.8 ± 0.05*	167
Lymphocyte (×10³/mL)	2.6 ± 0.1	2.4 ± 0.1	−8
Monocyte (×10³/mL)	0.2 ± 0.01	0.3 ± 0.01*	50

**P < 0.05 compared with control in Student's t-test. Results are mean ± SD.*

A parallel increase was observed in circulating leukocyte–platelet aggregates. Among polymorphonuclear leukocytes, both CD11b/CD41-positive and CD11b/CD62P-positive aggregates were significantly more abundant in biomass users. Similar increases were documented for monocyte–platelet aggregates. Logistic regression suggested that biomass use remained independently associated with higher CD11b expression on polymorphonuclear leukocytes and monocytes and with higher platelet CD62P expression. By contrast, the associations involving CD18 and aggregate counts appeared more sensitive to the influence of confounding factors such as environmental tobacco smoke and tobacco chewing.

Table 4. CD11b/CD18 expression on peripheral blood leukocytes

Marker / cell type	Control (n=155)	Biomass user (n=165)	% Change over control
CD11b			
Monocyte % of positive cells	98.8 ± 1.1	100 ± 0	1
Monocyte MFI	697.2 ± 87.9	1047.8 ± 171.2*	50
PMN % of positive cells	100 ± 0	100 ± 0	0
PMN MFI	683.5 ± 85.9	1150.2 ± 143.2*	68
CD18			
Monocyte % of positive cells	97.6 ± 2.4	99.5 ± 2.4	2
Monocyte MFI	412.9 ± 78.8	670.4 ± 89.8*	62
PMN % of positive cells	100 ± 0	100 ± 0	0
PMN MFI	385.1 ± 56.4	568.7 ± 64.4*	48

**P < 0.05 compared with control in Student's t-test. Results are mean ± SD; MFI, mean fluorescence intensity; PMN, polymorphonuclear leukocyte.*

Discussion

The findings support the view that chronic biomass smoke exposure is not limited to the respiratory compartment but extends to the circulation, where it is associated with a pattern of platelet and leukocyte activation consistent with a thrombo-inflammatory response. Upregulation of platelet CD62P implies enhanced platelet reactivity, while increased leukocyte CD11b/CD18 expression points to an activated adhesive phenotype capable of strengthening interactions with platelets and endothelium.[20-25] The concomitant rise in leukocyte–platelet aggregates is biologically meaningful because these mixed cellular complexes have been linked in other clinical settings to vascular inflammation and thrombotic risk.

Mechanistically, repeated exposure to biomass-derived particulate matter may promote systemic effects through oxidative stress, inflammatory mediator release, and possibly direct translocation of smaller particles across the alveolar-capillary barrier.[26-31] The markedly higher PM10 and PM2.5 concentrations in biomass kitchens provide a plausible exposure basis for the hematologic and immunologic perturbations observed here. In addition, the elevated platelet count and higher leukocyte counts may further amplify opportunities for cell-cell interaction in circulation.[32]

At the same time, the data should be interpreted with caution. The study design was observational and cross-sectional, which precludes causal inference. The biomass group differed from controls in passive smoke exposure, chewing habits, and socioeconomic status, all of which may influence inflammatory biomarkers. Indeed, the regression analysis indicates that these factors likely contributed to part of the signal, particularly for CD18 expression and leukocyte–platelet aggregate formation. Accordingly, the results are best regarded as strong evidence of association and biologic plausibility rather than definitive proof of a direct causal pathway.

Even with these limitations, the study remains important because it broadens the understanding of household air pollution from a respiratory problem to a systemic vascular concern. In populations where biomass fuel use is common, chronic exposure may contribute to cardiovascular vulnerability through persistent activation of circulating blood cells.[33-36] This interpretation strengthens the public health case for cleaner domestic energy, improved kitchen ventilation, and more detailed biomarker-based studies in exposed communities.

Conclusion

Women chronically exposed to biomass smoke during household cooking exhibited higher particulate exposure, increased leukocyte and platelet counts, upregulated CD11b/CD18 expression on circulating leukocytes, greater platelet CD62P expression, and more frequent leukocyte–platelet aggregates than women cooking with LPG. Collectively, these findings are consistent with a systemic thrombo-inflammatory state associated with long-term household biomass smoke exposure and support the need for interventions that reduce exposure among women in rural settings.

