



## Acute cigarette smoke exposure induces oxidative damage and inflammation in Wistar rats: impact on lungs and erythrocytes

Shahdat Hossain<sup>1\*</sup>, Marzan Sarkar<sup>2</sup>, Sujan Bhowmick<sup>3</sup>, Jakir Hussain<sup>4</sup>, Mahmudul Hasan<sup>5</sup>, Borhan Uddin<sup>6</sup>, Md. Asaduzzaman Khan<sup>7</sup> and Taslima Nahar<sup>8</sup>

<sup>1</sup>Laboratory of Alternative Medicine and Behavioral Neurosciences, Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka-1342, Bangladesh. E-mail: shahdat@juniv.edu

<sup>2</sup>Laboratory of Alternative Medicine and Behavioral Neurosciences, Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka-1342, Bangladesh. E-mail: marzan.bmbju@gmail.com

<sup>3</sup>Laboratory of Alternative Medicine and Behavioral Neurosciences, Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka-1342, Bangladesh. E-mail: sujan.bmbju@gmail.com

<sup>4</sup>Laboratory of Alternative Medicine and Behavioral Neurosciences, Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka-1342, Bangladesh. E-mail: jakir.bmbju@gmail.com

<sup>5</sup>Laboratory of Alternative Medicine and Behavioral Neurosciences, Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka-1342, Bangladesh. E-mail: mohsin.bmbju@gmail.com

<sup>6</sup>Laboratory of Alternative Medicine and Behavioral Neurosciences, Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka-1342, Bangladesh. E-mail: borhan-bmb@juniv.edu

<sup>7</sup>Key Laboratory of Epigenetics and Oncology, the Research Center for Preclinical Medicine, Southwest Medical University, Luzhou, Sichuan-646000, China. E-mail: asadkhan@swwu.edu.cn

<sup>8</sup>Laboratory of Alternative Medicine and Behavioral Neurosciences, Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka-1342, Bangladesh. E-mail: taslima\_bmb@juniv.edu

### Article Info

Volume 3, Issue 1, January 2021

Received : 21 July 2020

Accepted : 03 October 2020

Published : 01 January 2021

doi: [10.33472/AFJBS.3.1.2021.120-128](https://doi.org/10.33472/AFJBS.3.1.2021.120-128)

### Abstract

Smoking of cigarettes worsens morbidity and mortality rates by prompting cancer, pulmonary, cardiovascular, and oral diseases. Despite having all these severe risks of smoking, the habit is unacceptably widespread, particularly among teenagers in many countries, including Bangladesh. In this investigation, the acute effect of cigarette smoking on oxidative stress and inflammation (Tumor Necrosis Factor- $\alpha$  (TNF- $\alpha$ )) has been analyzed in the cigarette smoke (CS)-exposed rats. Exposure of smoke *in vivo* significantly increased the levels of Lipid Peroxide (LPO) and TNF $\alpha$  in the experimental rats, when compared to those of the control rats. Furthermore, washed erythrocytes isolated from control rats were exposed directly to CS *in vitro*. The time-dependent exposure of smoke to erythrocytes increased the oxidative stress in the erythrocytes, as indicated by increased levels of LPO in the erythrocytes. The toxic effect of acute cigarette smoke was also visible in the morphological studies of erythrocytes. Cigarette smoke-exposed erythrocytes revealed numerous leakage, breaks and membrane blobs in the plasma membrane of erythrocytes. Our results thus suggest that cigarette smoke not only affects the lung tissues but may also deteriorate the morphology of erythrocytes by instigating the oxidative stress in the erythrocytes.

**Keywords:** Cigarette smoke (CS), Lung, Erythrocyte, Lipid Peroxidation (LPO), Tumor Necrosis Factor- $\alpha$  (TNF- $\alpha$ ).

© 2021 African Journal of Biological Sciences. This is an open access article under the CC BY license (<https://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made.

\* Corresponding author: Shahdat Hossain, Laboratory of Alternative Medicine and Behavioral Neurosciences, Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka-1342, Bangladesh. E-mail: shahdat@juniv.edu

## 1. Introduction

Chronic cigarette smoke exposure is well-known for its detrimental effects on lung and erythrocyte. Information on the acute effect of cigarette smoke is very little comparing to that effect of chronic cigarette smoke exposure. An acute smoking model is a reasonably easier and more sensitive method of scrutinizing the specific effects of cigarette smoke on oxidative stress and inflammatory response.

Cigarette smoke is a source of Reactive Oxygen Species (ROS) such as superoxide anions, hydroxyl radicals, hydrogen peroxide, metallic ions and quinones. These oxidizing agents can upsurge the levels of oxidative stress in the smokers (Pryor and Stone, 1993; and Marnett et al., 2003) with increased Lipid Peroxides (LPO) contents and degradation products of extracellular matrix proteins (Van der Vaar et al., 2004). ROS present in cigarette smoke are responsible for cellular injury and inflammation (Khan et al., 2014).

