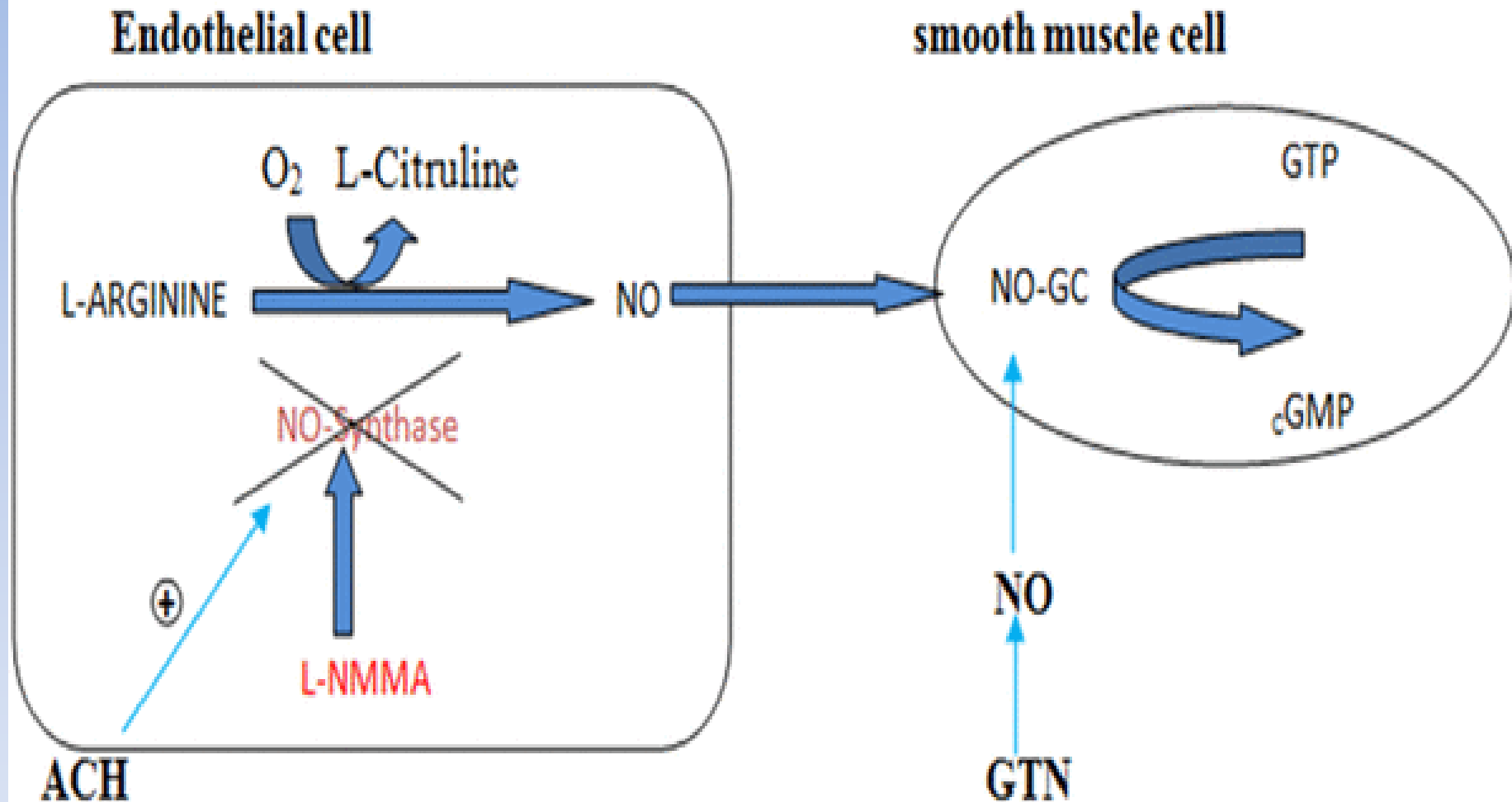


DIABETES and NITRIC OXIDE

Prof.Dr.Nuray ARI, 2018

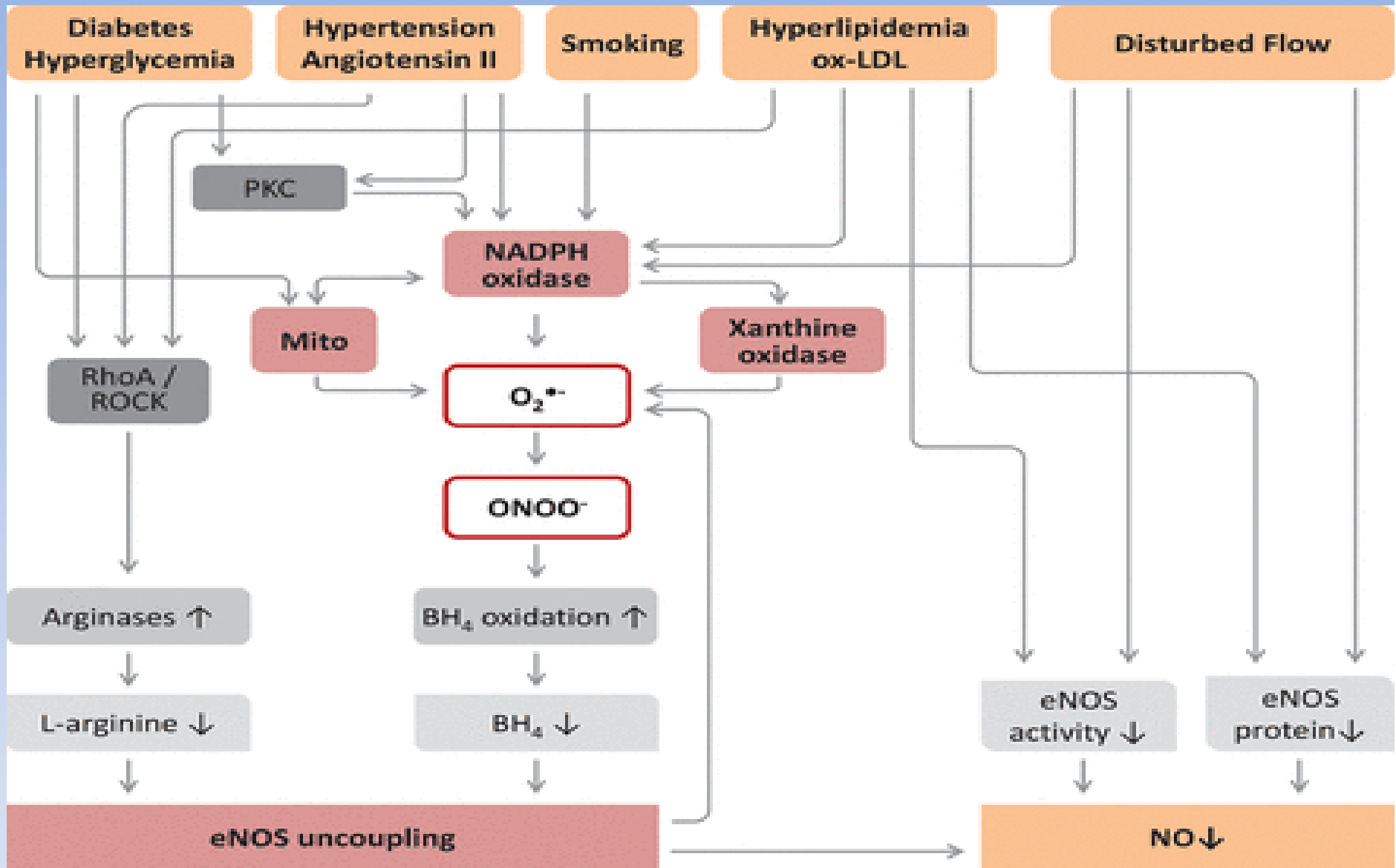
Endothelial Dysfunction

The endothelium and nitric oxide



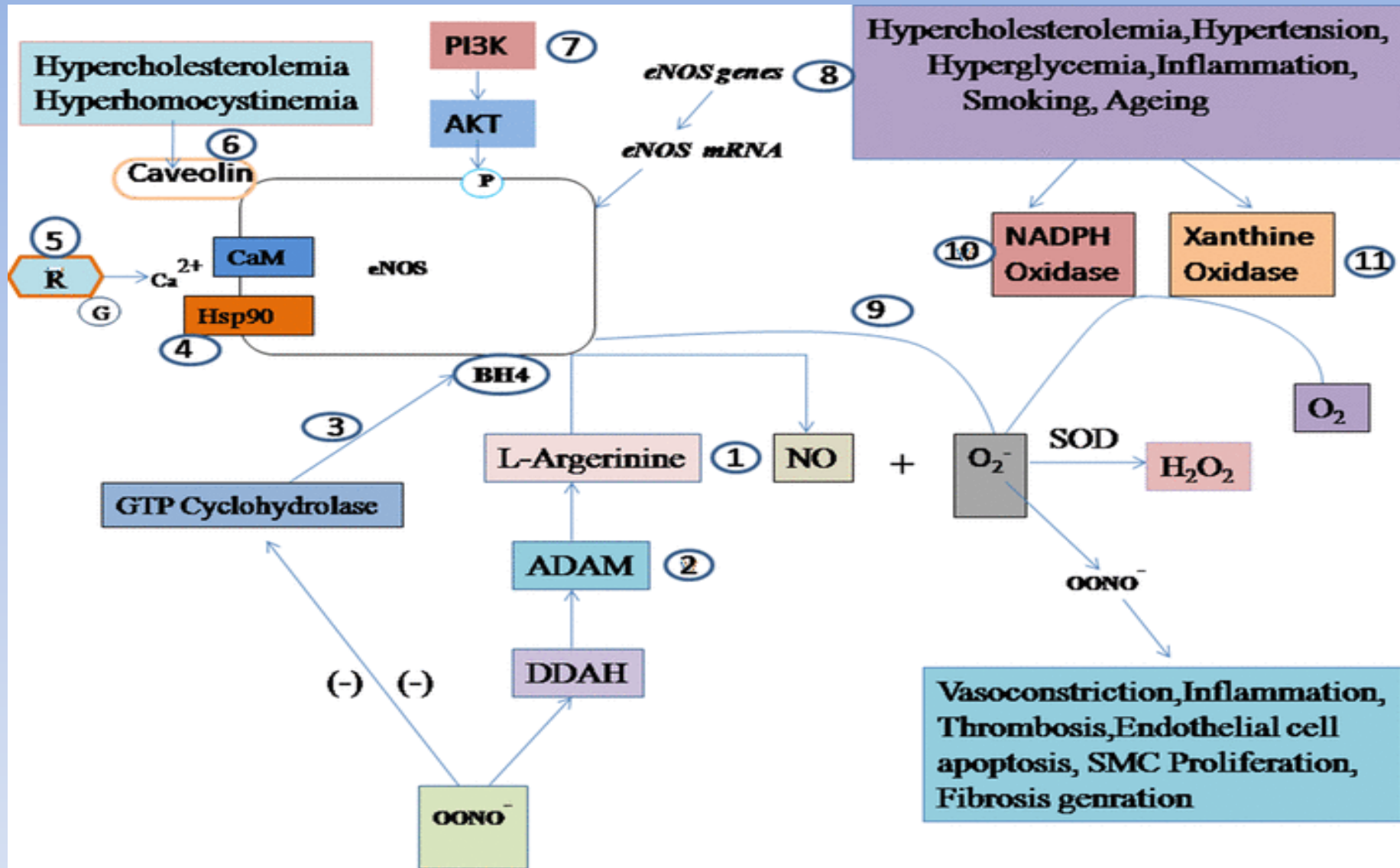
Vascular endothelium and nitric oxide production

Vascular endothelium dysfunction: a conservative target in metabolic disorders. . [Inflamm Res](#). 2018 doi: 10.1007/s00011-018-1129-8. [Epub ahead of print]. [Jamwal S](#) and [Sharma S](#).