Acknowledgements

The authors thank the Department of Environment, Government of West Bengal, for financial assistance in carrying out the study.

References

1. IIPS (International Institute for Population Sciences). National Family Health Survey (MCH and Family Planning): India 1992–93. Bombay: International Institute for Population Sciences, 1995.
2. Balakrishnan K, Sankar S, Parikh J, Padmavati R, Srividya K, Venugopal V, Prasad S, Pandey VL. Daily average exposures to respirable particulate matter from combustion of biomass fuels in rural households of southern India. Environ Health Perspect 2002; 110: 1069–75.
3. US EPA. US Environmental Protection Agency National Ambient Air Quality Standards (NAAQS). 15 August 2002. Retrieved from http://www.epa.gov/airs/riteria.html
4. WHO (World Health Organization). Health and environment for sustainable development. World Health Organization, 1997.
5. Smith KR. Indoor air pollution in developing countries: recommendations for research. Indoor Air 2002, 12: 198–207.
6. Schwartz J. Air pollution and blood markers of cardiovascular risk. Environ Health Perspect 2001; 109: 405–409.
7. Donaldson K, Stone V, Seaton A, MacNee W. Ambient particle inhalation and the cardiovascular system: potential mechanisms. Environ Health Perspect 2001; 109: 523–27.
8. Falanga A, Marchetti M, Evangelista V, Manarini S, Oldani E, Giovanelli S, Galbusera M, Cerletti C, Barbui T. Neutrophil activation and hemostatic changes in healthy donors receiving granulocyte colony-stimulating factor. Blood 1999; 93: 2506–14.
9. Celi A, Pellegrini G, Lorenzet R, De Blasi A, Ready N, Furie BC, Furie B. P-selectin induces the expression of tissue factor on monocytes. Proc Natl Acad Sci USA 1994; 91: 8767–71.
10. Engstad CS, Lia K, Rekdal O, Olsen JO, Osterud B. A novel biological effect of platelet factor 4 (PF4): enhancement of LPS-induced tissue factor activity in monocytes. J Leukoc Biol 1995; 58: 575–81.
11. Nagata K, Tsuji T, Todoroki N, Katagiri Y, Tanoue K, Yamazaki H, Hanai N, Irimura T. Activated platelets induce superoxide anion release by monocytes and neutrophils through P-selectin (CD 62). J Immunol 1993; 151: 3267–73.
12. Weyrich AS, Elstad MR, McEver RP, McIntyre TM, Moore KL, Morrissey JH, Prescott SM, Zimmerman GA. Activated platelets signal chemokine synthesis by human monocytes. J Clin Invest 1996; 97: 1525–34.
13. Neumann F-J, Marx N, Gawaz M, Brand K, Ott I, Rokitta C, Sticherling C, Meinl C, May A, Schomig A. Induction of cytokine expression in leukocytes by binding to thrombin-stimulated platelets. Circulation 1997; 95: 2387–94.
14. Ott I, Neumann F-J, Gawaz M, Schmitt M, Schomig A. Increased neutrophil-platelet adhesion in patients with unstable angina. Circulation 1996; 94: 1239–46.
15. Entmann ML, Ballantyne CM. Association of neutrophil with platelet aggregates in unstable angina. Should we alter therapy? Circulation 1996; 94: 1206–208.
16. Michelson AD, Barnard MR, Krueger LA, Valeri CR, Furman MI. Circulating monocyte–platelet aggregates are a more sensitive marker of in vivo platelet activation than platelet surface P-selectin: studies in baboons, human coronary intervention and human acute myocardial infarction. Circulation 2001; 104: 1533–37.
17. Konstantopoulos K, Grotta JC, Sills C, Wu KK, Hellums JD. Shear-induced platelet aggregation in normal subjects and stroke patients. Thromb Haemost 1995; 74: 1329–34.
18. Michelson AD, Barnard MR, Krueger LA, Frelinger AL III, Furman MI. Evaluation of platelet function by flow cytometry. Methods 2000, 21: 259–70.
19. Li N, Goodall AH, Hjemdahl P. A sensitive flow cytometric assay for circulating platelet–leukocyte aggregates. Br J Haematol 1997; 99: 808–16.

