



Magyar Sebész Társaság
Kísérletes Sebészeti Szekció XXVI. Kongresszusa
 Sebészeti Továbbképző Tanfolyam

Tudományos előadások összefoglalói

2017. szeptember 28-30. | Herceghalom, ABACUS Business & Wellness Hotel

METHANE MODULATES THE XANTHINE OXIDOREDUCTASE-DERIVED NITRIC OXIDE PRODUCTION UNDER ISCHEMIC CONDITIONS

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Background: Methane (CH₄) inhalation decreases tyrosine nitrosylation after ischemia-reperfusion injury (IRI), a process which involves nitric oxide (NO) and peroxynitrite formation. Xanthine oxidoreductase (XOR) is known to produce NO under hypoxia by reducing nitrite, and to generate superoxide upon reperfusion, thus playing a key role in both parts of such conditions. In previous experiments we have shown that the intestinal tissue NO levels were decreased in CH₄-treated rats during the ischemic phase, before the start of reperfusion. These data may suggest an influence of CH₄ on the NO metabolism under hypoxia, but to date, the in vivo interplay of CH₄ with NO has not yet been investigated. We aimed to examine the in vitro interactions of CH₄ with the NO regulating systems with special emphasis on XOR to shed light on the details of the in vivo mechanism.

Methods: Rat liver and ileum homogenates were incubated under anoxic conditions with or without 2.2% CH₄ to continuously monitor the release of NO purged from a liquid sample using a highly sensitive chemiluminescence-based NO detection method (Sievers Nitric Oxide Analyzer). In other experiments isolated XOR was used under similar conditions.

Results and Conclusion: We observed that CH₄ significantly decreases the rate of NO released in rat liver and ileum homogenates (17% and 6% reductions, respectively), a phenomenon which was in line with the effect observed in non-perfused tissues in vivo. In further in vitro studies, we confirmed that XOR is the major NO generating enzyme under anoxic conditions, accounting for up to 98% of NO measured. In experiments with XOR, nitrite and xanthine as electron donor, however, we detected 26% increase in the NO release in the presence of CH₄. In the next series CH₄ again reduced NO levels if XOR was co-incubated with denatured tissue homogenates. This opposing CH₄-induced effect might be attributed to an increased incorporation of NO in tissues, or to a shift in the substrate preference of XOR. Future research is definitely needed to elucidate these possibilities. Support: This work is supported by the grants NKFI K120232 and GINOP 2.3.2-15-2016-00015.