# Simple drug treatment may prevent nicotine-induced SIDS

June 3 2009

Josef Buttigieg, lead author of a new study that has found a class of drugs that could be effective in treating babies vulnerable to SIDS, and his academic advisor, Colin Nurse, professor in the Department of Biology.

(PhysOrg.com) -- A new study has identified a specific class of pharmaceutical drugs that could be effective in treating babies vulnerable to Sudden Infant Death Syndrome (SIDS), because their mothers smoked during pregnancy.

According to researchers at McMaster University, exposure of the fetus to nicotine results in the inability to respond to decreases in oxygen—known as hypoxia—which may result in a higher incidence of

SIDS. In the same study on rats, they found that the diabetic medication 'glibenclamide' can reverse the effects of nicotine exposure, increasing the newborn's ability to respond to hypoxia and likely reducing the incidence of SIDS.

The findings are published today in the Journal of Neuroscience.

"During birth the baby rapidly changes its physiology and anatomy so that it can breathe on its own," explains Josef Buttigieg, lead author who conducted his research as a PhD graduate student in the department of Biology. "The stress of being born induces the release of the hormones adrenaline and noradrenaline—collectively called catecholamines—from the adrenal glands. During birth, these hormones in turn signal the baby's lungs to become ready for air breathing."

For some months after birth, the adrenal glands act as a critical oxygen sensor. A drop in blood oxygen levels will stimulate the release of catecholamines, which in turn signals the baby to take a deep breath, when an infant rolls on its face or has an irregular breathing pattern during sleep, for example. However, the ability to release those hormones during moments of apnea or asphyxia is impaired due to nicotine exposure.

During those episodes, specific proteins sensitive to hypoxia stimulate the cell to release catecholamines. A secondary class of proteins then acts as a 'brake', ensuring the cells do not over excite themselves during this stressful time. However, exposure of the fetus to nicotine results in higher levels of this brake protein.

"The result is like trying to drive your car with the parking brake on. You might go a little bit, but the brakes hold you back," explains Buttigieg. "In this case, the adrenal glands do not release catecholamines during hypoxia -for example during birth or a self-asphyxiation

episode—often resulting in death."

But when researchers administered the drug glibenclamide in laboratory rats, which override the brake protein, the adrenal glands were able to respond to oxygen deprivation, therefore reversing the lethality of hypoxia.

"Our initial goal was really to understand how the nervous system regulates oxygen sensitivity of cells in the adrenal gland at a basic research level," says Colin Nurse, academic advisor on the study and a professor in the department of Biology. "We speculated that chemicals released from nerves might interact with adrenal cells and cause them to lose oxygen sensitivity. It turns out that nicotine mimics the effects of one of these chemicals, thereby allowing us to test the idea. The present study was significant in that it led to a mechanistic understanding of how nicotine works in this context."

Source: McMaster University (news : web)