The lung is the primary organ exposed to cigarette smoke, and numerous experimental and clinical evidences have shown that nicotine induces an inflammatory response in the lung (Carpagnano et al., 2003). Tumor Necrosis Factor- $\alpha$  (TNF- $\alpha$ ), a prominent pro-inflammatory cytokine, appears to be an important pathogenic factor in the development of disease, and is known to mediate a wide range of biological activities (Vassalli, 1992; and Kollias et al., 1999). A prominent risk factors for the development of these TNF- $\alpha$ -mediated diseases is cigarette smoking (Chang et al., 2014; and Karczewski et al., 2014). Nicotine is quickly absorbed into the circulatory system during smoking and concentration of blood nicotine increases rapidly. The fast absorption of nicotine from cigarette smoke via the lung, maybe due to the huge surface region of the alveoli and trivial airways, and suspension of nicotine in the fluid of pH 7.4 in the lung, facilitates transfer through the membranes (Grozio et al., 2007). Smoking can cause deadly lung infections like pneumonia, emphysema and lung cancer. In addition, adverse effects of parental smoking on the respiratory health of children have been a clinical and public health concern for decades. For children, exposure to secondhand smoke may lead to respiratory illnesses as a result of adverse effects on the immune system and on lung growth and development (Office on Smoking and Health, US, 2006).

The breakage of erythrocyte membrane called hemolysis, triggers the hemoglobin release and other internal constituents into the neighboring fluid or plasma (Ko et al., 1997). Nicotine exposure significantly increases the oxidative stress by generating called ROS (Ben Ahmed et al., 2010; and Yildiz et al., 1998). Oxidative damage and hemolysis caused by ROS have a major role in the expansion of diseases such as thalassemia, glucose-6-phosphate dehydrogenase deficiency, and sickle cell anemia. RBCs are extremely susceptible to oxidative damage as a result of the high polyunsaturated fatty acids (PUFA) content of their membranes and high cellular oxygen and hemoglobin (Hb) concentrations. Oxidation depletes membrane protein content, deforms erythrocytes inappropriately, and interrupts microcirculation (Yang et al., 2006; Evans et al., 1986; Yu, 2001; and Flynn et al., 1983). Carbon monoxide (CO) and nicotine from the smoke increase the risk of blood clots. Other chemicals in cigarette smoke injure the lining of coronary arteries, leading to atherosclerosis and finally to heart attack.

The present study aimed at characterizing the oxidative status, inflammatory response and the structural features of the lung and erythrocytes after acute cigarette smoke exposure.

## 2. Materials and methods

### 2.1. Chemicals

Bovine Serum Albumin (BSA), hydrogen peroxide ( $H_2O_2$ ),  $FeSO_4$  and 1, 1, 3, 3-Tetraethoxypropane (TEP) were procured from Sigma Aldrich (St. Louis, USA). TNF $\alpha$  was purchased from Santa Cruz Biotechnology, CA, USA. Horse Radish Peroxidase (HRP)-conjugated anti-rabbit secondary antibody was purchased from Cell Signaling Technology (CST, USA). The primary antibody anti-rabbit TNF $\alpha$  was purchased from Santa Cruz Biotechnology, USA. Tetramethylbenzidine was purchased from Invitrogen, Life Technologies, USA. ELISA grade BSA was from Sigma Aldrich. All other chemicals were of analytical grade.

### 2.2. Animals

Five-week-old male Wistar rats were purchased from ICDDR,B (Dhaka, Bangladesh), and were housed in an air-conditioned animal room with a 12:12 h dark: light cycle under controlled temperature ( $23 \pm 2$  °C) and humidity ( $50 \pm 10\%$ ). The rats were provided with a normal pellet diet with water ad libitum. All animal experiments were executed in accordance with the procedures outlined in the Guidelines for Animal

Experimentation of Jahangirnagar University compiled from the Guidelines for Animal Experimentation of the Bangladesh Association for Laboratory Animal Science.

### **2.3. Experimental design**

Five rats were confined at a time in a smoking chamber for two periods of 30 min twice daily for two consecutive weeks. Another five rats in the control group were confined in a similar chamber without smoke. Then after sacrifice, the effect of smoke on the lung, plasma and erythrocytes were examined.

### **2.4. Erythrocyte preparation**

Erythrocytes were prepared as defined by Hashimoto *et al.* (2015). After deep anesthesia with pentobarbital, rat's blood was collected from the inferior vena cava using heparinized syringe. Part of the blood was used for the purpose of plasma collection and the other part was mixed with Locke's solution (154 mM NaCl, 5.6 mM KCl, 2.3 mM CaCl<sub>2</sub>, 1 mM MgCl<sub>2</sub>, 3.6 mM NaHCO<sub>3</sub>, 5 mM glucose, and 5 mM HEPES; pH 7.20) and pelleted at 300 × g for 10 min in tubes. Supernatant was excluded and the erythrocytes were washed thrice by using the same Locke's solution. The buffy coat and upper portion of layer of the erythrocytes were removed in every wash. The remaining erythrocytes were immediately subjected to hemolysis and/or used for preparation of erythrocyte ghost membranes. Erythrocytes from the control rats were directly exposed to cigarette smoke for 15, 30 and 45 min, and the effect of smoke on the degree of hemolysis, levels of LPO was determined. Morphological examinations of the smoke-exposed erythrocytes were also conducted.