20. de Gaetano G, Cerletti C, Evangelista V. Recent advances in platelet–polymorphonuclear leukocyte interaction. Haemostasis 1999; 29: 41–49.
21. Jensen MK, de Nully Brown P, Lund BV, Nielson OJ, Hasselbalch HC. Increased platelet activation and abnormal membrane glycoprotein content and redistribution in myeloproliferative disorders. Br J Haematol 2000; 110: 116–24.
22. Gutensohn K, Geidel K, Brockmann M, Siemensen M, Krueger W, Kroeger N, Kuehnl P. Binding of activated platelets to WBCs in vivo after transfusion. Transfusion 2002; 42: 1373–80.
23. Frijns CJM, Kappelle IJ, van Gijn J, Nieuwenhuis HK, Sixma JJ, Fijnheer R. Soluble adhesion molecule reflect endothelial cell activation in ischemic stroke and in carotid atherosclerosis. Stroke 1997; 28: 2214–18.
24. Griesshammer M, Beneke H, Nussbaumer B, Grunewald M, Bangerter M, Bergmann L. Increased platelet surface expression of P-selectin and thrombospondin as markers of platelet activation in essential thrombocythemia. Thromb Res 1999; 96: 191–96.
25. Falanga A, Marchetti M, Evangelista V, Vignoli A, Licini M, Balicco M, Manarini S, Finazzi G, Cerletti C, Barbui T. Polymorphonuclear activation and hemostasis in patients with essential thrombocythemia and polycythemia vera. Blood 2000; 96: 4261–66.
26. Alvarez-Larran A, Garca-Pagan JC, Abraldes JG, Arellano E, Reverter JC, Bosch J, Cervantes F. Increased CD11b neutrophil expression in Budd-Chiari syndrome or portal vein thrombosis secondary to polycythemia vera. Br J Haematol 2004; 124: 329–35.
27. Joseph JN, Harrison P, Mackie IJ, Isenberg DA, Machin SJ. Increased circulating platelet–leukocyte complexes and platelet activation in patients with antiphospholipid syndrome, systemic lupus erythromatosus and rheumatoid arthritis. Br J Haematol 2001; 115: 451–59.
28. Rinder CS, Bonan JL, Rinder HM, Mathew J, Hines R, Smith BR. Cardiopulmonary bypass induces leukocyte–platelet adhesion. Blood 1992; 79: 1201–205.
29. Ellegard A. Cooking smoke and respiratory symptoms among women in low-income areas of Maputo. Environ Health Perspect 1996; 104: 980–85.
30. Zhang J, Morawska L. Combustion sources of particles: 2. Emission factors and measurements methods. Chemosphere 2002; 49: 1059–74.
31. Nemmar A, Hoet PH, Vanquickenborne B, Dinsdale D, Thomeer M, Hoylaerts MF, Vanbilloen H, Mortelmans L, Nemery B. Passage of inhaled particles into the blood circulation in humans. Circulation 2002; 105: 411–14.
32. Arnaout MA. Structure and function of the leukocyte adhesion molecules CD11/CD18. Blood 1990; 75: 1037–50.
33. Kishimoto TK, Rothlein R. Integrins, ICAMs, and selectins: role and regulation of adhesion molecules in neutrophil recruitment to inflammatory sites. Adv Pharmacol 1994; 25: 117–69.
34. Parkos CA. Molecular events in neutrophil transepithelial migration. Bioessays 1997; 19: 865–73.
35. Smith CW, Marlin SD, Rothlein R, Toman C, Anderson DC. Cooperative interactions of LFA-1 and Mac-1 with intercellular adhesive molecule-1 in facilitating adherence and transendothelial migration of human neutrophils in vitro. J Clin Invest 1989; 83: 2008–17.

36. Fogelstrand L, Hulthe J, Hulten LM, Wiklund O, Fagerberg B. Monocytic expression of CD14 and CD18, circulating adhesion molecules and inflammatory markers in women with diabetes mellitus and impaired glucose tolerance. Diabetologica 2004; 47: 1948–52.

Access this Article in Online	
Quick Response Code	Website: www.ijcrims.com
	Subject: Haematology

How to cite this article:
Madhuchanda Banerjee. (2021). Chronic Biomass Smoke Exposure Elicits Platelet-Driven Leukocyte Activation and Enhanced Circulating Leukocyte–Platelet Aggregate Formation in Rural Indian Women. Int. J. Curr. Res. Med. Sci. 7(6): 24-32.
DOI: http://dx.doi.org/10.22192/ijcrms.2021.07.06.005