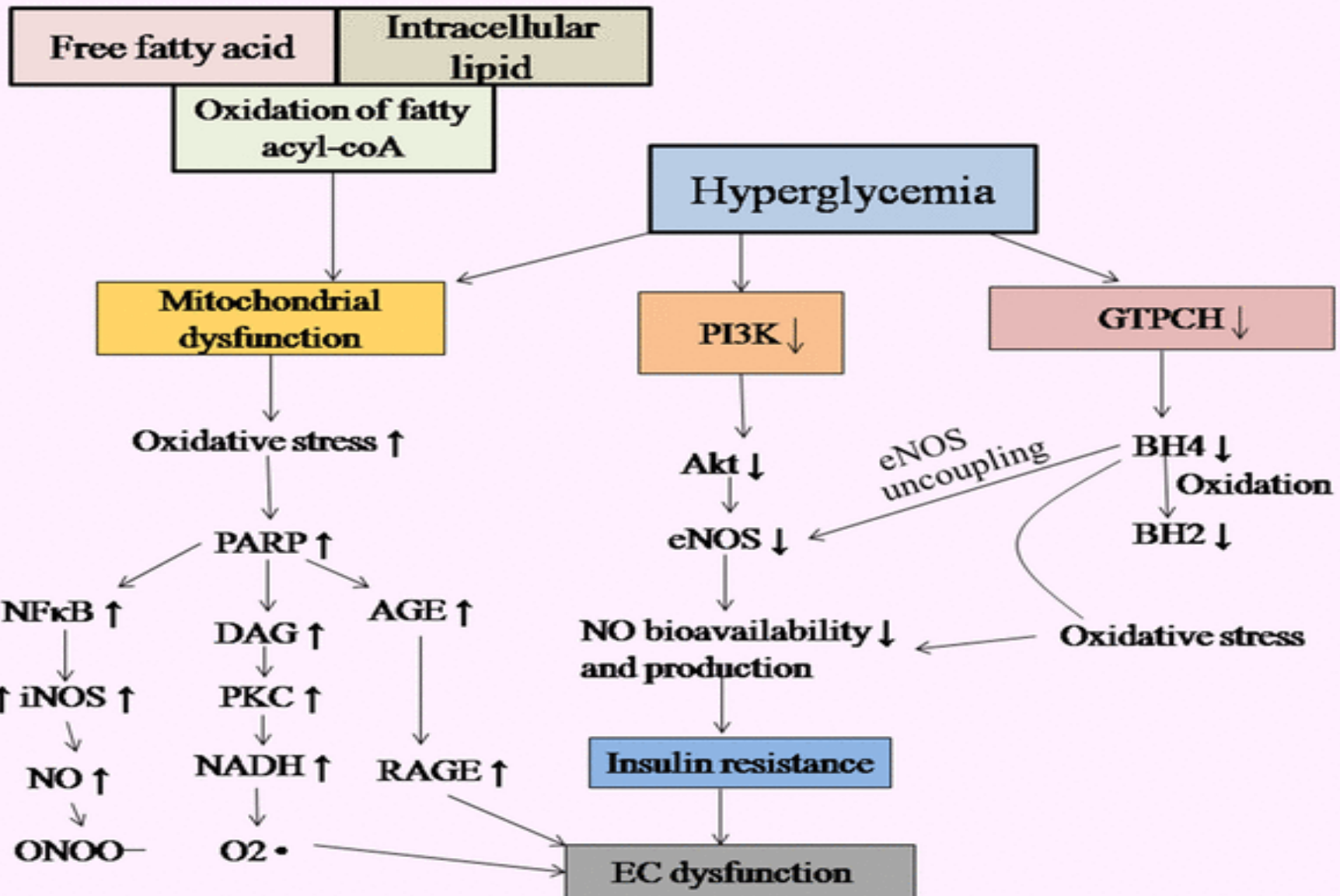
Cardiovascular risk factors induce vascular oxidative stress and reduce endothelial nitric oxide production

Roles of Vascular Oxidative Stress and Nitric Oxide in the Pathogenesis of Atherosclerosis. [Circ Res.](#) 2017 17;120(4):713-735 [Förstermann U et al.](#)