### **2.5. Hemolysis assay**

Hemolysis assay was performed as described by Hossain *et al.* (2015) with slight modification. Erythrocyte suspensions at 2% hematocrit in Locke's solution (mentioned earlier) from every rat of control and,—smoke exposed group as well as from *in vitro* direct smoke exposure were subjected to incubation for 1 h at 37 °C with freshly prepared Fenton's reagents [H<sub>2</sub>O<sub>2</sub> (45 mM)+ FeSO<sub>4</sub> (2 mM)]. Then, erythrocytes were settled down by centrifuging the samples at 300 × g for 10 min. The supernatant was aspirated and the degree of hemolysis was measured by determining the concentration of released hemoglobin (Hb) into the supernatant at 540 nm against Hb standard.

### **2.6. Erythrocyte morphology test**

Blood samples were obtained from *in vitro* direct exposure to smoke and from control blood group. The red blood cells were examined under light microscope (Eclipse 200, 100X magnification) for variations for morphological features e.g., erythrocyte's size, color, shape, etc.

### **2.7. Preparation of erythrocyte ghost membranes**

Washed erythrocytes were suspended in 40 volumes of ice-cold 5 mM Tris-HCl buffer (pH 7.0), containing 1 mM EDTA, and centrifuged (10000 × g, 60 min, 4 °C). Supernatant was discarded and washing was repeated until the erythrocyte membranes appeared whitish. The membranes were stored at –80 °C.

### **2.8. Preparation of lung homogenate**

After drawing blood, the lung from each rat was separated, perfused with ice cold saline. Afterwards, a 10% lung tissue homogenate was prepared with phosphate buffer (100 mM, pH 7.4) containing 1% phenylmethylsulfonyl fluoride (PMSF). The homogenate was centrifuged at 1000 × g to remove unbroken tissues and debris and the resultant homogenate was assigned as whole homogenate. A part of the whole homogenate was further spun for 1 h at 10000 × g to prepare cytosolic fraction to measure TNF- $\alpha$  in the lung tissues. The samples were stored at –20 °C until analysis.

### **2.9. Lipid peroxide (LPO) of erythrocyte ghost membranes and lung tissues**

The basal levels of LPO in the erythrocyte membranes and the lung tissues were determined by estimating the Thiobarbituric acid reactive substances (TBARs), as described in our previous publications (Hashimoto *et al.*, 2001; Hossain *et al.*, 2011; and Nahar *et al.*, 2013).

### **2.10. Tumor necrosis factor-alpha (TNF- $\alpha$ ) assay**

The multi well ELISA plate was coated with plasma and/or lung cytosolic fraction in 0.1 M sodium bicarbonate (pH 9.6) at 4 °C overnight and then blocked with 1% BSA in Tris-buffered saline (TBS). Then the plate was

incubated overnight at 4 °C with the primary antibody rabbit anti- TNF- $\alpha$  (1:1000). Horseradish peroxidase (HRP)-coupled anti-rabbit antibody was used as the secondary antibody and incubated for 2 h at room temperature before the addition of tetramethylbenzidine (TMB) substrate to develop color. The reaction was stopped by the addition of 0.1N HCl after incubation for 30 min at room temperature. ELISA wells coated with only 0.1 M carbonate buffer (pH 9.6) was used as blank. The plates were examined with a multi well plate reader (Erba Lisa Scan II, Mannheim, Germany) at 450 nm.

### 3. Statistical analysis

All the data were expressed as mean  $\pm$  SEM (standard error of mean). The significance of difference in means among different groups was determined by one-way analysis of variance (ANOVA), followed by Fisher's PLSD test for post hoc comparisons using Graph Pad Prism software version 5.0. A value of  $p < 0.05$  was considered statistically significant.

## 4. Results

### 4.1. Effect of cigarette smoke on erythrocyte morphology

In smoke exposed erythrocytes many morphological features were found which were different from those of the control erythrocytes. Cigarette smoke-exposed erythrocytes were deformed from the typical discoid shape to abnormal shape displaying numerous leakage, breaks and membrane blobs in the plasma membranes of erythrocytes as indicated in Figure 1.

