

HSP-90 and vasoregulation in portal hypertension

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A research team from Germany investigated the role of heat shock protein-90 (HSP-90) in neuronal NO-synthase (nNOS)-function and vasoregulation in the mesenteric vasculature. Their results showed that interaction and co-localization of nNOS and HSP-90 are evidenced. The inhibition of HSP-90 largely ameliorates enhanced nNOS-mediated vasodilation in portal hypertension making HSP-90 a potential therapeutic target during portal hypertension.

Neural vasoregulation represents a rapid and potent mode of altering vascular tone but has not been investigated thoroughly during [portal hypertension](#). Heat shock protein-90 (HSP-90) is well-known to act as a molecular chaperone optimizing endothelial and neural NO-synthase (eNOS, nNOS) enzyme activity and thus, NO production. Although HSP-90 has been shown to mediate in large parts the enhanced eNOS-dependent NO overproduction in the splanchnic circulation during portal hypertension, it is not clear what role HSP-90 plays in nNOS-mediated vasorelaxation in this scenario.

A research article to be published on April 21, 2010 in the [World Journal of Gastroenterology](#) addresses this question. This research relates to the utilization of the McGregor preparation enabling physiological and pharmacological testing of the whole mesenteric [vasculature](#) in its original anatomy and innervations. In contrast to arterial strips, this ensures testing of neural vasoregulation at close to in vivo conditions.

The investigators for the first time demonstrate a critical role of HSP-90

for nNOS-mediated vasorelaxation and furthermore, can provide evidence for this interaction being responsible in large parts for the well-accepted pronounced nNOS-dependent vasodilatation in portal hypertension. In addition, the authors visualize the localization of nNOS and HSP-90 in mesenteric nerves which can be appreciated as co-localized within the nerve axon. Finally, co-immunoprecipitation reveals a close protein-protein-interaction explaining the functional hemodynamic results presented. Therefore, HSP-90 may well have great potential to be identified as a future target in clinical trials focusing on amelioration of portal [hypertension](#) and associated hemodynamic disturbances.

More information: Moleda L, Jurzik L, Froh M, Gäbele E, Hellerbrand C, Straub RH, Schölmerich J, Wiest R. Role of HSP-90 for increased nNOS-mediated vasodilation in mesenteric arteries in portal hypertension. World J Gastroenterol 2010; 16(15): 1837-1844
www.wjgnet.com/1007-9327/full/v16/i15/1837.htm

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