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Extended Abstract

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Influence of the oxidative stress on the secretion of the endogenous antimicrobial peptides in hereditary blood diseases

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Among the illnesses of the blood, thalassemia occupies a extraordinary place, related with a reduction or whole absence of synthesis of globin chains of hemoglobin. Azerbaijan is considered as an endemic sector of these inherited blood diseases, which makes conducted researches relevant. The aim of this work was to study the relationship between the thiol repute of blood and the secretion of endogenous antimicrobial peptides. The blood of 57 sufferers aged 6-17 years was studied. All patients depending on the pathology have been divided into the following groups: crew I-20 children with a homozygous structure of β-thalassemia, group II-37 kids with G6PD deficiency. To investigate the degree of oxidative stress of the body, carbonylated proteins (CP) and thiol reputation (TS) of blood had been chosen as markers. To investigate the degree of secretion of endogenous antimicrobial peptides, a quantitative analysis of defensin and endotoxin in blood plasma was carried out the usage of the ELISA method. The lookup used to be carried out with the financial support of the Science Development Foundation of Azerbaijan. As a result of research, it used to be revealed that in group I patients, the amount of CP multiplied through 11%, in the crew II patients CP improved by 1.6% and TS decreased by way of 1.5%. The stage of defensin in crew I multiplied via 2%, and endotoxin through 1.7%. In crew II, these indicators elevated by using 1.7% and 2.3%, respectively. With the trade of the body's TS, the secretion of α-defensin was increasing. In β-thalassemia, carbonylated proteins enlarge in the blood, thiol repute decreases, which shows at the amplify of the affect of oxidative stress related with widespread infectious issues and activation of neutrophils. Reactive oxygen species (ROS) are generated as by-products of everyday mobile metabolic activities. Superoxide dismutase, glutathione peroxidase, and catalase are the enzymes involved in protecting cells from the unfavourable outcomes of ROS. ROS are produced in response to ultraviolet radiation, cigarette smoking, alcohol, nonsteroidal anti-inflammatory drugs, ischemia-reperfusion injury, chronic infections, and inflammatory disorders. Disruption of ordinary cell homeostasis by means of redox signaling may additionally end result in cardiovascular, neurodegenerative diseases and cancer. ROS are produced within the gastrointestinal (GI) tract, however their roles in pathophysiology and sickness pathogenesis have now not been nicely studied. Despite the protecting barrier supplied by using the mucosa, ingested substances and microbial pathogens can result in oxidative harm and GI inflammatory responses involving the epithelium and immune/inflammatory cells. The pathogenesis of quite a number GI illnesses such as peptic ulcers, gastrointestinal cancers, and inflammatory bowel disorder is in section due to oxidative stress. Unraveling the signaling events initiated at the cellular stage through oxidative free radicals as properly as the physiological responses to such stress is important to better understand ailment pathogenesis and to develop new therapies to manage a variety of conditions for which present day cures are not continually sufficient. Reactive oxygen species (ROS), also referred to as reactive oxygen intermediates (ROI), are byproducts of ordinary cell metabolism. Low and moderate amounts of ROS have recommended outcomes on quite a few physiological approaches which includes killing of invading pathogens, wound healing, and tissue repair processes. As discussed in section IV, ROS act as necessary signaling molecules. Cancer treatment by chemotherapeutic agents and radiotherapies rely generally on ROS technology to wreck malignant cells by using inducing apoptosis. However, disproportionate era of ROS poses a serious trouble to bodily homeostasis and reasons oxidative tissue damage. While herbal antioxidant pathways can restrict the damaging consequences of ROS, their levels can be motivated by using many oxidative stressors and maintained such that they make a contribution to tissue damage. ROS are produced in response to ultraviolet (UV) radiation, cigarette smoking, alcohol consumption, ingestion of nonsteroidal anti-inflammatory tablets (NSAIDs), and many other exogenous agents. Infections, ischemia-reperfusion (I/R) injury, and a number of inflammatory strategies additionally end result in accelerated degrees of ROS. Disruption of ordinary cell homeostasis via redox signaling contributes to sickness in actually each and every organ which includes the improvement of cancer. The gastrointestinal (GI) tract is a key supply of ROS. Despite the protective barrier furnished through the epithelial layer, ingested materials and pathogens can motive irritation through activating the epithelium, polymorphonuclear neutrophils (PMNs), and macrophages to produce inflammatory cytokines and different mediators that contribute in addition to oxidative stress. Various GI pathological prerequisites such as gastroduodenal ulcers, GI malignancies, and inflammatory bowel disease (IBD) occur in part from oxidative stress. Understanding the signaling activities initiated by means of free radicals as nicely as the physiological response to such methods is key to furthering our appreciation of ROS-mediated GI diseases with the attainable to enhance novel therapeutic interventions.

Bottom Note: *This work is partly presented at 10th Edition of International Conference on Structural Biology* March 15-16, 2018 Barcelona, Spain