

SIDS research confirms changes in babies' brain chemistry

September 14 2017, by David Ellis



Credit: University of Adelaide

University of Adelaide researchers have confirmed that abnormalities in

a common brain chemical are linked to sudden infant death syndrome (SIDS).

In the first study of its kind looking at babies outside the United States, researchers from the University of Adelaide's Adelaide Medical School investigated 41 cases of SIDS deaths and discovered striking abnormalities in chemical [serotonin](#) within the brain.

Serotonin, otherwise known as 5-HT, is a neurotransmitter found in different parts of the human body, including the central nervous system. Among its many roles, serotonin is involved in the regulation of sleep, and also control of the cardiovascular and respiratory systems.

This latest research, published in the *Journal of Neuropathology & Experimental Neurology*, confirms and supports the concept that brainstem dysfunction, resulting in significantly altered serotonin expression, is associated with some SIDS deaths.

SIDS is the sudden unexpected [death](#) of an infant under one year of age that cannot be explained after a thorough investigation, including an autopsy. It is the leading cause of death in infants between one month and one year of age in Australia and the developed world.

The research was conducted by PhD student Dr Fiona Bright under the supervision of University of Adelaide Professor of Pathology Roger Byard. Dr Bright graduates with her PhD from the University of Adelaide today (Thursday 14 September).

Her work builds on research conducted in the United States at the Boston Children's Hospital and Harvard Medical School, where Dr Bright was based for 18 months during her combined studies.

"Our research is significant because it has confirmed that abnormalities

in serotonin in the brain are most definitely linked to cases of SIDS. This helps to support the findings of the American research," Dr Bright says.

"Serotonin is a key neurochemical that plays an important role in the control and management of the complex respiratory, cardiovascular and autonomic systems within the human infant brainstem.

"Our research suggests that alterations in these neurochemicals may contribute to brainstem dysfunction during a critical postnatal developmental period. As a result, this could lead to an inability of a SIDS infant to appropriately respond to life-threatening events, such as lack of oxygen supply during sleep.

"Notably, the SIDS cases we studied were all linked to at least one major risk factor for SIDS, with more than half of the infants found in an adverse sleeping position and having had an illness one month prior to death," Dr Bright says.

Professor Byard says: "Better understanding of the complex role of these neurochemicals, and the exact causes of their dysfunction in the brain, will help future research to develop potential biomarkers for [infants](#) at increased risk of SIDS.

"Ultimately, we hope that this work will lead to improved prevention strategies, helping to save baby's lives and the emotional trauma experienced by many families."

This research was funded under a fellowship established by the River's Gift SIDS charity.

"River's Gift's primary objective is to fund world-leading SIDS research to make a tangible contribution to the discovery of a cure for this heart-breaking loss of life," says River's Gift General Manager Karl Waddell.

"The University of Adelaide research is a significant step towards achieving that objective."

More information: Fiona M. Bright et al. Medullary Serotonin Neuron Abnormalities in an Australian Cohort of Sudden Infant Death Syndrome, *Journal of Neuropathology & Experimental Neurology* (2017). [DOI: 10.1093/jnen/nlx071](https://doi.org/10.1093/jnen/nlx071)

Provided by University of Adelaide

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