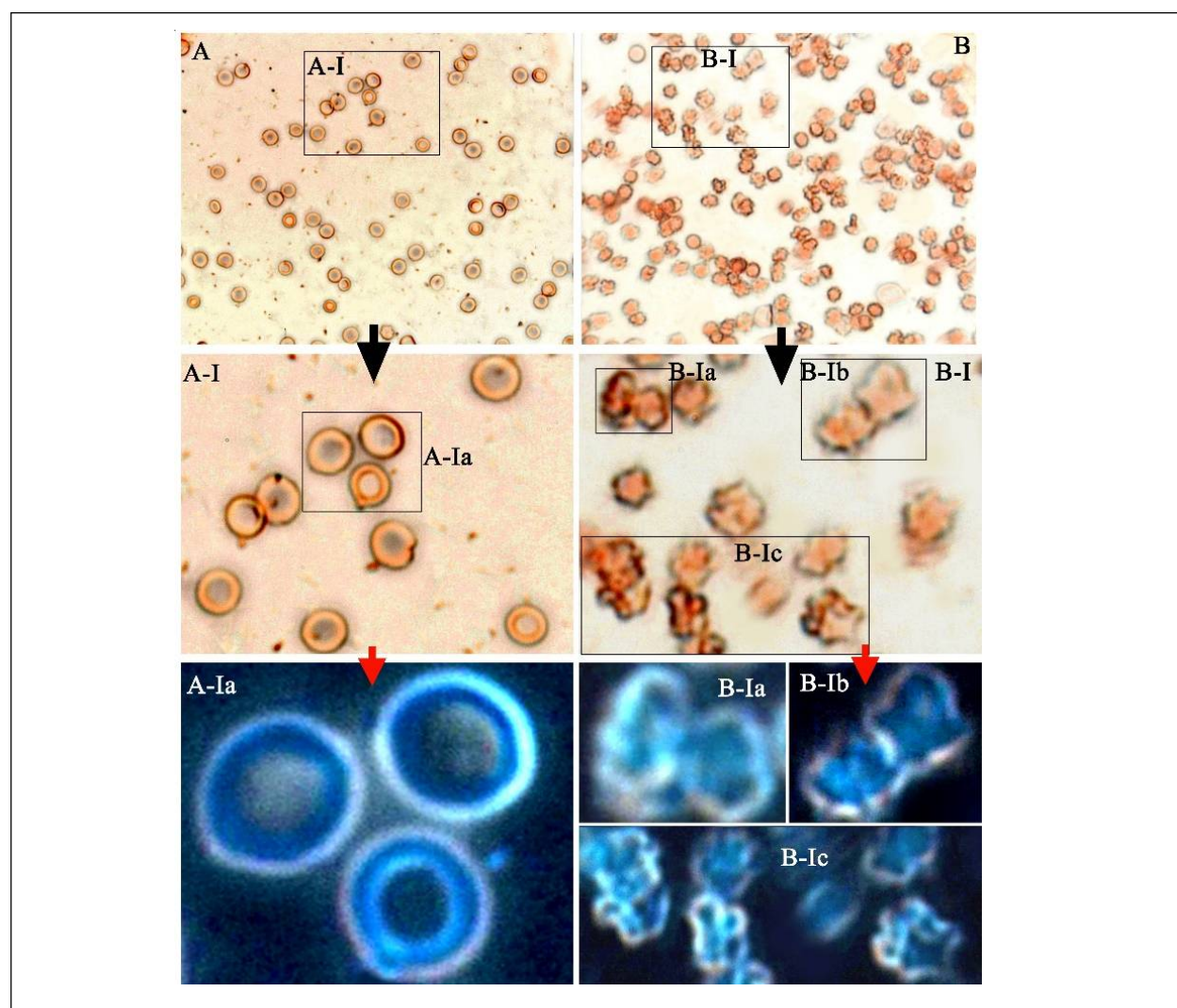


Figure 1: Acute exposure to cigarette smoke deforms erythrocyte morphology. Under the microscope: (A) smoke-unexposed control erythrocytes; and (B) cigarette smoke-exposed erythrocytes (100X magnification)



#### 4.2. Acute cigarette smoke exposure increases hemolysis and lipid peroxidation of erythrocytes *in vitro*

*In vitro* exposure of erythrocytes to smoke increases the level of hemolysis and time-dependent oxidative stress as indicated by the increased extent of LPO in the erythrocytes than those in the un-exposed erythrocytes under experimentation (Figures 2A and 2B). In case of *in vivo* exposure to smoke there is no significant induction on the level of erythrocytes hemolysis between the smoke exposed and the control rat group (data not shown).

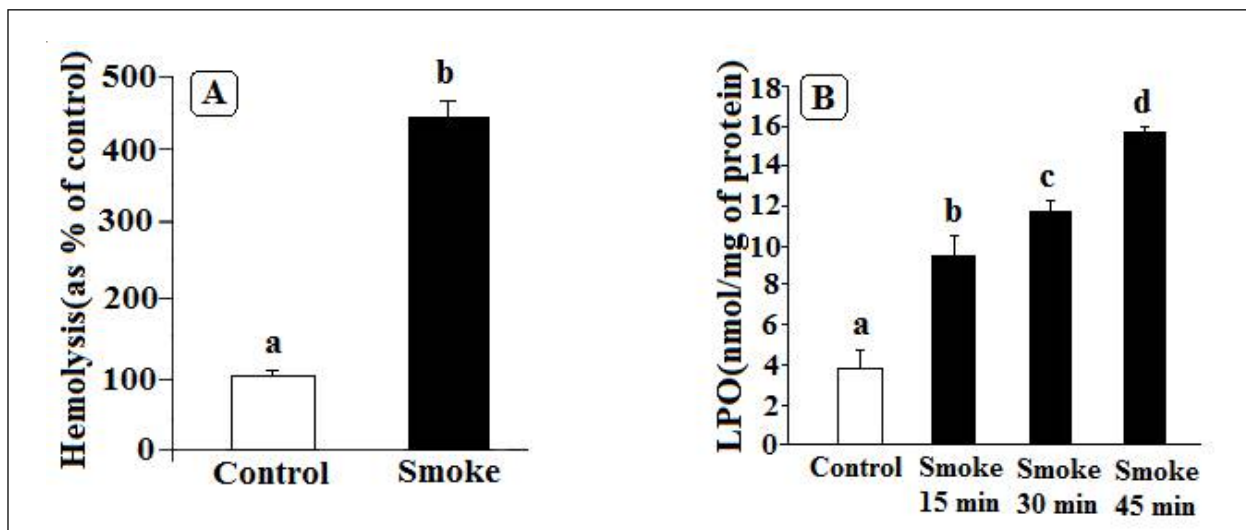


Figure 2: Acute exposure to cigarette smoke causes increased hemolysis and lipid peroxidation *in vitro*. (A) effect of *in vitro* cigarette smoking on hemolysis; and (B) time dependent lipid peroxidation of RBCs. Results are mean  $\pm$  SEM ( $n = 5$ ). Bars with different letters are significantly different at  $p < 0.05$

#### 4.3. Acute cigarette smoke exposure induces lipid peroxidation of erythrocytes and lung tissues *in vivo*

*In vivo* exposure of the rats to smoke significantly increased the levels of LPO in the erythrocytes (Figure 3A) and lung tissues (Figure 3B), when compared to those of the un-exposed control rats.

