Abstract No.62

Upregulation of NF-kB1/RelA in human bronchial wall of mustard gas induced patients

S. Yazdani[1], A.A. Imani Fooladi[4], M.H. Karimfar[2], M. Ebrahimi[1], M. Roudkenar Habibi[3], M.R. Nourani[1,]*

1- Chemical Injury Research Center, Baqiyatallah Medical Sciences University, Tehran, IRAN, E-mail: r.nourani@yahoo.com, 2- Zobol Medical Sciences University, Zabol, IRAN, 3- Research Center, Iranian Blood Transfusion Organization, 4- Research Center of Molecular Biology, Baqiyatallah Medical Sciences University, Tehran, IRAN

Sulfur mustard (SM) is a chemical warfare agent which has been used during Iran-Iraq war against Iranian troops. Nowadays there are more than 40000 people suffering from SM lesions, especially pulmonary disorder in Iran. SM disturbs scavenge of ROS and eventually causes chronic obstructive pulmonary disease (COPD) which is one of the most abundant inflammatory disease. Nuclear factor kB (NF-kB)/ Rel family is one of the most important proteins involved in inflammatory responses. They are members of DNA-binding protein factors that are required for transcription of many proinflammatory molecules. Existence of NF-kB is a well known marker in inflammatory status in animal model systems to expose the pathobiology of lung diseases. In this study we sought to address the expression of NF-kappaB1/RelA and presence of inflammation in bronchial wall biopsies of SM exposed patients. We considered NF-kappaB1/RelA as the primary heterodimer in lung inflammation. Ten normal individuals and twenty SM induced patients were comprised. Expression of NF-κB1/RelA in healthy and SM induced samples were measured by semi quantitative RT-PCR, Real-time PCR. Expression levels of NF-κB1 and RelA in SM exposed patients were upregulated about 2.53±0.32 and 3.83±0.87 folds respectively in compare to normal samples (P□0.05).

This is the first study about the induction of inflammatory molecules in patients exposed to SM. Here we suggest that over expression of NF-κB1/RelA molecules in COPD induced by SM, may follow an inflammatory procedure in bronchial wall of these patients at mRNA levels.

Key words: bronchial wall, mustard gas, NF-κB1, RelA, COPD.

Abstract No.63

Fluorescence spectroscopy study of human hemoglobin upon interaction with an anti-breast cancer drug

M. Saboury[1], M.R .Housaindokht[2], J. Chamani[1]*

1- Department of Biology, Faculty of Science, Islamic Azad University-Mashhad Branch, Mashhad, Iran, E-mail: mesabori@gmail.com
2- Department of Chemistry, Faculty of Science, Ferdowsi University of Mashhad, Mashhad, Iran.

.

Hemoglobin is the major component of red blood cell and as an allosteric tetrameric protein**;** it has an important role in carrying oxygen from lunge to different tissues. Hemoglobin was initially thought to reversibly bind only with oxygen, but they were later shown to react with other gasses such as CO and NO and with several organic molecules such as phospholipids and other membrane lipid.Tamoxifen is a non-steroidal anti-estrogen drug that is widely used in the treatment and prevention of breast cancer. It is currently used for the treatment of the both early and advanced ER^+ (estrogen receptor positive) breast cancer. Here, interaction between hemoglobin and tamoxifen was investigated at two pH (pH 7.4 and pH 8.4) using fluorescence spectroscopy. Fluorescence measurements were carried out in a Jasco 2500, Hitachi fluorescence spectrophotometer. The excitation wavelength was 280 nm, and the emission spectra were read at 300-600 nm. For describing the fluorescence quenching by tamoxifen using the Stern-Volmer and Scatchard equation. The results of Sten-Volmer and Scatchard plots reveal that K_{SV} and binding constant decreased with increasing pH. The intrinsic Fluorescence of Hb primarily originates from β-37 Trp that plays a key role in the quaternary State change upon ligand binding. Changes in emission spectra tryptophan are common in response to protein conformational transitions, substrate binding. Linear Stern-Volmer plots may either reveal the occurrence of just a binding site for quencher in the proximity of the fluorophore, or indicate the existence of a single type of quenching. Scatchard plots lead us to analyze the binding data by fitting the data to Hill equation for multi-set of binding site. Therefore, the difference between binding affinities two interaction is showed change structure of HB.

Key words: Fluorescence spectroscopy, human hemoglobin, Tamoxifen.
