POTENTIAL EFFECTS OF REACTIVE OXYGEN SPECIES (ROS).

Roland Amir, Centre de Santé des Fagnes, Nuclear Medicine Department.
Boulevard Louise,18 – 6460 Chimay. Belgium.

Introduction.

ROS are radicals, ions or molecules that have a single unpaired electron in their outermost shell of electrons.

Interaction with inflammatory processes.

ROS produced in vivo by endothelial, inflammatory and immune cells by various cellular pathways have two actions. The first one is its role in oxidative stress and tissue injury and second one is its participation in redox signalling.

Inflammation encompasses both these aspects of ROS function. The onset of the inflammatory cascade is the coordinated event that occurs in part via redox signalling events that cause the recruitment and adherence of immune cells to the site of infection or injury. Indeed immune cells, dendritic cells, epithelial cells and endothelial cells all produce ROS upon activation during either infectious or sterile tissue damage. ROS are critical effectors that participate in a plethora of redox-regulated cellular events eventually leading to the resolution of inflammation. Excessive ROS generation, however, results in inflammatory tissue damage, organ failure and the development of a variety of chronic inflammatory diseases [(1)].

Complementary data.

Hydrogen peroxide (H2O2) is produced by reduction of superoxide (O2-) through dismutation. Hydroxyl radical (OH-) arises from electron exchange between O2- and H2O2 via the Harber-Weiss reaction or it is also generated by the reduction of H2O2 by the Fenton reaction. When generated under strictly regulated conditions, these ROS, in particular O2- and H2O2, may act as signalling molecules that mediate physiological processes, such as cell growth, differentiation, metabolism, and survival of cardiomyocytes. This type of intracellular signalling pathway is termed “redox signalling”. On the other hand, excess production of ROS damages DNA, protein, and lipids, thereby cell death in the heart [(2)]

Conclusion.

Studies over the past two decades in various organisms, tissues and cell types have led to a shift in our understanding of ROS: we no longer view them just as molecules that invoke damage but now also appreciate their role in regulating signalling pathways that impinge on normal physiological and biological responses.

Reference.

1-Jose p. Redox signalling and the onset of the inflammatory cascade.
Immunity and inflammation in health and disease, 2018.
2-Yasuhiro et al. Oxidative stress and cardiac muscle.
Muscle, 2012.