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Glutathione pathway and GST polymorphisms in the immune response to pathogen; case study: SARS-CoV-2: The missing piece of the covid-19 puzzle?

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This review discusses the immune response to pathogen, with a focus on covid-19 and reduced (GSH) status, the role of GSH in T cell metabolism and response to viral infection, the polymorphism of genes responsible for GSH levels and how these impact covid-19. GSH maintains homeostasis in normal cells via scavenging reactive oxygen species (ROS), serving as a reservoir of cysteine. The effector function of T cell is essentially regulated by the metabolic activity of GSH. The clonal expansion of T cells leads to its requirement for increased glutamine utilization. Rising ROS concentrations is controlled by T cell via the use of endogenous antioxidants, especially GSH, whose synthesis is catalysed by glutamate cysteine - ligase (GCL). GST polymorphisms occur in most lungs, pancreatic, ovarian, cervical, colorectal and prostate cancers. The severity of illness in patients with underlying conditions could be associated with the status of GSH, GCLC, GST (glutathione-S-transferase). Furthermore, GSTs are involved in synthesizing prostaglandins, leukotrienes, progesterone, testosterone, and tyrosine-degradation. The involvement of GSTs in protein-protein interactions with vital kinases in the regulation of cellular signalling controls the stress response, proliferation, and apoptosis. Moreover, glutathione insufficiency may also facilitate greater stimulation of von Willebrand factor, a vital clotting factor responsible for platelet aggregation and coagulation. It has been demonstrated that the Nrf2 antioxidant gene expression pathway was suppressed in covid-19 patients.

Due to the implication of GST in kinases and immune systems, the activities of the various drugs utilized for the treatment of covid-19 require readdressing to consider the significance of polymorphisms in various therapies. This polymorphism in drug metabolizing enzymes (DMEs), including those from the GST gene family, could also influence individual and population response to covid-19 vaccines. Other key players of oxidative stress include Nrf2, KEAP1, and ACE2 receptors, which are regulated by RAS and implicated in the pathogenesis of covid-19.

Speaker Biography

Bene A Ekine-Afolabi is a graduate of River State University of Science and Technology in Applied Biology (Medical Microbiology option); with an MRes degree at University of East London, United Kingdom. She had her PhD study and worked at the Department of Natural Sciences, Middlesex University, UK. Trained in practical approach to toxicology in drug development (American College of Toxicology/British Toxicology Society). She done her research in Microbiology, Molecular Biology and Cancer: Her current focus of research (which has yielded eight designed models), is on the Investigation of molecular mechanism of colorectal cancer and due to the current pandemic, has been involved in drug development for COVID-19. She has Harvard University part-sponsored training in therapeutic research in Cancer Biology and Therapeutic. She has been involved in three published peer reviewed article, two manuscript awaiting publication, among which one is on covid-19 and was submitted to the Chief Medical Officer of United Kingdom to assist in response to the pandemic.

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