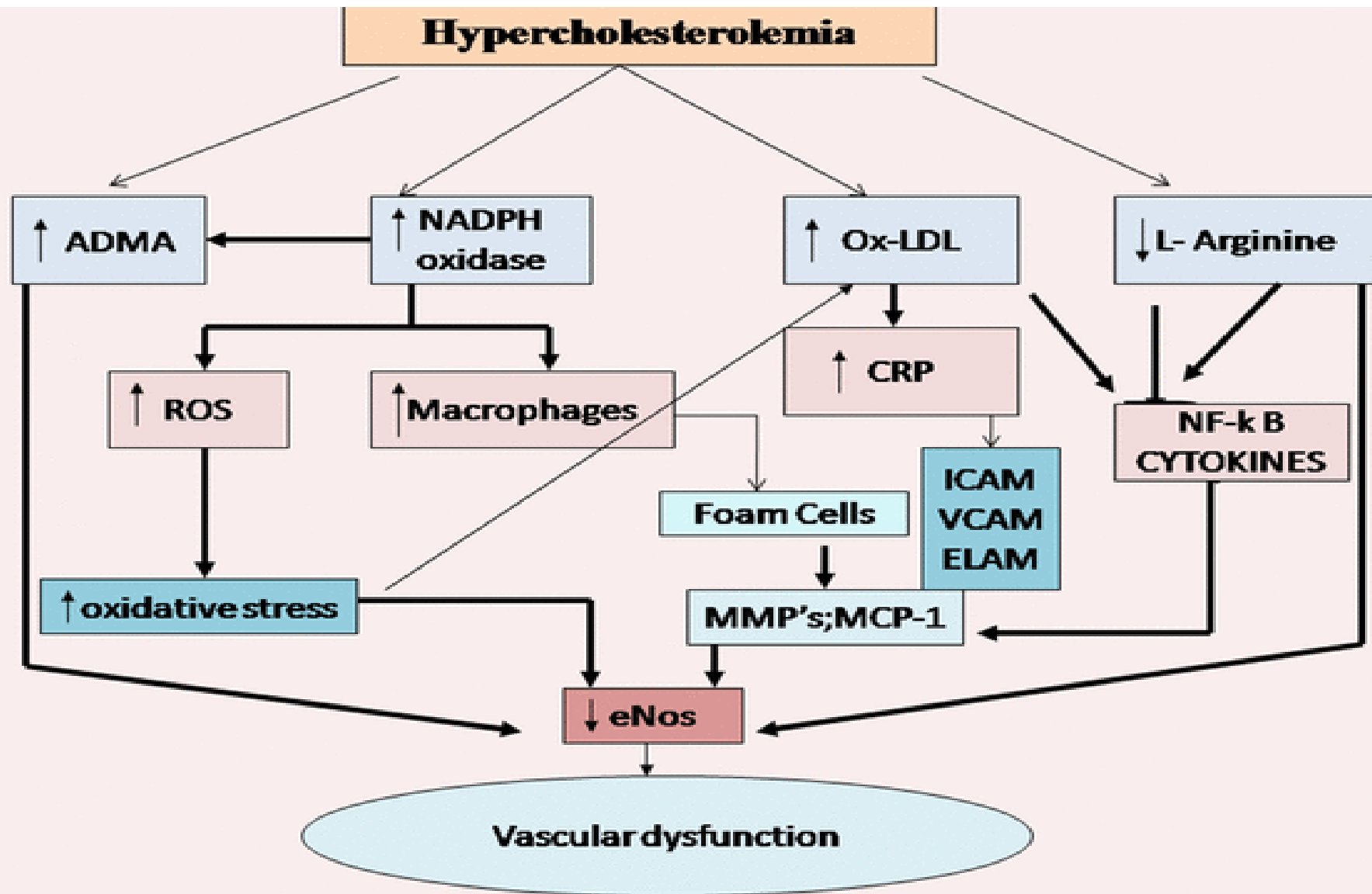


Risk factors for vascular endothelial dysfunction.

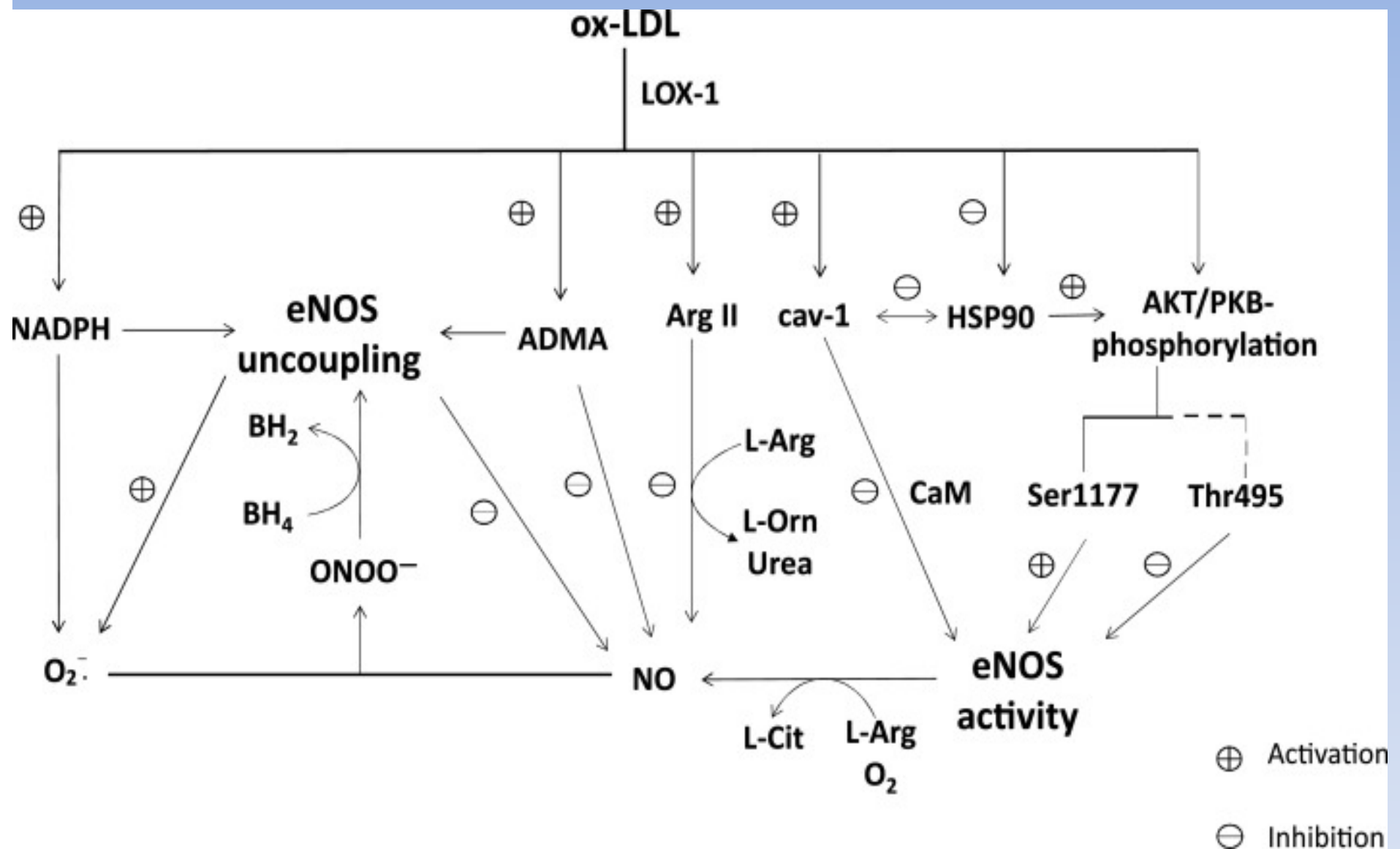
Vascular endothelium dysfunction: a conservative target in metabolic disorders. [Jamwal S](#) and [Sharma S. *Inflamm Res.* 2018 doi: 10.1007/s00011-018-1129-8. \[Epub ahead of print\].](#)



