NO signalling: the molybdoenzymes role

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Cardiovascular diseases (CVD), metabolic syndromes and other related diseases are major concerns of our modern society. CVD, in particular, are a leading cause of morbidity and mortality in all areas of the World, except Africa, despite recent improvements in outcomes. A common denominator to these diseases is a compromised nitric oxide (·NO) bioavailability. NO is a signalling molecule that controls a wide variety of functions in humans, including the well known vasodilation, but also platelet aggregation, neurotransmission, apoptosis, gene expression and many other. Its formation is mainly catalysed by specific arginine,O_2-dependent NO synthase (NOS) isoforms and the NO lifetime is controlled through its rapid oxidation to nitrate. In the last decade, a new nitrate-nitrite-NO pathway has emerged as a physiological alternative to the "classic" NOS pathway to support cell functioning under hypoxic conditions (when the O_2-dependent NOS is hampered). In this communication, we will show that the molybdoenzymes xanthine oxidoreductase (XD and XO) and aldehyde oxidase (AO) are able to reduce nitrate and nitrite to NO and describe the conditions under which the NO generation takes place. In vitro and in situ studies will be presented and discussed to show the relevance of these two enzymes on the NO metabolism in conditions associated with a reduced NO bioavailability.

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