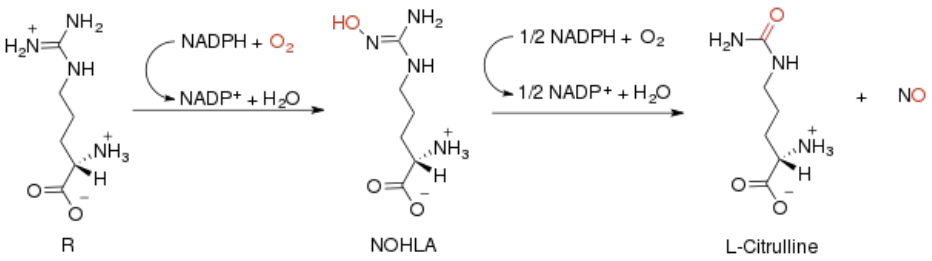
NO-synthase



NO-synthases (NOS) catalyze the oxidation of *L -arginine* to NO · and L -citrulline to form the intermediate metabolite Nω-hydroxy- L - arginine. The synthesis is influenced by a number of **cofactors** such as tetrahydropterin (BH_4), flavin mononucleotide (FMN), flavindinucleotide (FAD), reduced thiols, endogenous NOS inhibitor - asymmetric dimethylarginine (ADMA) and substrate availability. In addition, the activity of NOS I and III depends on the presence of a *calmodulin complex with* Ca^{2+} ($CaM\text{-}Ca^{2+}$).

Isoform	Cell type	Basal NO concentration .	Stimulated NO concentrations .
Type I (nNOS)	neurons, skeletal muscle, smooth muscle	low	temporarily low
Type II (iNOS)	macrophages, myocytes, smooth muscles, hepatocytes	none	consistently high
Type III (eNOS)	endothelial cells, platelets	low	temporarily high

The effect of NO · in a given biological system depends on its concentration, diffusibility and concentration of other bioreactants (superoxide dismutase , catalase , xanthine oxidase , guanylate cyclase, SH-groups, OH-groups, reactive oxygen species , hemoglobin). The resulting NO · can thus gain an electron to form a nitroxyl anion (NO^-) or, conversely, lose an electron to form NO^+ (nitrosonium ion). Both nitroxyl and nitrosonium ions then react with other molecules or radicals. The immediate metabolite of NO · in blood plasma is nitrite (NO^{2-}), which enters erythrocytes and oxidizes to nitrate (NO^{3-}). Another way is to interact with the superoxide anion O_2^{2-} · to form peroxynitrite ($ONOO^-$). It then oxidizes thiols or thioethers, or reacts with polypeptide tyrosine, guanosine, degrades carbohydrates, induces lipid peroxidation, and cleaves DNA . These processes play a key role, for example, in vascular endothelial dysfunction

Links

Source

- MASOPUST, Jaroslav, et al. *Cell pathobiochemistry.* 1st edition. Prague: Charles University, 2nd Faculty of Medicine, 2003. 344 pp. 88–92. ISBN 80-239-1011-6 .