evidenced by increased lipid peroxidation but also by deficits in some antioxidants (vitamin C, glutathione, thiol proteins) and trace elements (selenium). *E-mail address:* j.pincemail@chuliege.be

DOI:10.1016/j.freeradbiomed.2021.08.192

PP118

Oxidative Stress Status and its correlation with Redox Status as Measured By An Electrochemical (PAOT®) Methodology: A Pilot Study in Critical COVID-19 Pneumonia Survivors

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Background: In most serious COVID-19 forms which required prolonged stay in intensive care unit, pulmonary, cardiovascular, renal, neurological and psychological sequelae have been reported after the infection. All these complications can be sustained by chronic inflammatory problems and/ or increased oxidative stress. Material and Methods: Biomarkers of the systemic oxidative stress status (OSS) including enzymatic and non-enzymatic antioxidants, total antioxidant capacity of plasma (PAOT®-Sore), trace elements, oxidative damage to lipids and inflammation markers, were investigated in 12 patients admitted to a revalidation center for post-19 COVID pneumonia. Results: From blood samples collected two months after hospital discharge and one month after admission to the revalidation center, vitamin C, thiol proteins, reduced glutathione, gamma-tocopherol and beta carotene were significantly decreased compared to reference values. By contrast, lipid peroxides and markers of inflammation (neutrophils, myeloperoxidase) were significantly higher than the norms. Lipid peroxides was strongly correlated with Cu ($r = 0.95$, $P < 0.005$) and Cu/Zn ratio (0.66, $P = 0.020$). Using an electrochemical method (PAOT®), total antioxidant capacity (TAC) evaluated in saliva and urine negatively correlated with copper and lipid peroxides. Similar findings were obtained for PAOT®-skin score. Conclusions: Systemic OSS was strongly altered in patients admitted in revalidation after C0VID-19 infection. This suggests the need for supplementing these patients with antioxidants. *E-mail address:* j.pincemail@chuliege.be

DOI:10.1016/j.freeradbiomed.2021.08.193

PP119

Evaluation of anti-tumoral and/or anti-angiogenic compounds on the MDA-MB-231 and HMEC redox balance

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Angiogenesis, the formation of new blood vessels from other pre-existent ones, is highly regulated by a balance between stimulators and inhibitors under physiological conditions. However, a persistent angiogenesis is related to many pathological conditions, including cutaneous, ophthalmic and inflammatory diseases, as well as cancer. Interestingly, reactive oxygen species (ROS) have been found to stimulate or block the angiogenic response, acting as a double-edged sword in endothelial cells, because high or sustained ROS concentration is detrimental, while transient or low levels of ROS can activate signaling pathways that promote angiogenesis. Moreover, elevated rates of ROS have been found in almost all cancers, where they promote many aspects of tumor development and progression. However, tumor cells also express higher levels of antioxidant proteins to detoxify ROS, suggesting that a delicate balance of intracellular ROS levels is required for cancer cell function, and an alteration in this balance could regress the tumor2. Our group has studied the redox potential of several anti-tumoral and anti-angiogenic compounds (HT, DMF and GR-24) in human breast cancer cells (MDA-MB-231) and in human endothelial cells (HMEC). Our data show that in presence of these compounds, tumor and endothelial cells decrease the reduced sulfhydryl group levels, suggesting that these compounds can diminish the levels of intracellular reduced glutathione. On the other hand, we have seen that DMF and GR-24 reduced the cell viability of HMEC in the H_2O_2 cytotoxicity assay, indicating that these compounds could sensitize cells to oxidative stress. Additionally, DMF, GR-24 and HT modulate the catalase and superoxide dismutase activities in tumor and endothelial cells, and HT was capable of reducing intracellular ROS production in both cell types. Taking altogether, our data reveal that these compounds alter the redox balance of tumor and endothelial cells, and their antitumor and antiangiogenic activity could be, at least in part, due to the imbalance in the cellular redox state. *E-mail address:* torresvargas@uma.es

DOI:10.1016/j.freeradbiomed.2021.08.194