In vitro effects of hypolipidemic drugs on oxidative stress

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Generation of reactive oxygen species is important for the normal physiology as well as pathogenesis of many diseases. Cells defend themselves against Reactive Oxygen Species (ROS) damage by the help of antioxidants present within and outside the body. Antioxidants remove free radical intermediates and inhibit oxidation. An imbalance between oxidants and antioxidants within the body results in oxidative stress, which can leads to cardiovascular and many other diseases (Brea *et al.*, 2012).

Hypolipidemic drugs like statins are widely used as cholesterol lowering agents with cholesterol independent effects such as on oxidative stress (Parihar *et al.*, 2012). We have studied the *in vitro* effects of hypolipidemic drugs on neutrophils as to understand the mode of action of these drugs on oxidative stress. We have used enhanced chemiluminescence (CL) assay for the detection and quantitative analysis of ROS. Oxidative burst is stimulated by NADPH oxidase by reducing molecular oxygen produced superoxide anions. Myeloperoxidase is present in azurophilic granules of neutrophils. After phagocytic activation, myeloperoxidase catalyzes the production of highly bactericidal hypochlorous acid (HOCL) from H_2O_2 and Chloride ions (Cl^-) as substrates (Gross *et al.*, 2009).

Atorvastatin, Simvastatin, Lovastatin, Gemfibrozil and Nicotinic acid were found to be significantly inhibiting the ROS production. This shows that statins have direct free radical scavenging activity and are capable of decreasing the generation of ROS *via* vascular NADPH oxidase, thus inhibiting respiratory burst of phagocytes (Beltowski, 2005).

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