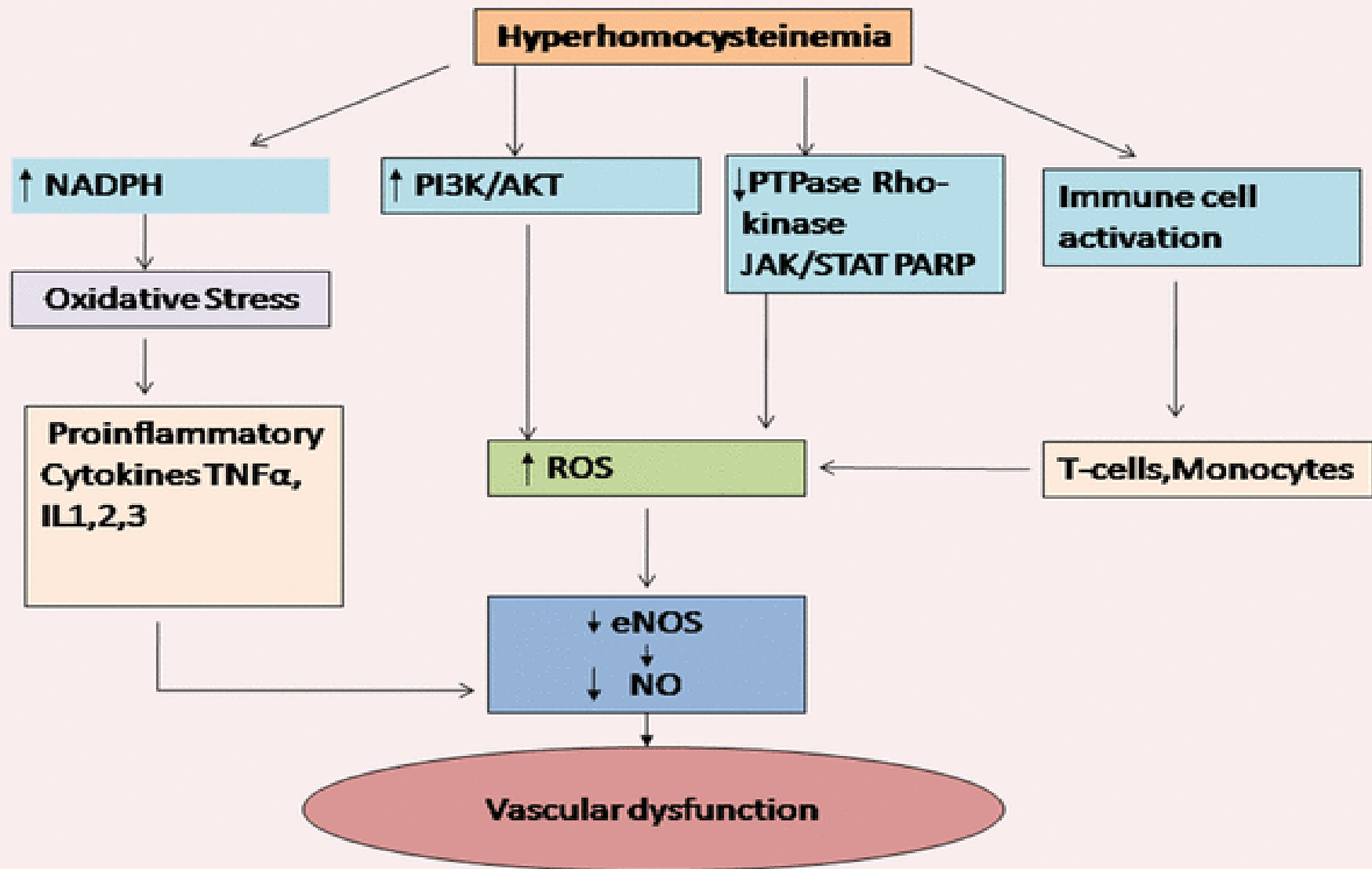
Vascular endothelium dysfunction in diabetes [Inflamm Res.](#) 2018 doi: 10.1007/s00011-018-1129-8. [Epub ahead of print]. Vascular endothelium dysfunction: a conservative target in metabolic disorders. [Jamwal S](#) and [Sharma S](#).