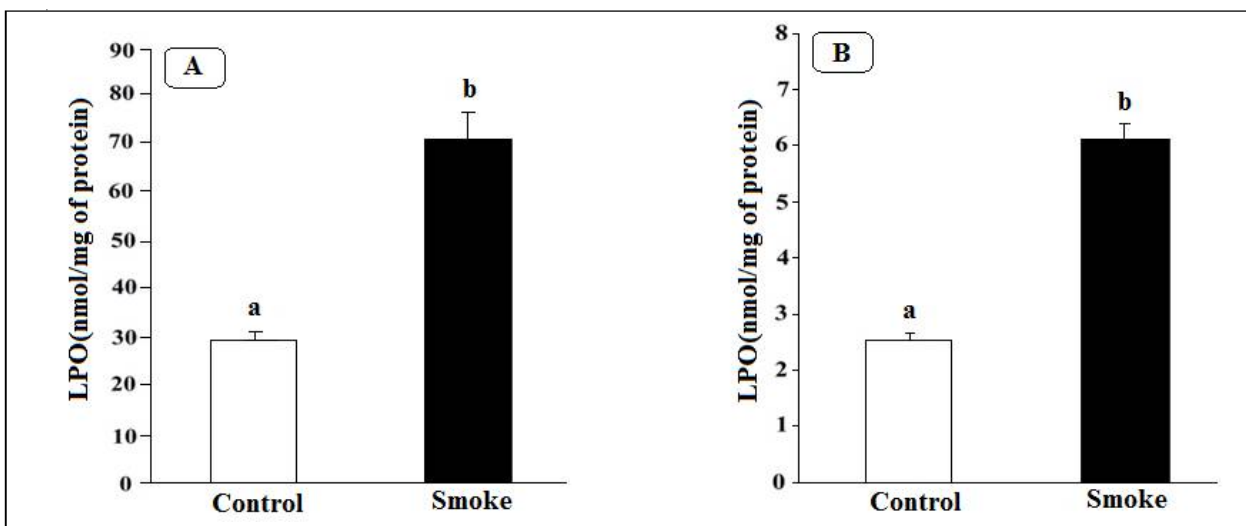
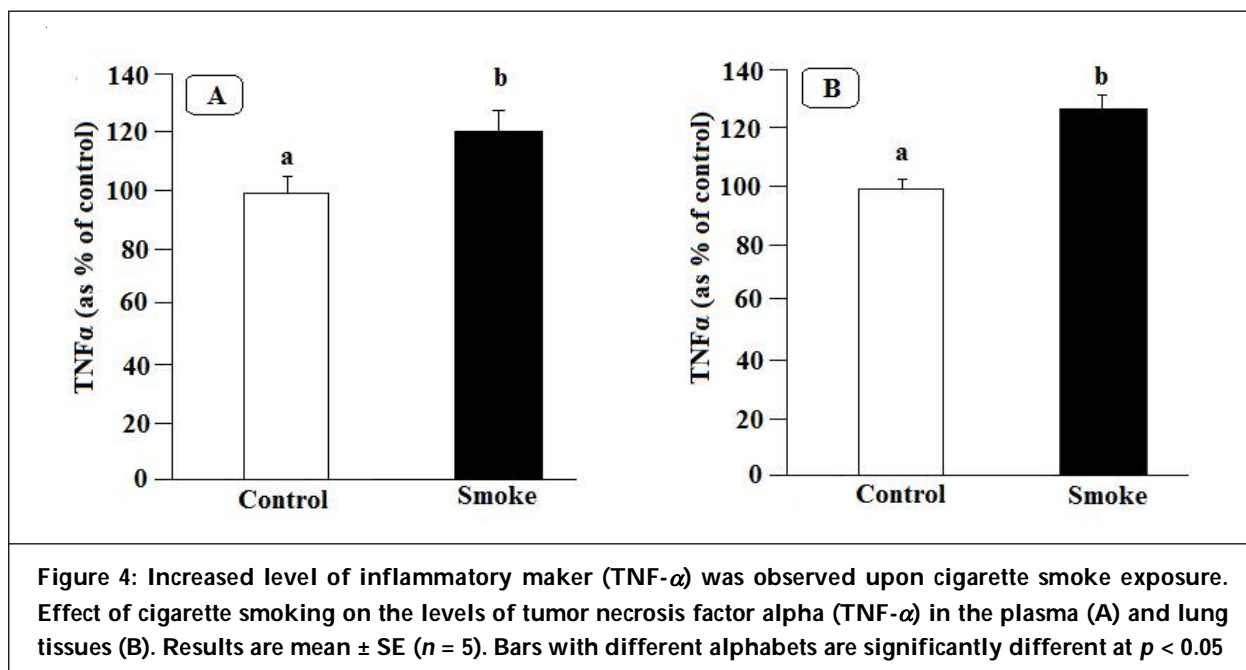


Figure 3: Increased lipid peroxidation was observed in erythrocytes and lung tissues upon cigarette smoke exposure. (A) *in vivo* cigarette smoking on lipid peroxidation of erythrocyte and (B) lung tissues. Results are mean  $\pm$  SEM ( $n = 5$ ). Bars with different alphabets are significantly different at  $p < 0.05$ .

