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Inducible production of nitric oxide by olfactory ensheathing cells in response to bacteria

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The olfactory pathway represents a vulnerable route for pathogens such as pneumococcus, meningococcus, mumps, rabies, poliovirus, Herpes Simplex 1, West Nile Virus and Borna Disease Virus to access the central nervous system from the nasal cavity. Olfactory ensheathing cells (OECs), glial cells which ensheath the olfactory nerves from the nasal cavity to the olfactory bulb are in a prime position to assist with host immunity. This project investigates possible mechanisms relating to OECs' hypothesised role in host immunity, including the production of nitric oxide (NO), a potent antibacterial and antiviral agent. OECs were incubated with *Escherichia coli* and *Staphylococcus aureus*. Nitrite and NO production were analyzed using high performance liquid chromatography and live cell imaging, mRNA levels were assessed using Real-time RT-PCR, and inducible nitric oxide synthase (iNOS) expression by immunocytochemistry. An *in vivo* rat model was established to investigate iNOS expression in the compromised olfactory pathway. We show that elevated levels of nitrite were detected in bacteria-treated OECs, compared to untreated OECs and this was attenuated by the NO synthase inhibitor L-NMMA. Bacteria-treated OECs produced elevated levels of NO using the diaminofluorescein dye DAF2-DA (also attenuated by L-NMMA). mRNA was detected for iNOS in OECs but not for neuronal nitric oxide synthase or endothelial nitric oxide synthase. Expression of iNOS was elevated in bacteria-incubated OECs compared to untreated OECs. It was found that in the zinc sulphate treated rat olfactory pathway, bacteria were able to infiltrate the compromised olfactory tissue and that iNOS expression was up regulated in OECs. The potential immune capacity of OECs, as indicated by NO production, is significant in the context of OECs being trialed as an agent of neural repair because of the damaging inflammatory reactions present in injury.