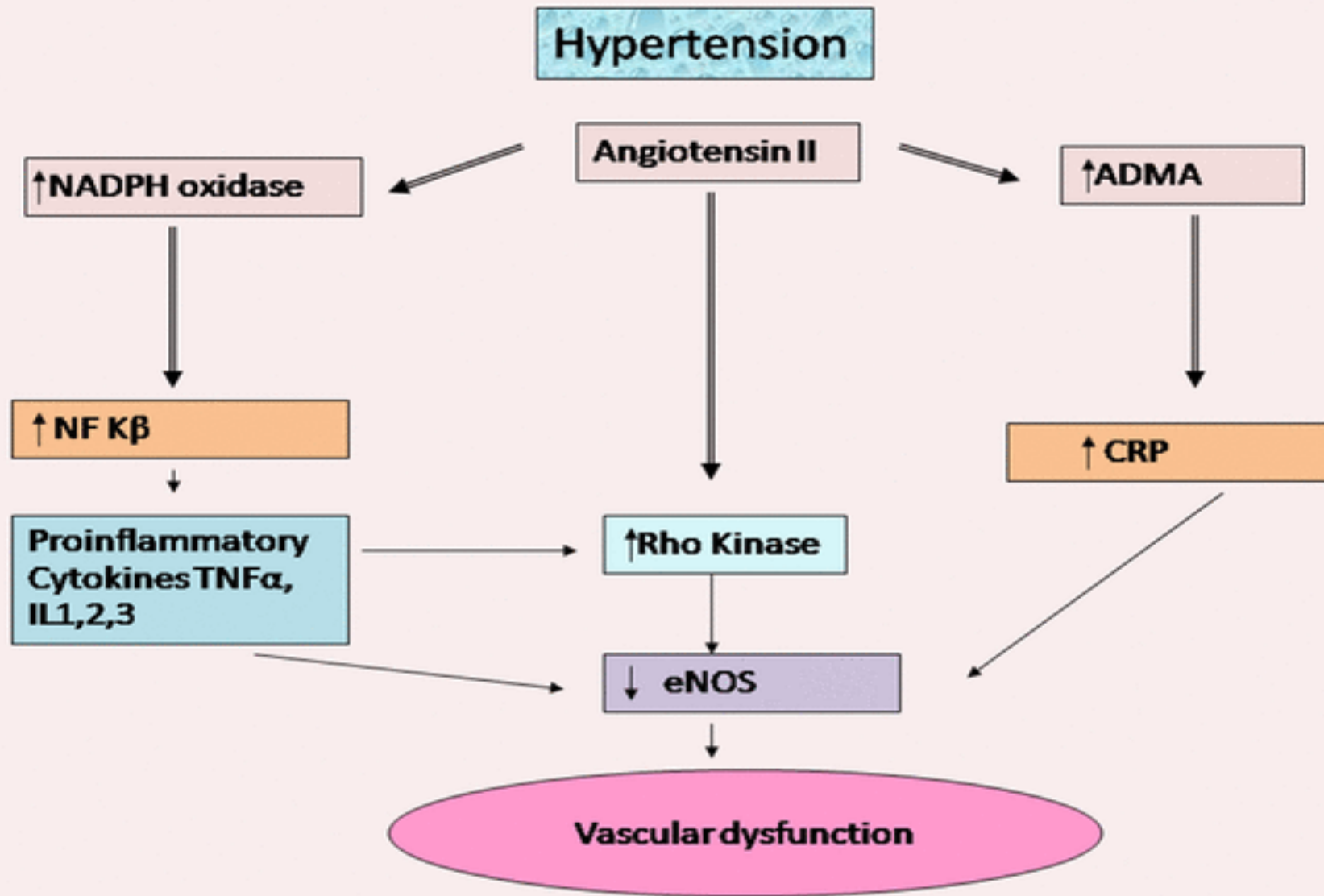
Hypercholesterolemia and vascular endothelium dysfunction [Inflamm Res.](#) 2018 doi: 10.1007/s00011-018-1129-8. [Epub ahead of print]. Vascular endothelium dysfunction: a conservative target in metabolic disorders. [Jamwal S](#) and [Sharma S](#).