#### 4.4. Acute cigarette smoke exposure increases TNF- $\alpha$ level in plasma and lung tissues

*In vivo* exposure of rats to cigarette smoke significantly increases the level of TNF- $\alpha$  in plasma (Figure 4A) and lung tissues (Figure 4B), when compared to those of the un-exposed control rats.

All these results collectively highlighted the destructive effect of cigarette smoke on lung and erythrocytes which are the risk factors for many fatal diseases including lung cancer and coronary heart diseases.



## 5. Discussion

For better understanding of the chronic outcome of smoking, it is significant to study the acute response to cigarette smoke, since repetitive smoke effects may congregate and eventually lead to irreversible damage.

We, therefore, investigated the acute effects of cigarette smoking on lung and erythrocytes in Wistar rats.

Cigarette smoking is the major avoidable cause of lung cancer in developed and developing countries like Bangladesh and, reportedly,—the second prominent reason of death in today's world. Nicotine is one of the major toxic components of cigarette smoke (WHO, 2007; and Leea et al., 2016) with destructive and malignant consequences (Momi et al., 2013; and Singh et al., 2011). Study shows that cigarette smoking strongly induces apoptosis in the alveolar epithelium (Kwon et al., 2012) and accelerates the progression of pulmonary fibrosis (Sekhon et al., 2004). The principal cause of the chronic obstructive pulmonary disease (COPD), is the reactive oxidants present in cigarette smoke that induces inflammation in the lung and its airways leading to airway narrowing with difficult breathing (Brody and Spira, 2006; and Wang et al., 2018). Acute cigarette smoke experience in humans is associated with increased blood pressure and heart rate, which have potentially injurious effects in patients with heart failure, coronary artery disease, and arrhythmias (Holly et al., 2014). Experimental and clinical proofs have supported the crucial role of nicotine exposure in the pathogenesis of organ disorders by enhancing the oxidative stress (Jung et al., 2001; Kovacic and Cooksy, 2005; and Suleyman et al., 2002).

The plasma membrane of erythrocyte is a dynamic organelle system that controls cellular structure as well as functions. Structure and functions of erythrocytes are susceptible to alterations as a consequence of interactions with xenobiotics and damage to any of its constituents may change the integrity of cell's structure and function (Nandita et al., 2008). The well-characterized product of the LPO of erythrocytes, is malondialdehyde (MDA), a highly reactive and bifunctional molecule, which is responsible for cross-linking erythrocyte phospholipids and proteins to weaken a variety of the membrane-related functions, that ultimately lead to diminishing erythrocyte survival (Siddhartha et al., 2012). Smoking drops the erythrocyte count and hemoglobin level while upsurge the leukocytes (Zafar et al., 2003).

Cigarette smoking exerts an inflammatory stimulus on macrophages, which initiate the production of inflammatory cytokines, such as TNF- $\alpha$ , which might be an important early event in the development of disease states associated with smoking (Fernandez et al., 2003). The incidences of lung cancer are significantly higher in smokers and female smokers are more prone compared with the male smokers (Zafar et al., 2003).

In our study, the LPO level of lung in smoke-exposed rats was higher than that in the un-exposed rats. Erythrocyte lipid peroxidation in smoke-exposed rats was also higher than that in the un-exposed rats. The toxic effect of *in vitro* cigarette smoke was also visible in the morphological studies of erythrocytes. Cigarette smoke-exposed erythrocytes displayed numerous leakage, breaks and membrane blobs in the plasma

membranes of erythrocytes. TNF- $\alpha$  in plasma and lung was higher in smoke-exposed rats than that of the unexposed rats. So, we can convincingly state that cigarette smoking has detrimental effects on both the special respiratory machineries, i.e., erythrocyte and the lung.

## 6. Conclusion

Our results thus suggest that cigarette smoke not only affects the lung tissues, but also deteriorates the morphology of erythrocytes by increasing the oxidative stress. Oxidative and inflammatory injury due to acute cigarette smoke speed up functional and conformational changes in lung and erythrocytes which, we believe is detrimental to the health of both active and passive smokers.

## Conflicts of interest

The authors declare that there are no conflicts of interests regarding the publication of this paper.

## Acknowledgment

The authors gratefully acknowledge the contribution of the University Grant Commission-Higher Education Quality Enhancement Program (UGC-HEQEP) for the partial instrumental support (CP358).

