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**High metabolic activity of some Protozoa pathogens result in the high production of reactive oxygen intermediates. How is this possible?**

While encountering pathogens, host immune systems generate Reactive oxygen species (ROS) as defense mechanisms to clear pathogens and is one of the earliest antimicrobial defence mechanisms put forward by phagocytic cells. They act as signaling molecules and thus, the elevated levels of ROS is expected to dampen the persistence of the microbes within the host cell. These ROS include hydroxyl radicals, hydrogen peroxide ( $H_2O_2$ ), and superoxide anions ( $O_2^-$ ). ROS have the nature of unpaired electrons in their outer orbital that render them as highly reactive species. As a direct approach, ROS damages pathogen's DNA through distortion of bases. In an indirect way, it activates pro-inflammatory cytokines, thus creating an environment not suitable for the microorganisms. ROS has also been found to cause apoptosis of the host cell, thereby ensuring complete elimination of the pathogen.

Conversely, various chemotherapeutic drugs against trypanosomatids-caused ROS induction result in oxidative stress, eventually leading to apoptotic manifestations. Oxidative stress is one of the host defense mechanisms to control the infection, while detoxification is one of the crucial counteracts at the parasite front for successful host-parasite interaction. Therefore, oxidative stress is a good tool for better

understanding of parasite biology, pathogenesis, and host-pathogen interactions. *Entamoeba histolytica* is an intestinal protozoan parasite that causes amebic dysentery, extra intestinal and liver abscesses in millions of inhabitants of endemic areas. *E. histolytica* trophozoites are microaerophilic and have been shown to consume oxygen and tolerate low levels of oxygen pressure. They are also exposed to highly toxic reactive oxygen and nitrogen species (ROS or RNS) during tissue invasion, colonization and extra intestinal propagation. The capacity of *E. histolytica* trophozoites to survive reactive oxygen and nitrogen species is integral to its pathogenic potential and disease outcome.

It is plausible that microbes can thrive in oxidative environments by decreasing host responses mediated by ROS during infection or inducing several genes encoding antioxidant enzymes. However, ROS at high concentrations have an adverse effect on any cellular components. Based on the significant impact on antimicrobial actions, antimicrobials efficiently kill bacteria by inducing ROS and targeting production of ROS, thus strengthening antimicrobial activity. Therefore, the balance of ROS needs to be investigated to control microbial infections and associated diseases.