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Title: Interaction of the glutamatergic and nitrergic signaling system in the airway hyperreactivity

Dr. Anna 28064 Strapkova astrapkova@jfmed.uniba.sk and Dr. Martina 28065 Antosova antosova@jfmed.uniba.sk . [1] Department of Pharmacology, Comenius University, Jessenius Faculty of Medicine, Martin, Slovakia (Slovak Republic), 03601 .

Body: It is only little information regarding a possible interaction of glutamatergic and nitrergic system in the airways hyperractivity (AHR). We investigated the effect of agents modulating the activity of these systems on the experimental ovalbumin-induced AHR as well as on the changes of exhaled nitric oxide (eNO) levels. We used the agonists of NMDA receptors - N-methyl-D-aspartic acid (NMDA) and monosodium glutamate (MSG), selective competitive antagonist (DL-2-amino-5-phosphonovaleric acid – AP-5) and selective non-competitive antagonist (dizocilpine - MK-801) of these receptors. We used also non-specific inhibitor of NO synthases N-omega-nitro-L-arginine metylester (L-NAME). The AHR to histamine or acetylcholine was evaluated in in vitro conditions. NMDA administration caused the increase of tracheal smooth muscle response in ovalbumin-induced HR to acetylcholine. The effect of MSG was less pronounced. MK-801 as well as AP-5 provoked the decrease of reactivity mainly to acetylcholine in tracheal smooth muscle, while the former, non-competitive antagonist was more effective. We recorded the changes in eNO levels. The activation of NMDA receptor with NMDA or MSG increased eNO levels. The inhibition of NO synthase with L-NAME caused the fall of eNO levels. We suppose here the participation of constitutive isoforms of NO synthases mainly. MK-801 shows the more expressive effect on the eNO levels during sensitisation than AP-5 group. The results bring a whole new look regarding the relationship of the glutamatergic and nitrergic system in the airway inflammatory diseases. Supported by Centre of experimental and clinical respirology II, VEGA 14/0010/10, MZ SR 2007/46-UK-11.