## References

- Ben Ahmed, H., Khelifi, S., Rtibi, K. E., Fazaa, S. and Gharbi, N. (2010). [Indicators of oxidative stress in weanling and pubertal rats following exposure to nicotine via milk. \*Hum Exp Toxicol.\* 29\(6\), 489-496.](#)
- Brody, J. S. and Spira, A. (2006). [Chronic obstructive pulmonary disease, inflammation, and lung cancer. \*Proc. Am. Thorac. Soc.\* 3, 535-538.](#)
- Carpagnano, G. E., Kharitonov, S. A., Foschino-Barbaro, M. P., Resta, O., Gramiccioni, E. and Barnes, P. J. (2003). [Increased inflammatory markers in the exhaled breath condensate of cigarette smokers. \*European Respiratory Journal.\* 21\(4\), 589-593.](#)
- Chang, K., Yang, S. M., Kim, S. H., Han, K. H., Park, S. J., and Shin, J.I. (2014). [Smoking and rheumatoid arthritis. \*Int J Mol Sci.\* 15, 22279-22295.](#)
- Evans, R. C., Omorphos, S. C. and Baysal, E. (1986). [Sickle cell membranes and oxidative damage. \*Biochemical Journal.\* 237, 2659.](#)
- Fernandez-Real, J. M., Broch, M., Vendrell, J. and Ricart, W. (2003). [Smoking, fat mass and activation of the tumor necrosis factoralpha pathway. \*Int J Obes Relat Metab Disord.\* 27, 1552-1556.](#)
- Flynn, T. P., Allen, D. W., Johnson, G. J. and White, J. G. (1983). [Oxidant damage of the lipids and proteins of the erythrocyte membranes in unstable hemoglobin disease; evidence for the role of lipid peroxidation. \*Journal of Clinical Investigation.\* 71\(5\), 1215-1223.](#)
- Grozio, A., Catassi, A., Cavalieri, Z., Pleari, L., Casario, A. and Russo, P. (2007). [Nicotine, lung and cancer. \*Anticancer Agents, Med. Chem.\* 7, 461-466.](#)
- Hashimoto, M., Hossain, S., Katakura, M., Mamun, A. A. and Shido, O. (2015). [The binding of A \$\beta\$ 1-42 to lipid rafts of RBC is enhanced by dietary docosahexaenoic acid in rats: implicates to Alzheimer's disease. \*Biochimica et Biophysica Acta—Biomembranes.\* 1848\(6\), 1402-1409.](#)
- Hashimoto, M., Shahdat, H., Shimada, T., Yamasaki, H., Fujii, Y., Ishibashi, Y. and Shido, O. (2001). [Relationship between age-related increases in rat liver lipid peroxidation and bile canalicular plasma membrane fluidity. \*Experimental Gerontology.\* 37, 89-97.](#)
- Holly, R. Middlekauff, M. D., Jeanie Park, M. D., Roya, S. and Moheimani, B. S. (2014). [Adverse effects of cigarette and noncigarette smoke exposure on the autonomic nervous system; mechanisms and implications for cardiovascular risk. \*Journal of the American College of Cardiology.\* 64\(16\), 201.](#)
- Hossain, S., Bhowmick, S., Islam, S., Rozario, L., Jahan, S., Hassan, M., Sarkar, M., Choudhury, B. K., Ahmed, S. and Shahjalal H. (2015). [Oral administration of ganoderma lucidum to lead-exposed rats protects erythrocytes against hemolysis: implicates to anti-anemia. \*Evidence Based Complementary and Alternative Medicine.\* Article ID; 463703.](#)