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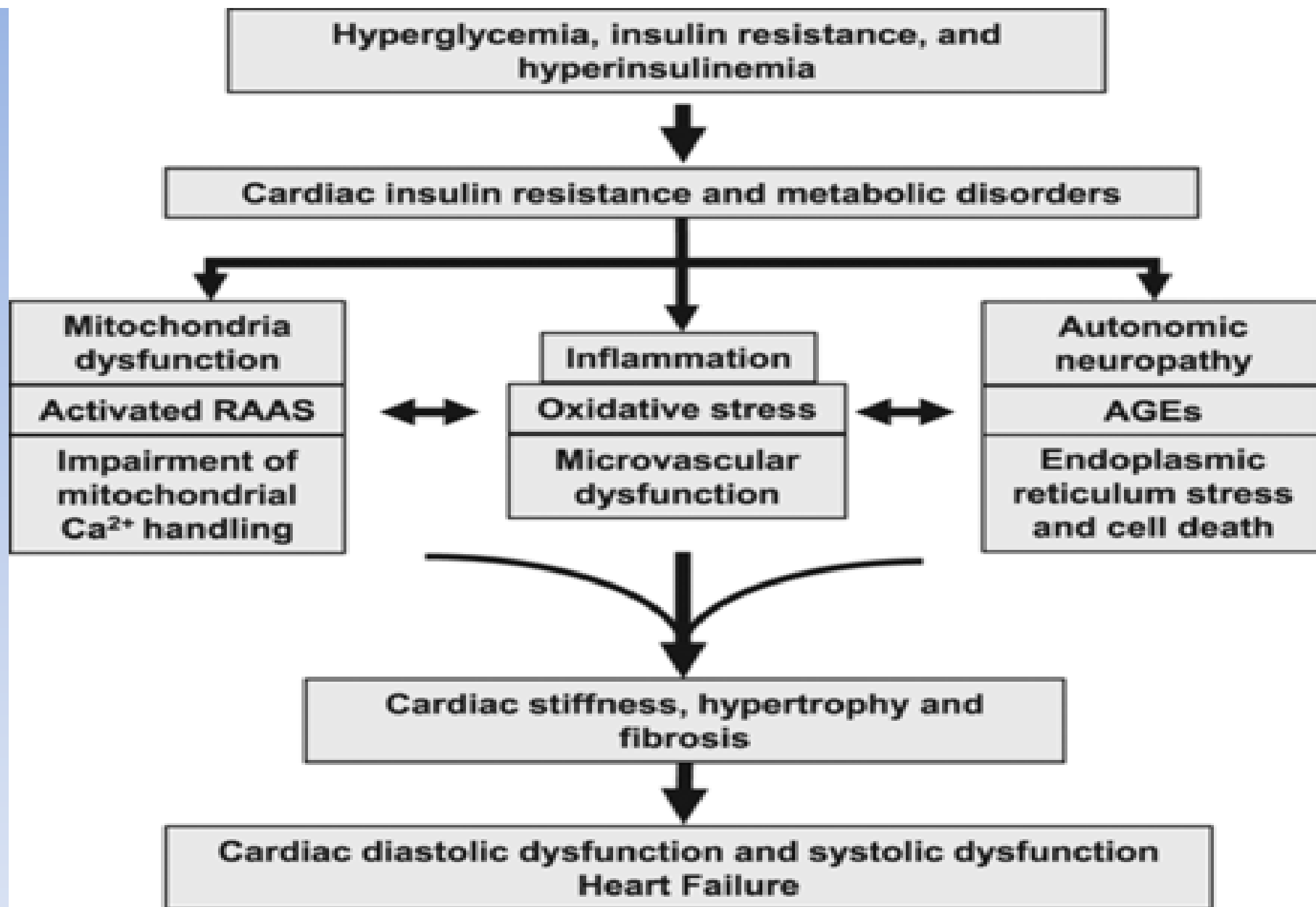


Hyperhomocysteinemia and vascular endothelium dysfunction [Inflamm Res.](#) 2018 doi: 10.1007/s00011-018-1129-8. [Epub ahead of print]. Vascular endothelium dysfunction: a conservative target in metabolic disorders. [Jamwal S](#) and [Sharma S](#).



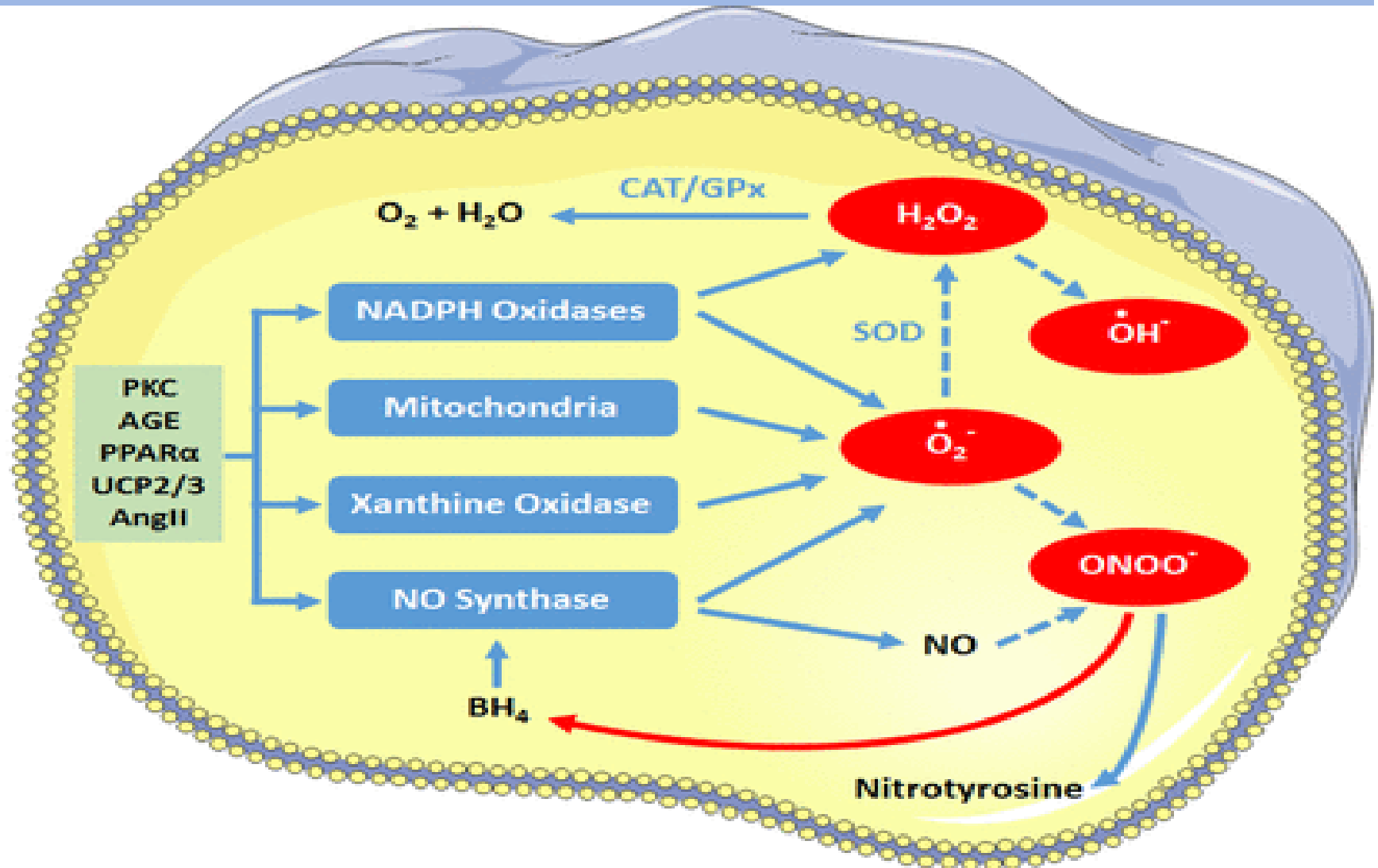
Hypertension and vascular endothelium dysfunction [Inflamm Res.](#) 2018 doi: 10.1007/s00011-018-1129-8. [Epub ahead of print]. Vascular endothelium dysfunction: a conservative target in metabolic disorders. [Jamwal S](#) and [Sharma S](#).

