Anesthesia sends neurons down the wrong path in unborn rat babies

While there has long been concern that exposure to anesthesia affects brain development, a study from the University of Illinois at Urbana-Champaign and the University of California at San Francisco has provided further evidence of the deleterious effects of anesthesia on brain development. The study, published in the journal Developmental Neuroscience, showed that exposure to anesthesia during the early stages of brain development can disrupt the normal organization of neurons in the developing brain, leading to changes in the brain's structure that may contribute to the development of autism.

The study involved the use of a mouse model to examine the effects of anesthesia on brain development. The researchers exposed pregnant mice to anesthesia during the critical period of brain development, and then examined the brains of the offspring at adulthood to assess the impact of anesthesia on brain function.

They found that exposure to anesthesia during the early stages of brain development led to changes in the structure of the brain, including a reduction in the number of neurons and a disruption of the normal organization of neurons in the developing brain. These changes were associated with changes in behavior, with offspring exposed to anesthesia showing a greater susceptibility to autism-like behaviors and a reduction in social interactions.

The findings of this study are significant because they provide further evidence of the potential long-term effects of anesthesia on brain development, and highlight the need for further research to understand the mechanisms underlying these changes. It is hoped that this work will help to inform future guidelines for the use of anesthesia in pregnant women, and ultimately reduce the risk of exposure to anesthesia during critical periods of brain development.

Call for immediate treatment

Doctors call for immediate diagnosis and treatment of Parkinson’s disease as more than 2% of Chinese people above 65 were overtaken by this disease.

Mammalling a healthy body weight is no simple matter. A better understanding of how the body regulates appetite could help to tackle the issue. Understanding how the appetite is regulated is a long-standing goal, a team led by researchers at Baylor College of Medicine and the University of Texas Southwestern Medical Center conducted a study in mice that found the gene SLC1A2 is responsible for regulating the function of neurons in the hypothalamus. The study was published in the journal Cell, and the team’s approach could be used to develop new treatments for obesity.

The team identified a protein called cortistatin, which was previously known to be involved in the regulation of body weight. They found that cortistatin is produced by neurons in the hypothalamus, a brain structure that plays a key role in the regulation of appetite and body weight.

Using a combination of genetic and biological techniques, the team was able to show that cortistatin was produced by neurons in the hypothalamus of mice that were genetically modified to overexpress cortistatin, and that this overexpression was associated with decreased appetite and reduced body weight.

The team also found that cortistatin was produced by neurons in the hypothalamus of mice that had been treated with cortistatin, and that this treatment was associated with reduced appetite and reduced body weight.

The results of this study suggest that cortistatin is a key regulator of body weight, and that it could be used to develop new treatments for obesity. The team is now working to understand the mechanisms underlying the regulation of cortistatin, and to develop new strategies for targeting this pathway to treat obesity.