Alcohol, pregnancy and brain cell death

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Rutgers University's Dipak Sarkar, professor in the Department of Animal Science at the School of Environmental and Biological Sciences, director of the Endocrine Research Program, and a faculty member of the Center for Alcohol Studies. Credit: Rutgers University

Rutgers University Professor Dipak Sarkar has received a $3.5 million MERIT Award from the National Institutes of Health (NIH) to continue researching the damaging effects of alcohol on the nervous systems of the unborn.

The MERIT (Method to Extend Research In Time) Award will extend NIH support another 10 years for one of Sarkar's research grants, now in its 13th year. Sarkar has five active grants that support the work of 16 research assistants, including post-doctoral students, graduate students, undergraduates, and a senior scientist, who collaborate on his research

projects. Sarkar jokingly says he needs five grants "just to feed these people."

"Alcohol consumption during pregnancy is a significant public health problem and may result in a wide range of adverse outcomes for the child," Sarkar says. "Many Fetal Alcohol Syndrome patients have problems coping with stress; they have learning disabilities, infections, and increased susceptibility to diseases."

These problems stem from the alcohol-induced destruction of neurons in the part of the brain known as the hypothalamus. (A graphic will show its location in the brain) These beta-endorphin neurons produce the endorphin hormone and are particularly vulnerable during the early development of the fetus.

Sarkar is a professor in the Department of Animal Science at the School of Environmental and Biological Sciences, director of the Endocrine Research Program, and a faculty member of the Center for Alcohol Studies. His interest in alcohol research began in 1990 when he serendipitously observed the neuron-killing effect of a small dose of alcohol while working on neuronal development.

Sarkar's research has shown that a seemingly irreversible reduction in the number and function of beta-endorphin neurons results in a permanent impairment of stress and immune system functions throughout life. While the body often displays the ability to recover from damage or disease, this does not seem to come into play with the loss of beta-endorphin neurons.

Sarkar says that preliminary data on the reduced function of beta-endorphin neurons is pointing toward "epigenetic" changes as a causal factor - changes in biochemistry that inhibit the genes responsible for these particular neurons. The genes themselves become abnormal and,

while they may be producing some cells, the cells do not produce endorphin.

"One thing we cannot reverse is the death of these cells, but maybe we can reverse those epigenic alterations that are ultimately responsible for their demise," Sarkar says.

His continuing research is aimed at discovering the molecular mechanism involved in alcohol's toxic action on beta-endorphin neurons. A clear understanding of the underlying mechanism could offer a starting point from which to develop pharmaceuticals for fetal alcohol patients in the future.

Beta-endorphin neurons are also known as opioids because, like opium-based narcotics, their hormone products have the ability to reduce pain and increase a sense of well-being. Their loss would consequently have an opposite effect, reducing the ability to manage stress.

Consistent with this condition but unrelated to fetal alcohol exposure, there is substantial evidence that people with depression, schizophrenia, and other psychological disorders also have lower numbers of opioids, Sarkar says.

These neurons also have connections with the lymphatic system, which is engaged in transporting immune cells to and from the lymph nodes and can stimulate an immune response. Again, a reduction in the number of opioids can lessen the immune response and decrease the body's ability to fight infection and disease.

Beyond stress and immune function, the opioid system is also very much involved in metabolism. Sarkar notes that researchers are finding substantial evidence that an altered opioid system is involved in the metabolic changes leading to diabetes as well as obesity.

Source: Rutgers University (news : web)

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