Diabetic Cardiomyopathy

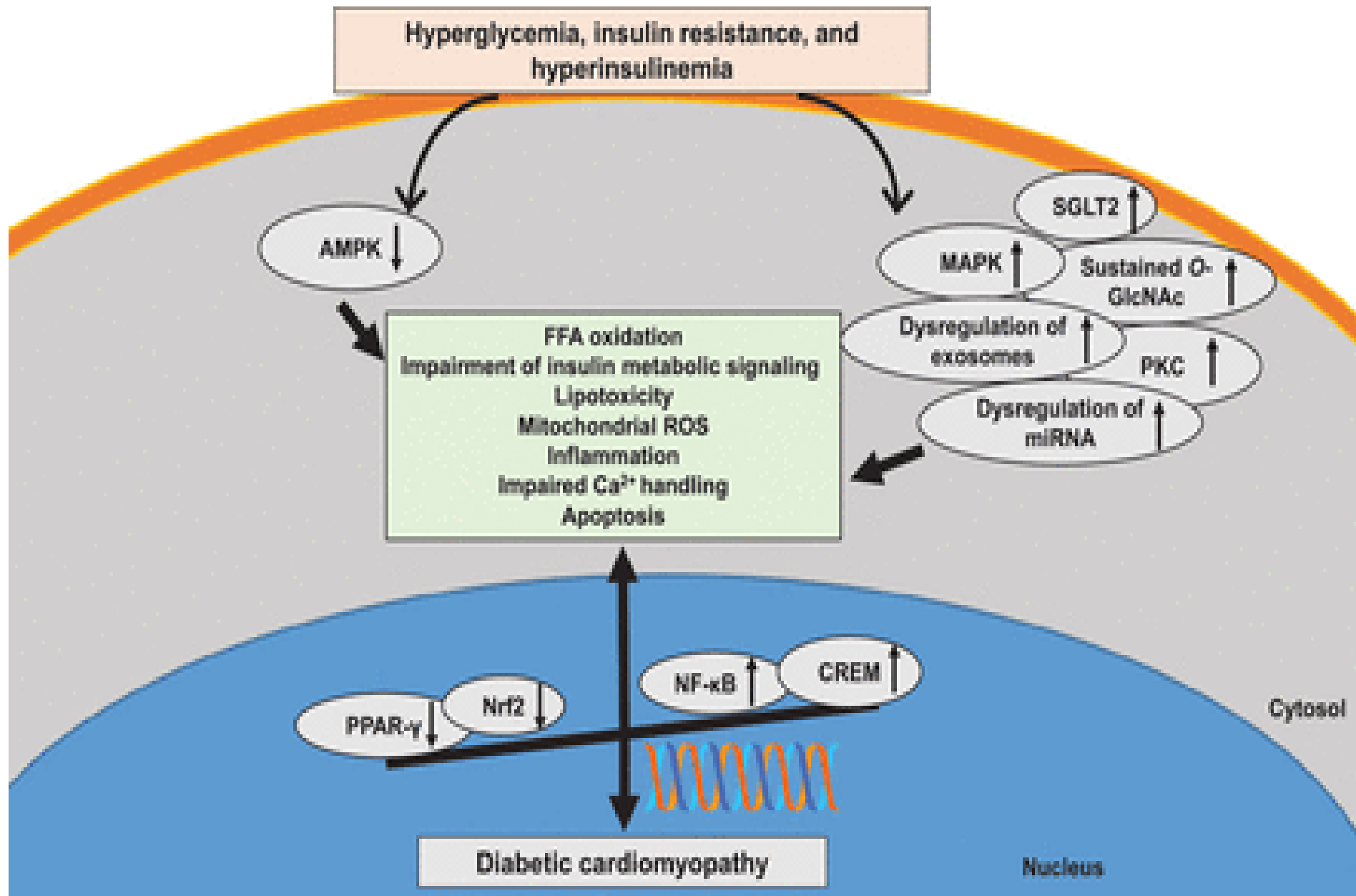


Pathophysiological mechanisms of diabetic cardiomyopathy. [Circ Res.](#) 2018 Feb 16;122(4):624-638. *Diabetic Cardiomyopathy: An Update of Mechanisms Contributing to This Clinical Entity.* [Jia G et al.](#)

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Reactive oxygen species production in diabetes. Various stimuli, primarily hyperglycaemia, activate major ROS sources, NADPH oxidases, mitochondria, xanthine oxidase and NO synthase, to generate superoxide prior to rapid dismutation to hydrogen peroxide. Under physiological conditions, hydrogen peroxide is degraded by endogenous scavengers, although when formed at increased concentrations it generates highly reactive hydroxyl radicals via the Fenton or Haber-Weiss reactions. Similarly, superoxide may combine with NO to form peroxynitrite, which may generate either cytotoxic nitrotyrosine or oxidise tetrahydrobiopterin, thereby reducing NO generation. Increases in activity/expression indicated by blue arrows, reductions indicated by red arrows. [Heart.](#) 2018,104(4):293-299. Reactive oxygen species signalling in the diabetic heart: emerging prospect for therapeutic targeting. [Wilson AJ et al.](#)



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