- Hossain, S., Chowdhury, I. H., Basunia, M.A. et al. (2011). *Syzygium cumini* seed extract protects the liver against lipid peroxidation with concurrent amelioration of hepatic enzymes and lipid profile of alcoholic rats. *J Complement Integr Med.* doi:10.2202/1553-3840.1445
- Jung, B. H., Chung, B. C., Chung, S. and Shim, C. (2001). Different pharmacokinetics of nicotine following intravenous administration of nicotine base and nicotine hydrogen tartarate in rats. *J Control Release.* 77, 183-190.
- Karczewski, J., Poniedzialek, B., Rzymiski, P., Rychlewska-Hanczewska, A., Adamski, Z., Nahar, T., Rahman, A., Choudhury, B.K., Choudhury, S.K., Mahmud, I. and Uddin, B. (2014). The effect of cigarette smoking on the clinical course of inflammatory bowel disease. *Prz Gastroenterol.* 9, 153-159.
- Khan, M. I., Bukhari, M. H., Akhtar, M. S. and Brar, S. (2014). Effect of smoking on red blood cells count, hemoglobin concentration and red cell indices. *Pakistan Journal of Medical and Health Science.* 8(2).
- Ko, F. N., Hsiao, G. and Kuo, Y. H. (1997). Protection of oxidative hemolysis by dimethyl diisoeugenol in normal and beta-thalassemic red blood cells. *Free Radical Biology and Medicine.* 22, 215-222.
- Kollias, G., Douni, E., Kassiotis, G. and Kontoyiannis, D. (1999). On the role of tumor necrosis factor and receptors in models of multiorgan failure, rheumatoid arthritis, multiple sclerosis and inflammatory bowel disease. *Immunol Rev.* 169, 175-194.
- Kovacic P. and Cooksy, A. (2005). Iminium metabolism for nicotine toxicity and addiction: oxidative stress and electron transfer. *Med Hypotheses.* 64, 104-111.
- Kwon, K. Y., Jung, H. R., Hwang, I. and Choi, W. (2012). Evaluation of bronchiolar and alveolar cell injuries induced by short and long-term exposure to sidestream smoke. *The Korean Journal of Pathology.* 46, 151-161.
- Leea, H. M., Kima, C. W., Hwanga, K. A., Choi b., D. W. and Choi a, K. C. (2016). Three components of cigarette smoke altered the growth and apoptosis of metastatic colon cancer cells via inducing the synthesis of reactive oxygen species and endoplasmic reticulum stress. *Environmental Toxicology and Pharmacology.* 45, 80-89.
- Marnett, L. J., Riggins N. J. and West, J. D. (2003). Endogenous generation of reactive oxidants and electrophiles and their reaction with DNA and protein. *L Clin Invest.* 111, 583-593.
- Momi, N., Ponnusamy, M. P., Kaur, S., Rachagani, S., Kunigal, S. S., Chellappan, S., Ouellette, M.M. and Batra, S.K. (2013). Nicotine/cigarette smoke promotes metastasis of pancreatic cancer through a9 nAChR-mediated MUC4 upregulation. *Oncogene.* 32, 1384-1395.
- Nahar, T. Uddin, B. Hossain, S., Sikder, A. M. and Ahmed, S. (2013). Aloe vera gel protects liver from oxidative stress-induced damage in experimental rat model. *J Complement Integr Med.* V.10(1). doi:10.1515/jcim-2012-0020
- Nandita, Singh, P. S. and Rajini (2008). Antioxidant-mediated protective effect of potato peel extract in erythrocytes against oxidative damage. *Chemico-Biological Interactions.* 173, 97-104.
- Office on Smoking and Health, US (2006). *The health consequences of involuntary exposure to tobacco smoke, A report of the surgeon general.* Atlanta (GA): Center for disease control and prevention. US.
- Pryor, W. A. and Stone, K. (1993). Oxidants in cigarette smoke. Radicals, hydrogen peroxide, peroxyhydrate, and peroxyhydrate. *Ann NY Acad Sci.* 686, 12-28.
- Sekhon, H., Proskocil, B. J., Clark, J. A. and Spindel, E. R. (2004). Prenatal nicotine exposure increases connective tissue expression in fetal monkey pulmonary vessels. *Eur Respir J.* 23, 906-15.
- Siddhartha, S., Saha, Chakraborty, A., Ghosh, S. and Ghosh, M. (2012). Comparative study of hypocholesterolemic and hypolipidemic effects of conjugated linolenic acid isomers against induced biochemical perturbations and aberration in erythrocyte membrane fluidity. *European Journal of Nutrition.* 51, 483-495.
- Singh, S., Pillai, S. and Chellappan, S. (2011). Nicotinic acetylcholine receptor signaling in tumor growth and metastasis. *J Oncol.* 456-743.
- Suleyman, H., Gumustekin, K., Taysi, S., Keles, S., Oztasan, N., Aktas, O. Altinkayanak, K., Timur, H. Akcay, F., Akar, S., Dane, S. and Gul, M. (2002). Beneficial effects of *Hippophae rhamnoides* L. on nicotine oxidative stress in rat blood compared with vitamin E. *Biol Pharm Bull.* 25, 1133-1136.



- Van der Vaar, H., Postma, D. S. and Timens, W. (2004). *Acute effects of cigarette smoke on inflammation and oxidative stress: a review. Thorax. 59, 713-721.*
- Vassalli, P. (1992). *The pathophysiology of tumor necrosis factors. Annu Rev Immunol. 10, 411-452.*
- Wang, G., Mohammadtursun, N., Sun, J., Lv, Y., Jin, H., Lin, J., Kong, L., Zhao, Z., Zhang, H. and Dong, J. (2018). *Establishment and evolution of a rat model of side stream cigarette smoke-induced chronic obstructive pulmonary disease. Frontiers in physiology. V-9, article no-58. doi: 10.3389/fphys.2018.00058*
- WHO (2007). *Why is tobacco a public health priority? URL <http://www.who.int/tobacco/about/en>*
- Yang, H., Chen, S., Chang, N., Chang, J., Lee, M., Tsai, P., Fu, H. H., Kao, W. W., Chiang, H. C., Wang, H. H. and Hseu, Y. C. (2006). *Protection from oxidative damage using Bidens pilosa extracts in normal human erythrocytes. Food and Chemical Toxicology. 44, 1513-1521.*
- Yildiz, D., Ercal, N. and Armstrong, D. W. (1998). *Nicotine enantiomers and oxidative stress. Toxicology. 130, 155-165.*
- Yu, L. (2001). *Free radical scavenging properties of conjugated linoleic acids. Journal of Agriculture and Food Chemistry. 49, 3452-3456.*
- Zafar, I., Naseer, K., Mohammad, Nisar, M., Rashida, M., Asadullah, Shumaila, B. and Mohammad, S. A. (2003). *Effect of cigarette smoking on erythrocytes, leukocytes and haemoglobin. Journal of Medical Sciences. 3(3), 245-250.*

**Cite this article as:** Shahdat Hossain, Marzan Sarkar, Sujan Bhowmick, Jakir Hussain, Mahmudul Hasan, Borhan Uddin, Md. Asaduzzaman Khan and Taslima Nahar (2021). *Acute cigarette smoke exposure induces oxidative damage and inflammation in Wistar rats: impact on lungs and erythrocytes. African Journal of Biological Sciences. 3(1), 120-128. doi: 10.33472/AFJBS.3.1.2021.120-128.*